

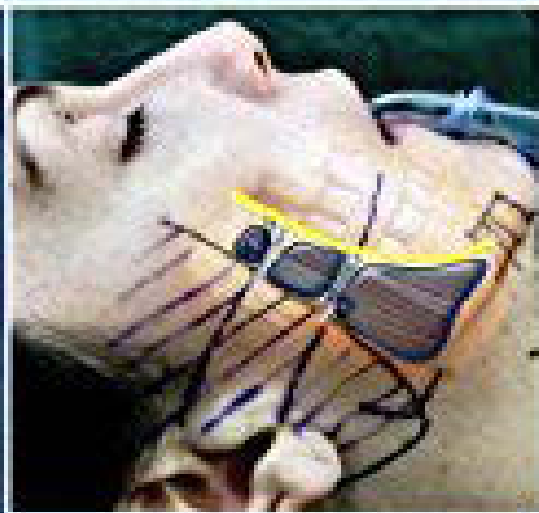


Plastic and Reconstructive Surgery

Approaches and Techniques



Edited by
Ross D. Farhadi
Neil W. Bulstrode
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Plastic and reconstructive surgery

Plastic and reconstructive surgery

Approaches and techniques

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Foreword

Plastic surgery is a unique specialty, defined by concept rather than anatomical area. As such, it has grown enormously over the last 70 years and continues to evolve with changes in technology, improved understanding of anatomy and patient-centred outcomes. This progression leads to a vast and ever increasing array of new techniques and options for reconstruction, benefiting both our patients and the many other medical and surgical specialties that consult the plastic surgeon. With many tools at their disposal and the wide array of clinical maladies that they treat, the plastic surgeon has evolved into a problem solver. This synergy between plastic surgery and other surgical specialties has enabled these other specialties to utilize the problem-solving skills of the plastic surgeon to expand their therapeutic spectrum and tackle increasingly more difficult problems. However, to be an effective problem solver it is incumbent on the plastic surgeon to be familiar with the latest developments of our broad and expanding field. This highlights the necessity of a single-volume text that is comprehensive and practical, covering the full spectrum of plastic surgery and presenting the current state of the art of our specialty. This is exactly what the editors of *Plastic and reconstructive surgery: Approaches and techniques* set out to achieve in producing this excellent textbook.

It is truly an international effort at all levels, as the editors, from Australia (Ross D. Farhadieh), the UK (Neil W. Bulstrode) and Canada (Sabrina Cugno), have joined forces to recruit over 130 international contributors and produce a resource of over 1100 pages that provides a well-organized and thorough, yet succinct, text of the essentials of current plastic surgery. The editors are all highly qualified and accomplished young plastic surgeons, and they have been able to provide a global perspective of our specialty. Many of the contributors are world-renowned experts; however, there is also a new generation of young rising stars whose contributions are equally good, providing a new, fresh and contemporary feel.

Each chapter is clearly organized and provides an overview of the principles and the most recently described basic science essentials, as well as clinical applications and techniques, and pertinent bibliography for additional reading. The critical core information is provided for each topic, providing an excellent synopsis and reference for the student and practitioner.

Although aimed primarily at the trainee, I believe that it will also serve as an excellent and quick reference for the seasoned practising surgeon faced with complex problems requiring reconstruction throughout the body. It will also be an especially useful resource for senior plastic surgery trainees preparing to take their Board and Fellowship exams. The final chapter, dealing specifically with the certification process and fellowship exams, is interesting, and provides useful information and different perspectives of the qualifying processes in the British, European, Australian and North American systems.

It has been an honour and pleasure for me to have been asked to contribute the Foreword to this new textbook, which very nicely fulfills one of the traditions of surgery of passing down knowledge from one generation of problem solvers to the next. The new generation is likely to face even more complex problems and, using the latest techniques, solve them more elegantly than we can now. But the principles in plastic surgery never change, and this textbook provides the intrinsic fundamentals that all trainees must know, even as the field is ever expanding.

I congratulate the editors for their Herculean effort in recruiting an international cast of distinguished surgeons and thank the authors for flawlessly summarizing the huge avalanche of new information that has graced our specialty; finally, I thank the publishers, Wiley Blackwell, for producing this timely, impressive and comprehensive plastic surgery compendium.

Julian J. Pribaz
Professor of Surgery
Harvard Medical School

Dedication

To inspirational mentors and dedicated apprentices everywhere.

Preface

During my plastic surgery training there appeared to be a plethora of summary and broad-stroke single-volume plastic surgery textbooks, all of which lacked adequate detail. Conversely I would encounter multivolume behemoths, detailed reference texts that always seemed leaden and difficult to digest. In my experience neither of these options fully addressed the needs of a trainee surgeon, or for that matter a more senior surgeon. Thus was born, on a long flight from Sydney to London, the notion of compiling a single-volume textbook that seeks to achieve the perfect balance of detail and palatability. To that end, in compiling this textbook we approached some of the world's leading authorities in the various fields of plastic surgery. This was with the belief that not only could readers benefit from such experts' enormous experience, but they could also gain practical insights from the ability of such experts to sift through the ever increasing volume of literature and distil what is relevant and applicable to everyday practice.

My co-conspirators in this endeavour, Mr Neil Bulstrode from Great Ormond Street and Dr Sabrina Cugno from Montreal Children's Hospital, had the perfect blend of enthusiasm and sense of humour to see this work through. I am grateful to them for their advice, time, effort and, most importantly, their friendship, all of which made this compendium possible. I also wish to thank all of our colleagues who took time out of their busy lives to make this volume possible. It has been an extraordinary experience for all of us to collaborate on this project. Our special thanks go to Professor Julian Pribaz for

being kind enough to review the volume and write the Foreword. We also wish to thank Wiley Blackwell for their continued support for this project.

On a personal note I wish to thank my mother Tadjvar, my brother Arash and wife Yassi for enduring me during the last 2 years, and for their unwavering support. My parents have been my guiding light in demonstrating the importance of a strong work ethic and integrity. I also wish to thank Messrs Neil Bulstrode and Adriaan Grobbelaar for the extraordinary fellowship opportunities and friendship they have afforded me. I am very grateful to Messrs Ash Mosahebi and Jian Farhadi, who kindly accommodated me in their operating theatres and taught me a great deal about the art of plastic surgery. In Melbourne I wish to extend my sincere thanks to Mr Bryan Mendelson, who during my visits showed immense patience and generosity in teaching me the philosophy and techniques of facial aesthetic surgery. My gratitude also goes to Mr Stephen Flood, who illustrated by example a sensible approach to plastic surgery and kindness in mentorship; this continues to serve as an aspiration. As a plastic surgeon I am most indebted to Professor Wayne Morrison, who has curiosity, humility, dedication to teaching and generosity of will and spirit, tempered with a crisp sense of humour, in equal inspirational measure. It was truly an honour and a privilege to serve as his registrar.

Rostam D. Farhadieh
Sydney
January 2015

About the companion website

This book is accompanied by a companion website:



www.wiley.com/go/farhadieh/plasreconsurgery

The website includes:

- Powerpoints of all figures from the book for downloading

PART I

Basic science and principles

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CHAPTER 1

Wound healing and scar formation

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TYPE I – CLASSICAL CUTANEOUS WOUND HEALING

A wound, in the context of skin, is a breach in the barrier that distinguishes an organism from its environment. The process through which the organism works in order to address this breach is 'wound healing' which, because of the important role the skin plays in the survival of the organism, is quite literally vital, and conserved through evolution. In the normal course of events, a lower species accepts tissue loss and heals a wound by exposure, licking, picking and, at the molecular level, scarring. The single most important impact on wound healing in humans is the early closure of wounds, by apposition with sutures in incisional wounds, and skin replacement in excisional wounds. Humans can deny significant skin and composite tissue loss by a 'like for like' replacement in the specialty of plastic and reconstructive surgery, and here we can boast a form of 'supranormal wound healing'.¹

Wound healing classifications

There are many ways to classify wound healing. In simple terms, we can consider:

- four phases – coagulation, inflammation, fibroplasia and remodeling;
- four types – fetal, adult, acute and chronic;
- four ages – young, plateau, regressing, atrophic; and
- two systems of healing – epidermal and dermal.

We can also classify wound healing in terms of clinical features and their wound management.

Phases of wound healing

Wound healing can be considered a process of four sequential but overlapping phases by which the body closes a breach in tissue continuity. There are many ways to define such a complex

process. Key to understanding the standard classification of the process is a consideration of: the timing, the cellular activation or influx and the chemical mediators.²

Coagulation

This *immediate* response to cutaneous injury involves two cascades: the clotting cascade, with the formation of platelet clot which adheres to the collagen exposed following endothelial disruption; and the complement cascade and complement-mediated vasodilatation. Histamine (released by mast cell degranulation) and kinins contribute to vasodilatation and increased vascular permeability. The first cells involved then are platelets and mast cells, and the first mediators histamine, kinins, platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF- β). The increasing interest in platelet-rich plasma (PRP) clinically in regenerative medicine is based on these early cascades.

Inflammation

Between the time of injury and around 4 days post injury, the clinical signs of inflammation develop. Classically these are redness, swelling, pain and loss of function. These result from inflammatory mediators and the capillary leak into the extracellular space that they coordinate. The next inflammatory cell type to arrive at the wound is the macrophage, followed by neutrophils and then lymphocytes. Keratinocytes in the wound edge and follicle remnants migrate and proliferate, and fibroblasts are chemo-attracted, and become activated.

Fibroplasia

From day 4, and for 2–4 weeks, the wound bed becomes vascularized, and type III collagen is laid down by fibroblasts to replace any dermal loss. In the absence of epidermal cover, this appears clinically as granulation tissue. Closed wounds become red and raised for a while. Hydrated glycosaminoglycans form a ground substance for the collagen fibrils. This phase is characterized by fibroblast proliferation, but also by keratinocyte proliferation.

Remodelling

From a few weeks to 18 months or more, the wound goes through a long phase of remodeling. Fibroblasts mature into myofibroblasts to contract the wound. Type III collagen is gradually replaced by type I collagen. Disorganized collagen becomes lamellar.

Repair versus regeneration

There are significant differences between the wound healing seen in fetal life and that seen in postnatal life. So-called scarless healing occurs for a period in the fetus, however this is not absolute but dependent on gestational age and wound size.³ In late fetal life scarring does occur, and before this point in time, a large enough wound will still result in scarring. In postnatal life, scarring is the inevitable and permanent consequence of wounding beyond the epidermal basement membrane. An adult lower vertebrate such as a salamander can regenerate an amputated limb but will not aspire to climb Mount Everest or graduate from Oxford! Although regenerative medicine attempts to harness the same plasticity seen in lower vertebrate regeneration, what is generally achieved is only 'partial regeneration', and not replacement of like with like. The ongoing concern in work to manipulate adult/somatic stem cells to achieve true regeneration is around a loss of control and the risk of carcinogenesis.

Acute versus chronic wound healing

Acute wound healing is hard to distinguish absolutely from chronic wound healing, and the processes at a cell and molecular level may be similar.⁴ Chronic wound healing occurs when healing takes longer than might be anticipated in a fit, healthy person, and is often associated with comorbidity. Such wounds seem to become stuck in the inflammatory or proliferative/fibroplastic phases. Local wound management can only usefully begin following an assessment and optimization of systemic comorbid conditions.

Epithelial/epidermal versus mesenchymal/dermal wound healing

The epidermis provides the ultimate barrier between the body and environment. The main cell type is the keratinocyte. Regeneration occurs from a population of follicle stem cells.⁵ The dermis provides the structural support to the epidermis and other related adnexae. The main dermal cell type is the fibroblast. As in embryogenesis, there is ongoing 'cross-talk' between the epidermis and dermis in somatic cutaneous wound healing, the epidermal element of which has been relatively underplayed. Even less consideration has been given to the contribution of subcutaneous fat to cross-talk.

Wound healing and scarring

In many ways, wound healing and scarring are inseparable – the one leads to the other at some level and over time. When does a wound become a scar? That will probably depend at a molecular level on very early wounding and wound management events. Although a scar is the inevitable and permanent consequence of postnatal wounding beyond the basement membrane and a compromise between regeneration and repair, at a clinical level, its significance is largely patient related and subjective. This can now be captured clinically by the Patient and Observer Scar Assessment Scale (POSAS), which draws patients into their own management.⁶

Most patients would perceive a wound as a scar from the time that the wound is no longer open, and often that equates to the absence of exudate and any requirement for dressing care. Clinically, even a normotrophic scar will go through a natural progression to maturity. When young, it will be active. Most young scars exhibit features of hypertrophy. Most scars then go through a plateau period of relatively little clinical change in the absence of treatment. Most scars then go through a period of regression of inflammatory signs and symptoms, and eventually settle to a mature state. Some scars, after many months or years, and sometimes because of treatment, become very thin, pale and atrophic.

When then does a normotrophic scar become a pathological scar? A normotrophic scar results from uneventful primary healing, but there is wide variation with age, site and skin type, and a period of hypertrophy is not unusual. Hypertrophic scarring is classically seen in paediatric burn wounds that have struggled to heal.⁷ The scar becomes red, raised, painful and itchy around 2–3 months following wound closure, particularly in wounds that have taken more than three weeks to heal. The scar settles over 18 months to 2 years, but often incompletely. Hypertrophic scars occur particularly in extreme Fitzpatrick skin types. Presumably, there is a bell curve distribution within the population, where those at the extreme end of hypertrophic scarring could be termed pathological. Although a keloid scar shares many of the features of a hypertrophic scar, and may represent an extreme example of the same, it is defined by extension beyond the confines of the injury, by almost inevitable progression beyond 2 years, seldom regressing; by being refractory to most treatments and recurring within 4–6 months of cessation of most treatments; and by behaving pathologically like a benign tumour. Understanding such an extreme phenotype may prove key to the effective management of more normotrophic scarring.

Epidermal wound healing

Adult epidermal stem cell biology is relatively well understood. Keratinocyte stem cells reside primarily in the bulge region of the hair follicle and, by asymmetric division, populate the interfollicular basement membrane with transit amplifying cells.⁸ These divide a number of times to provide the differentiating cells of the stratifying epidermis. A huge array of small

peptides are involved in coordinating the response to epidermal wounding by autocrine, paracrine and juxtacrine signaling.⁹ In a human model of epidermal healing, a number of phases of keratinocyte activity are suggested, as shown in Figure 1.1.¹⁰

Acute activation

Almost immediately following wounding, the epidermis expresses interleukin 1 β (IL-1 β) and interleukin 6 (IL-6) and the dermis TGF- β 1, committing transit amplifying cells to mitosis. Although TGF- β 1 is antiproliferative, it is pro-migratory to keratinocytes.¹¹

Early activation

Towards 24h following wounding, keratinocyte proliferation and migration are clear. Epidermal expression of TGF- α and IL-6 is accompanied by dermal fibroblast keratinocyte growth factor (KGF) and IL-6 expression. A paracrine loop of epidermal IL-1 β induction of the potent keratinocyte mitogen KGF from dermal fibroblasts seems likely.¹² TGF- α both is a keratinocyte mitogen and induces the migratory K6/K16 keratinocyte cyto-

skeletal phenotype in the suprabasal compartment.¹³ It may also recruit nearby follicles by juxtacrine signalling.¹⁴

Restitution

Over weeks, homeostasis is restored, with relatively late activation of the bulge to restore the transit amplifying population.

Dermal wound healing

Wound healing studies have concentrated far more on the dermis than epidermis, and particularly on macrophage production of TGF- β isoforms. This multifunctional growth factor appears to play a key role in dermal healing and scarring.¹⁵ Although the TGF- β 1 isoform promotes scarring, the TGF- β 3 isoform appears to have the opposite effect.¹⁶ Juvista (Avotermin) was developed to improve the quality of normotrophic scarring, but failed in a European phase 3 clinical trial.¹⁷ TGF- β , however, remains a key pharmaceutical target.

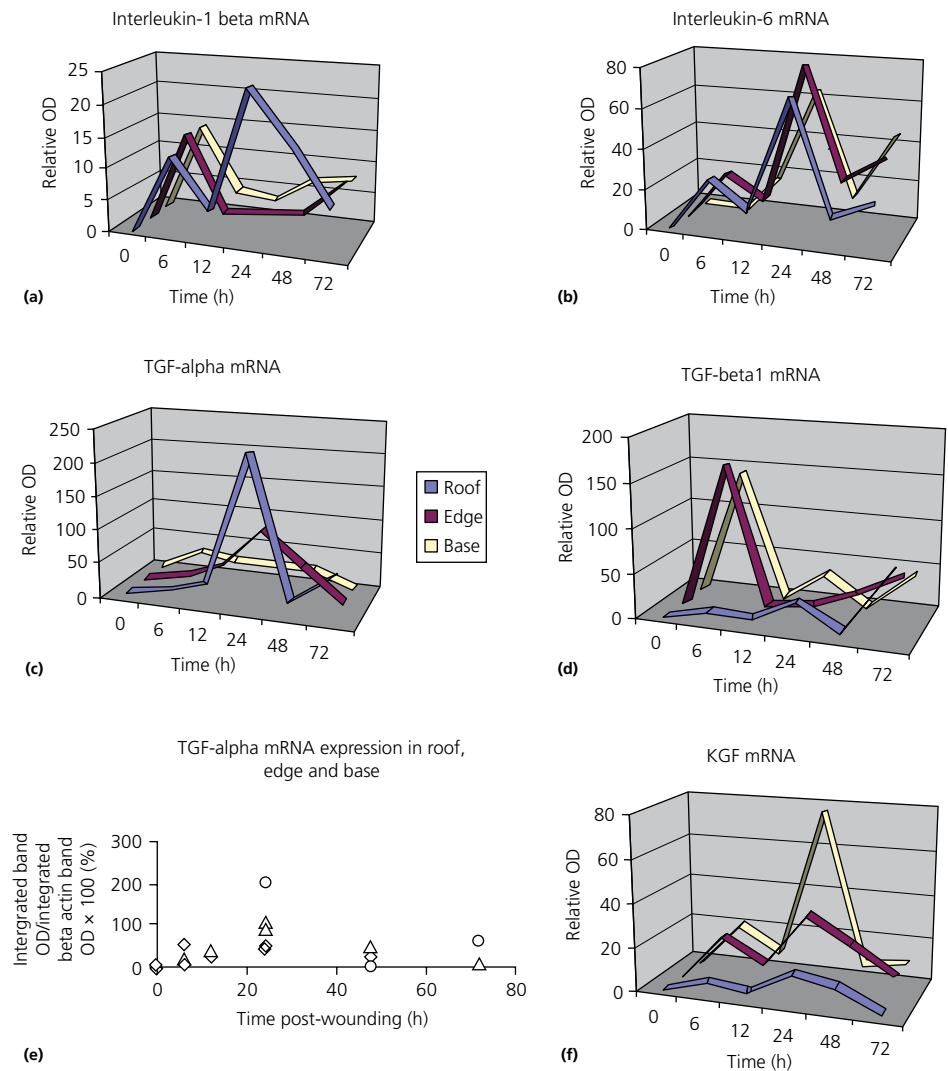


Figure 1.1 Temporo-spatial cytokine and growth factor gene expression in human suction blister wound healing. TGF, transforming growth factor; KGF, keratinocyte growth factor; relative OD, relative optical density.

A preoccupation with one growth factor, albeit a powerful, multifunctional factor with isoforms of different action, and for each a dose-dependent heterogeneity of responses, is arguably to ignore the complexity of wound healing cascades. There are many factors at play, often pleiotropic, and there is significant redundancy – so that many factors can contribute to the same outcome (i.e. rapid closure by scar). The growth factor that has shown most promise when delivered alone is PDGF.¹⁸

The dermis is far more than an extracellular matrix populated by fibroblasts. It hosts a vascular network (arterial, venous and lymphatic) and a neural network, and supports the follicle and other adnexal stem cell niches. It must also, it seems, interact with the subcutaneous fat. One approach to tissue engineering to provide for wound tissue loss is to synthesize an appropriate nanotechnology scaffold for key cellular elements to populate and develop. This can be *biofunctionalized* by incorporating a latent growth factor.¹⁹

Wounds can be classified based on clinical management and outcome as shown in Table 1.1.

'Normal'/primary incisional wound healing (type Ia)

Incisional wound healing occurs following surgical access, or 'incisional' or lacerating trauma.²⁰ In the former, the wound will begin sterile, and in the latter some degree of contamination is usual. In neither instance is there significant tissue loss. Classically, in modern medical practice, these wounds are formally closed,

although the timing of that closure will vary, affecting the quality of the healing processes and the scar that results.

Early closure (type lai)

If an incisional wound is closed directly, the healing will tend to be optimal. Elective surgical wounds are closed at the end of the procedure performed under sterile conditions. Traumatic lacerating wounds will generally be cleaned and closed the same day, and before significant bacterial colonization occurs. A relatively arbitrary time limit to early closure has evolved in practice of 48 h from injury. Beyond this, it is considered likely that colonization may be significant enough to deleteriously affect healing, and as a consequence the quality of scarring, which may become hypertrophic.

Late closure (type laii)

If a type Ia wound is sutured after 48 h, wound colonization may result in infection, dehiscence and delayed healing. Counterintuitively, according to current dogma if the wound is left open until day 4 or 5, as in 'delayed primary closure', satisfactory results can be achieved at a stage when the inflammatory response has become established – although a recent Cochrane review found no evidence for this.²¹

No closure (type laiii)

An incisional wound beyond the epidermal basement membrane that is left unclosed will gape and behave much like a deep excisional wound, healing by classical 'secondary intent'.²² The time

Table 1.1 A revised wound classification based on clinical management and outcome

Type I: Classical	Wound ± intervention	Character of healing
(a) 'Normal'/primary incisional – incisional, no tissue loss	(i) Closed early (<48 h)	Low risk of infection Minimal line scar
	(ii) Closed late (>48 h)	Increased risk infection Increased risk chronicity More significant scar
	(iii) Unclosed	Healing by granulation (similar to type Ibii), but in absence of tissue loss) High risk of chronicity
'Normal'/secondary excisional – tangential tissue loss, no replacement	(i) Above mid-dermis	Rapid re-epithelialization (<10 days) Minimal clinical scar
	(ii) Below mid-dermis	Slow/absent re-epithelialization High risk of infection and chronicity Closure by scar
Type II: Neoclassical		
(a) 'Supranormal' by skin replacement	(i) Early split- or full-thickness skin grafting of type Ibii)	Present gold standards Cosmetic and functional problems remain Donor defect
	(ii) Biotechnological skin replacements (Cuono technique, <i>in vitro</i> composite grafts)	Ideally autologous and with viable cellular elements Opportunity to improve on types Ibii and I lai
(b) 'Supranormal' with apparent acceptance of tissue loss	(i) Type Ibi treated with cultured keratinocyte allografts or vapour-permeable membrane	Follicle recruitment by activated extended follicle units
	(ii) Chronic full-thickness wound treated with cultured keratinocyte allografts or vapour-permeable membrane	Simulation of an acute wound environment

to healing will be slow, because closure will rely on wound base contraction and edge re-epithelialization. As a consequence, the quality of the scar will tend to 'pathological'.

'Normal'/secondary excisional wound healing (type Ib)

Where surgery requires skin excision, or cutaneous trauma is tangential (e.g. burn injury or friction loss) and the tissue loss is not replaced, then the time to healing and the scar quality will depend on the depth of the loss. Re-epithelialization and restoration of barrier function from adnexal remnants will be slower, the deeper the injury.

Above mid-dermis (type Ibi)

Tangential tissue loss above the mid-dermis leaves a partial-thickness wound that will heal in around one week under ideal circumstances, and result in a 'controllable' scar – as in the surgical split-thickness skin graft donor site. If a traumatic tangential injury clearly reaches the mid-dermis acutely (i.e. a mid-dermal burn injury), then optimal wound management is key to prevent extension of the tissue loss to type Ibii. In extreme Fitzpatrick types, even these type Ibi wounds can scar pathologically.

Below mid-dermis (type Ibii)

Tangential tissue loss below the mid-dermis leaves a partial-thickness wound that will take three weeks or more to heal, and as a consequence will be more liable to chronicity and significant scarring.⁷ It is at this depth, or beyond, that intervention is considered. A full-thickness defect can only re-epithelialize from the wound edge, and will otherwise close substantially by scar contraction of the base.

Abnormal wound healing

Systemic and local factors may reduce the quality of the skin and/or affect wound healing adversely. These are important to recognize and optimize.

Systemic factors

Systemic factors may be congenital or acquired. There are a handful of congenital conditions that affect the processes of healing, and in some instances the clinical quality of the skin and healing. A range of defects in collagen synthesis is seen in Ehlers–Danlos syndrome, and healing is poor.²³ The skin is vulnerable, and healing slow in epidermolysis bullosa, where for example, in the junctional variant, laminin 5 is deficient in the epidermal basement membrane zone.²⁴ The autosomal recessive premature ageing condition progeria manifests many features of normal 'acquired' ageing.²⁵

With age, the changes in healing processes are fairly global, resulting in a delay in wound closure and a reduction in wound strength. How much these changes are the result of increasing comorbidities associated with age, rather than age itself, is not entirely clear. Other acquired systemic factors include: nutrition, drugs, diabetes and smoking. Vitamin C deficiency is the classical example of a nutritional factor involved in wound healing. Although scurvy is not likely with Western diets, vitamin C is an essential cofactor for collagen synthesis. Vitamin A deficiency is also rare in the developed world, but vitamin A can reverse steroid-induced collagenase activity. Zinc is important to many enzyme systems, and deficiency can be seen in large burn injury. In those same injuries, albumin can plummet to around 10 g/L, and although this will delay healing, closure can be achieved.

Obesity is epidemic now in the Western world, and associated with many comorbidities and wound complications following surgery.²⁶ Plastic surgery reconstructions following bariatric surgery are challenging. Large blood vessels will have developed to support the tissue volume, and these may contribute to postoperative bleeding complications. Despite these hypertrophic vessels, tissue perfusion may be poor, and the tissue lymphoedematous and critically colonized. Furthermore, closure after such excisional surgery is by definition under tension, so that infection and dehiscence are more common.

Anti-inflammatory systemic glucocorticoids, non-steroidal anti-inflammatory drugs and chemotherapy drugs globally suppress the cellular responses to wounding. Chemotherapeutic angiogenesis inhibitors, such as bevacizumab, a vascular endothelial growth factor-neutralizing antibody fragment used in colonic cancer, cannot be prescribed six weeks before or after surgery to limit the wound healing risks.²⁷

Diabetes mellitus may affect healing in a variety of complex ways, particularly in the lower limbs. Patients with diabetes are susceptible to atherosclerosis in larger vessels, and tissue oxygen delivery is further reduced by the stiffness of the red blood cells, and the higher oxygen affinity of glycosylated haemoglobin.²⁸ These effects are compounded by any neuropathy, and impaired cellular immunity, phagocytosis and bacterial killing.

Smoking may affect wound healing in both immediate and longer term ways.²⁹ Nicotine causes sympathetic vasoconstriction, and carbon monoxide shifts the oxygen dissociation curve to the left. Long-term smoking accelerates atherosclerotic changes. Smoking appears to be a particular problem in surgery to superficial soft tissue planes, where wide skin undermining with the sacrifice of multiple perforators, and closure under tension are combined, as in abdominoplasty surgery.

Local factors

One of the most controllable local factors for incisional wounds is surgical technique and the handling of tissue. Tissue handling within the specialty can be observed, par excellence, under the microscope during a microvascular anastomosis, where poor

handling results in anastomotic thrombosis.³⁰ Local factors often reflect systemic comorbidities, so that poor blood supply and oxygen delivery, and even critical bacterial colonization are more often than not a local manifestation of a systemic factor. Conversely, the radiotherapy that results in local thromboendarteritis obliterans and causes healing problems over time may also have systemic effects.³¹ In terms of recurrence, radiotherapy is the most effective treatment for keloid scars, damping down the ‘overhealing’ provided it follows extralesional excision directly.³² Breaches in the skin are inevitably colonized by commensals. With time and increasing bacterial number, the body mounts an inflammatory response, and the colonization is termed critical. Critical colonization is not anticipated until around 48 h as a rule of thumb. Once 10^5 organisms are present per gram of tissue, the wound may be considered infected. Chronicity and some level of colonization go hand in hand. Bacterial biofilms are prevalent in chronic wounds, including anaerobic organisms not isolated by standard culture systems, and this is an area of particular current interest in such wounds³³ – and also of course in subcutaneous/cavity wounding and scarring (e.g. breast capsular contracture).³⁴

Mechanotransduction

There is a sense that the skin is constantly subclinically injured to some degree by sheer forces, and indeed even the force of gravity.³⁵ This may drive the baseline turnover of the skin. The effects of physico-mechanical forces on cell behavior, termed *mechanotransduction*, are becoming increasingly recognized and understood in wound healing.³⁶ In 1861, Karl Langer observed that the skin exhibits intrinsic tension,³⁷ and Langer’s lines, defined by the direction in which circular excisional wounds will elongate to ellipses by anatomic site, are used today to orientate excisional surgery. *Tensegrity* describes the way in which mechanical forces regulate biological systems via perturbations in structural architecture; disruption of tensional integrity triggers cellular pathways that restore mechanical homeostasis.³⁸ Cells also actively generate intracellular tension, *cell traction forces*, as they interact with their environment during, for example, migration.³⁹ A cell-centric view of mechanotransduction is, however, inadequate. Conformational changes in the extracellular matrix by mechanical forces can reveal cryptic binding sites and expose growth factors. Non-structural, extracellular, *matricellular* proteins (e.g. connective tissue growth factor) are increasingly implicated in the regulation of healing and scar formation.⁴⁰ Mechanosensing in the skin is a feature not just of fibroblasts but also of keratinocytes and nociceptors. It is quite possible that physical cues during wound healing direct, in part, stem cell fate within that niche.

Any therapeutic effects of silicone gels, pressure garments and linear taping may work through mechanical offloading and mechanotransduction pathways. The use of Botox A to control tension across healing facial scars and improve scar quality is an interesting new approach.⁴¹ Vacuum-assisted closure has become a common approach to complex wound management,

and although poorly understood, must rely to a large extent on mechanotransduction.

TYPE II – NEOCLASSICAL CUTANEOUS WOUND HEALING

‘Supranormal’ healing by skin replacement (type IIa)

Classically, intervention to close a wound was sometimes considered ‘tertiary’ wound healing. The great variety of techniques now available suggest a more structured classification.

Early split-thickness or full-thickness skin grafting of type Ibii (type IIai)

Those tangential traumatic or excisional wounds that are unlikely to heal in a reasonable timescale and are therefore likely to scar are most commonly closed with split-thickness skin autograft. Where the wound is full thickness, the environment optimal and the defect limited in size, a full-thickness autograft will provide a superior reconstruction. Of course, any number of local and distant skin and fasciocutaneous flaps are considered for defects that have resulted in an ungraftable bed, and this category of skin replacement feeds into other classifications of flap reconstruction. Skin and fasciocutaneous free flaps are increasingly being used to close defects in hostile comorbid conditions (e.g. in ‘vasculoplastics’ practice).⁴²

Biotechnological skin replacements (type IIaii)

In recent years, products, often xenograft in nature, have been developed to provide the quality of a full-thickness graft reconstruction from a split-thickness skin graft donor site. ‘Dermal regeneration templates’, such as Integra, engraft to provide a ‘neodermis’ to support a thin autograft in two operative stages.⁴³ A single-stage Integra system has been available following the success of single-stage Matriderm grafting.⁴⁴ Included here also is the system developed by Cuono and colleagues that combines allograft dermis with autologous cultured keratinocytes in the closure of full-thickness burn excision beds.⁴⁵ Cultured keratinocyte technology represents one of the most established forms of somatic stem cell therapy, and sits most coherently within plastic and reconstructive surgery.

‘Supranormal’ healing with apparent acceptance of tissue loss (type IIb)

When tangential tissue loss is accepted and no apparent attempt is made to replace like with like to the level of loss, then there are still interventions that seem to present some clinical advantage. George Winter presented his understanding of tangential wound healing in a porcine partial-thickness

excisional model that included both edge and base contributions to restoration of epidermal barrier function, and introduced the world to the concept of 'moist wound healing'. This spawned a massive industry in moist wound healing dressing systems, initially vapour-permeable membranes.⁴⁶ Although this transformed the 'dry' wound management of the time, it has not proven a panacea, and far more sophisticated biological systems have evolved since (see below). The clinical evidence base for these, however, has been slow to evolve on a cost basis, and so marketplaces have yet to develop around economy of scale.

Type Ibi treated with cultured keratinocyte allograft or biological dressing (type IIbi)

Cultured keratinocyte allografts have been used for decades to accelerate partial-thickness wound healing.⁴⁷ Although they do not survive transplantation long term,⁴⁸ they present a temporary and coordinated 'growth factor factory'. It may also be that they provide a juxtacrine mechanism for 'discontinuous follicle recruitment' by bridging adnexal remnants separated by tangential partial-thickness wounding.¹⁰ Biobrane is a conforming bilayer of porcine collagen and nylon.⁴⁹ It is at least haemostatic and adheres to a clean partial-thickness wound until shed when the epidermal barrier has been restored. The evidence for its efficacy and detail of any mechanism has never been established, but a role for the limitation of colonization of adherence seems likely.

Chronic full-thickness wound treated with cultured keratinocyte allograft or biological dressing (type IIbii)

Chronic full-thickness wounds are a huge financial burden to any healthcare system, and generally associated with comorbidity.⁵⁰ Any comorbid condition should be optimized in the first instance. There then may be some further benefit from cell-based therapy or biological dressings. Both cultured keratinocyte allografts and autografts have been used to treat such wounds.⁵¹ It is suggested that even with poor clinical autograft take, a more acute healing picture is seen, at least clinically – healing is 'kick-started'. The wound bed can be modulated with, for example, hyaluronic acid, an important component of the embryonic extracellular matrix, to improve clinical take, perhaps by supporting a niche-like stem cell environment.⁵²

Fat

There has been a huge recent interest in fat grafting, not only for augmentation including following subcision of indented scars and scar-related fat atrophy, but also to improve healing and the quality of the overlying scar.⁵³ It is fascinating to reflect that Sir Harold Gillies may have used whole-fat grafts in acute closure of

craniofacial traumatic wounds with the same intent many decades ago.⁵⁴ It has been suggested that any effect on healing and scarring results not from the grafted fat directly, much of which is lost, but from stimulation of a local mesenchymal stem cell response. The current controversy is around the method of enrichment of autologous fat to provide safe augmentation long term, and the resolution of this will run parallel with resolution of controversy around any effect on healing and scarring. A recent randomized controlled study in normal human volunteers demonstrated that enrichment of autologous fat with cultured autologous adipose-derived stem cells was significantly more effective, in graft survival terms, than a more standard approach.⁵⁵

Lymphoedema

Lower limb lymphovenous disease is a recognized cause of recurrent cellulitis and chronic wound healing. There is a newly recognized patient base in the morbid obese and postbariatric population, and with the evolution of supra-microsurgical techniques, the promise of new therapies (e.g. lymphovenous anastomosis and lymph node transplantation).⁵⁶

Future

Modern genomic and proteomic techniques allow us to define the processes that control tissue volume and its nature in healing at a molecular level – broadly: migration, proliferation, differentiation and apoptosis. Those techniques require very little material from biopsy, and we should expect to see more evidence from controlled longitudinal human studies available to support our understanding of the different types of healing. The sheer complexity of the pathways and interactions within countless networks will require a systemsomics, or systems biology, approach, calling on applied mathematics and computer modelling. Resources to support controlled human studies of wound healing and interventions are limited in part by a perception that the area is mundane. There are, however, many gaps in our basic understanding of what are quite fundamental processes. Cell-based therapies are expensive, but will continue to offer the most rationale wound management solutions until our understanding is more complete. Longitudinal cost-benefit analyses of novel therapies remain few and far between.

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CHAPTER 2

Basic skin flaps and blood supply

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Introduction

Flap surgery is the commonest procedure in plastic surgery and is the essence of the discipline. Critical to its success is an understanding of the soft tissues' blood supply and its compliance and mobility. As all flaps 'rob Peter to pay Paul' it is also about conceptualizing the secondary defect and minimizing its consequences. The art and craft of plastic surgery necessarily requires an aesthetic sense and experience.

Evolution

For almost a century Manchot¹ and Salmon's² detailed studies of the skin's vascular design were overlooked by clinicians. With limited understanding and the simplistic belief that the skin's blood supply was based on a random distribution in the horizontal plane, local flap surgery was unpredictable and its progress curtailed by an adherence to dogmatic rules such as length-to-width ratios and the superiority of proximal over distally based flaps. A generation of surgeons failed to appreciate the simple observation that circumferentially incising large skin lesions in the process of their elevation and removal did not compromise their circulation. The explanation, of course, was that their blood supply was derived from the depths and not horizontally. Surgeons no doubt were aware of the circulation from below but the reality was that sufficient numbers of these vessels had to be divided to permit the flaps to transpose or rotate, and ultimately it was the base fed by the horizontal input that was the critical lifeline. Not until it was shown that flaps could be completely islanded and still live was it possible to move flaps based on these deep vessels. In 1970, Milton elegantly highlighted the fallacy of the length-to-breadth ratio using pig studies to demonstrate the existence of arterialized zones of the integument that would survive over extreme lengths even if completely islanded, provided they retained their arterial source at their base.³ These exciting findings breathed life back into the clinical study of the soft tissue's blood supply. True to

the adage that history is written by the victors, the blind acceptance of random patterns proposed by Harold Gillies⁴ was derived in part from the neglect of the publications of the Dutchman Johannes Esser (1917), Gillies' plastic surgery counterpart for the German army in World War I.⁵ Instead of the complex tube pedicle, Esser performed one-stage 'arterialized biological island flaps' based on the palpable arteries of the head and neck region. He clearly recognized the fundamental concepts of the flap's axial blood supply.

The first clinical application of the new 'axial pattern' concept was the groin flap, based on the superficial circumflex iliac branch of the femoral artery.⁶ The wide arc of rotation of this very long and narrow-based flap of like tissue expanded the single-stage reconstructive options for the regional wounds previously manageable only by multistage transfers requiring protracted immobilization and hospitalization.

What had been anecdotally reported in the early literature and had been empirically adopted in practice, the Indian forehead flap for nasal reconstruction and the epigastric flap in the lower abdomen, now made sense.⁷ The former flap unwittingly captured the supratrochlear/supraorbital vessels and the latter the superficial epigastrics.

With this new awareness, omentum, although not skin, was quickly recognized for its application as an arterialized flap by virtue of it wearing its blood supply on the outside of its surface. The vasculature could be pruned to critical arterial pedicles and tunnelled from the abdominal cavity to cover far-flung defects and then skin grafted.

Other fundamental concepts of skin blood supply were soon elucidated, such as the myocutaneous flap, where the skin relied for its blood supply on vessels perforating through the underlying muscle. Providing the muscle was raised on its blood supply, the overlying skin would survive even when completely islanded. As muscles typically are vascularized often by a single source at their origin, the muscle pedicle added even further length to the arc of flap rotation. The gracilis⁸ and latissimus dorsi myocutaneous flap,⁹ reinventing the earlier findings of Tansini,¹⁰ were the first to be described

and widely adopted. Muscle flaps without skin followed and the anatomical articulation of their vascular basis further expanded the options for one-stage locoregional reconstructions (Figure 2.1).¹¹

Fasciocutaneous flaps recognized for the first time that a significant contribution to skin blood supply was in the plane of the fascia and provided this fascia was included within the flap, large areas of previously unreliably vascularized skin would survive.¹² This particularly applied to the lower limb. Initially they were designed on the assumption their vascularity was in the plane of the fascia and in the limbs flaps were based proximally to capture its source. It was soon clear that much of this fasciocutaneous blood supply derived from perforating branches of named deep vessels emerging vertically between septofascial muscle planes from deep axial vessels, and furthermore these zones could be completely islanded from their proximal connections. Mathes and Nahai have classified fascia and fasciocutaneous flaps into three types: type A, direct cutaneous pedicle; type B, septocutaneous pedicle and type C, musculocutaneous pedicle.¹¹

This led to the unifying concept of angiosome blood supply, where the tissue is considered as a three-dimensional territory or somite structure, akin to vertebrate embryological development, where not only skin but whole mesenchymal somites including skin, muscle and bone have a vascular zone.¹³ This was supported by meticulous cadaver studies and expanded the earlier work of Manchot and Salmon.

Concurrent with this explosion in the understanding of the skin's blood supply, advances were being made in microvascular surgical techniques and instrumentation, initially by the neurosurgeons,¹⁴ but quickly adopted for plastic surgery. These techniques had immediate applications for replantation, but the possibility of transplanting toes and territories of skin by anastomosing specific vessels was now opportune. The

first such flap was an omental transfer to the scalp.¹⁵ Skin flaps soon followed.^{16–18}

Current understanding of the blood supply to skin

Flap surgery involves the transfer of tissue with its vascularity preserved. This requires an understanding of the physiology and anatomy of the integument's blood supply.

Physiology of the skin's blood supply

The blood supply (12.8 mL/100 g/min) to the skin greatly exceeds its metabolic needs because of its homeostatic role in thermoregulation. Perfusion of the skin's capillary beds is regulated by both local and systemic neurohumoral mechanisms. These act on precapillary sphincters and arteriovenous anastomoses, influencing the filling and emptying of the dermal plexuses and in turn not just the circulation of the skin and subcutaneous tissue, but also insensible heat loss and venous return to the heart.

Immediately after elevation of a skin flap, perfusion is transiently reduced by transection of blood vessels, inflammation, a hyperadrenergic state (associated with sympathectomy) and possible reperfusion injury. With no pharmacological means to reliably manipulate these effects at the capillary level, unless the design and execution of a flap includes an adequate circulation to overcome this ischaemic phase the flap may fail.

Anatomy of the skin's blood supply

The circulation of the skin and its underlying structures consists of a three-dimensional continuous vascular network. Conceptually this can be broken into *horizontal* and *vertical* components. Running parallel with the skin, and constituting

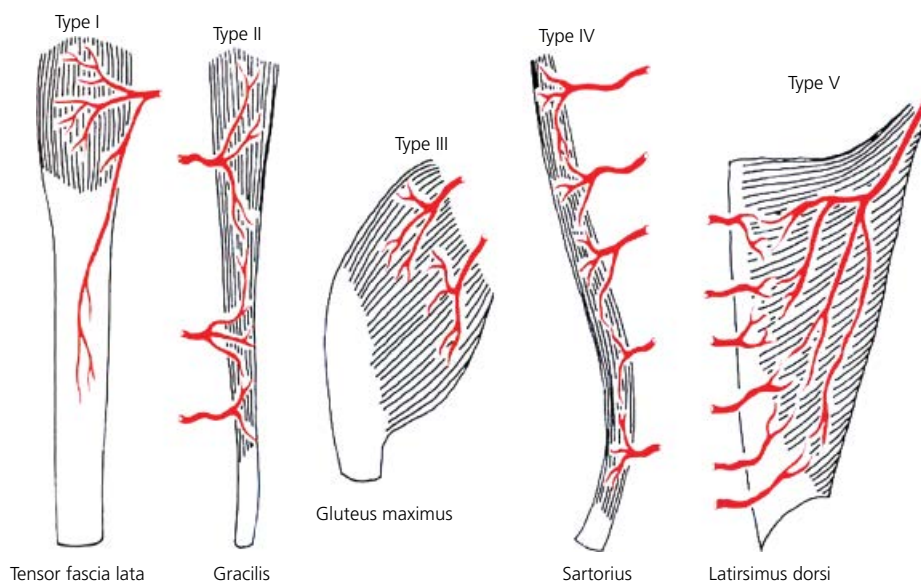


Figure 2.1 Mathes and Nahai classification. Patterns of vascular anatomy: type I, one vascular pedicle; type II, dominant pedicle(s) and minor pedicle(s); type III, two dominant pedicles; type IV, segmental vascular pedicles; type V, one dominant pedicle and secondary segmental pedicles. Source: Mathes and Nahai, 1981.¹¹ Reproduced with permission of Lippincott Williams & Wilkins.

the horizontal component of the skin's blood supply are the numerous vascular plexuses (Figure 2.2a). Most important of these are the subdermal plexus and the deeper suprafascial plexus. Where no deep fascia exists, an equivalent structure such as the panniculus carnosus (e.g. platysma, palmaris brevis and the dartos muscles in humans) serves a similar purpose. These are the vascular bases of (random) cutaneous and fascio-cutaneous flaps (Table 2.1).

Vessels arising vertically from their source arteries either *directly* supply the skin, or *indirectly* supply the skin after nourishing deeper structures such as muscle and bone. These are known as 'perforators' and they arise from the deep named vessels, along their axial course, with highest density over the less mobile soft tissue that is adherent to underlying septa. This is particularly evident in the limbs. Perforators anastomose initially with the prefascial plexus before continuing on to the subdermal plexus and are the vascular pedicles on which islands of skin and other soft tissue components may be based (Figure 2.2b). It follows that separate islands of skin and fascia, each based on a separate perforator but ultimately deriving from a common arterial axial vessel, can be raised on this common axis. This permits complex reconstructions with multiple independently oriented flaps.

The blood supply of the skin and its underlying structures can also be divided into vascular territories, or *angiosomes*.¹³ Each territory is connected to its adjacent territory by bidirectional arterioles, the direction being interchangeable and determined by the relative pressure in each territory. The angiosome is the vascular basis for composite flaps.

Drainage of the skin is by a reciprocal three-dimensional venous network of avalvular bidirectional veins with a dominant subdermal component. This in turn drains into large-calibre subcutaneous veins or *venae comitantes* that run with perforators. Superficial lymphatics follow subcutaneous veins and deeper lymphatics follow arteries.

Venous flaps are generally transferred as free microvascular flaps, where the flap is elevated superficially with only the venous system, thereby obtaining a thin flap and reducing morbidity.¹⁹ The flap is arterialized through its veins by anastomosing them to an artery in varying configurations, the tissue being nourished by retrograde perfusion. Understandably, their reliability is inconsistent.

Indications for flaps

Skin defects should ideally be repaired by replacement of what is missing and generally this will include fat as well as skin. In many cases the local laxity of skin will allow direct closure and conversion of the deformity to a linear scar. Sometimes, however, although the wound can be technically closed directly this may create a dish deformity with dog-ears at either end. Removal of these dog-ears only aggravates the underlying problem of missing tissue. Here, well-designed local flaps from redundant areas can redistribute the tension so as to preserve available tissue and restore normal contour.

Defects that cannot be closed directly will need grafting or replacement with flaps. Skin grafts take by engaging with the

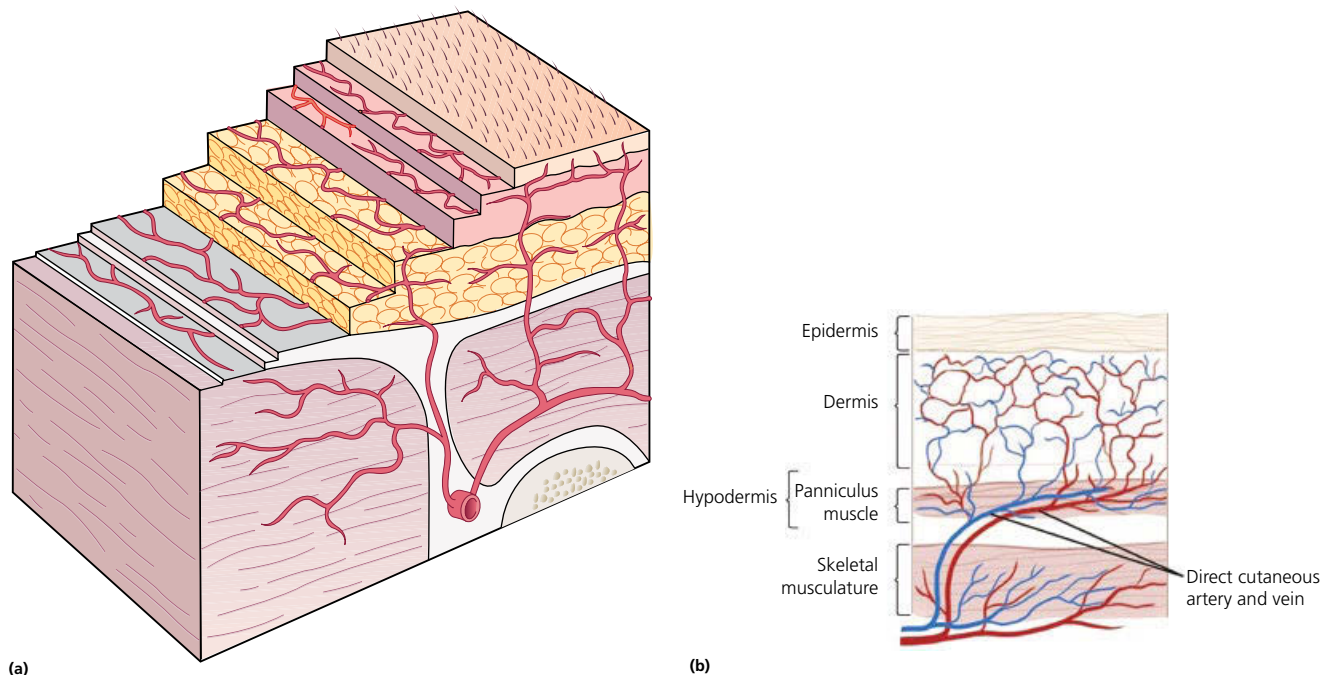


Figure 2.2 (a, b) Illustration showing subdermal plexus and suprafascial plexus. Also perforators.

Table 2.1 Classification of flaps based on movement and blood supply

Movement	Blood supply
(a) Local skin flaps	
Advancement	
In-continuity – rare	Random – intact skin base
Islanded (e.g. V–Y)	Random – subcutaneous base
Keystone	Perforator base
Transposition	
In-continuity (e.g. rhomboid, bilobed, Z-plasty)	Random – intact skin base
Islanded (e.g. propeller)	Perforator base
Rotation (e.g. scalp)	Random; arterialized
Combination	
(b) Regional (locoregional) flaps	
Transposition	
Cutaneous (e.g. groin flap, forehead, rhinoplasty, Abbe lip flap)	Subcutaneous arterialized
Muscle/myocutaneous	Arterial pedicle
Fasciocutaneous	Septo(myo)fascial perforators
Neurovascular island flaps (e.g. digits)	Neurovascular A–V pedicles
(c) Distant flaps	
Two-stage pedicle (e.g. cross finger, groin to hand)	Random; arterialized
Multistaged ‘waltzed’ pedicle (e.g. tube pedicle)	Random
(d) Free flaps	
Arterial	Arterial – microvascular anastomosis
Venous	Venous blood supply
(e) Prelamination and prefabrication	
(f) Allotransplantation –	
Opportunistic free flap allograft in patient simultaneously requiring organ transplant (e.g. abdominal wall with bowel transplant)	
(g) Tissue engineering –	
Although prefabricated and prelaminated flaps were the early prototypes of tissue engineered flaps, cell-seeded artificial scaffolds and/or adipogenic matrices are being investigated	Experimental

vasculature of the underlying bed. They are inappropriate in avascular circumstances (exposed bone, tendon, fracture sites, irradiated tissue and mobile beds) and here flaps are required as they possess an independent blood supply. Grafts often contract and may be a poor colour match; where the underlying fatty tissue is missing, they may result in contour defects and adherence to deeper tissues. Apart from burns and extensive injuries where large areas of skin are required it is generally accepted, particularly with the wide range of flap options available, that skin grafts are inferior to flaps and are rarely first choice. Other exceptions include the dorsum of the hand and foot, where thin skin is required. Few flaps meet these needs. Full-thickness skin grafts have an important place on the face, where the tissues are thin with little subcutaneous fat (eyelids, inner canthus,

proximal nose and sometimes nasal tip). Flaps from regions adjacent to these sites are invariably too thick.

Elsewhere, flaps are indicated. Small defects are closed by local flaps from the immediate area and find their greatest expression in the head and neck region, particularly in association with skin cancer resection. Flaps may be in-continuity (transposition, rotation) or islanded (advancement), and their execution defines the quality of the plastic surgeon. Because of their near-perfect tissue match the well-executed local flap may be difficult to spot. Larger defects will require locoregional flaps from a distance and are most likely transposed on their narrow base to maximize their reach (arc of rotation). Because their skin texture, colour and thickness may not match that of the original defect they may require subsequent revisional surgery. Such distant flaps may be fasciocutaneous, myocutaneous or muscle flaps with skin grafts. Composites of tissue may be included – muscle or tendon, fascia or bone – allowing functional reconstruction of complex defects. Their vascular basis will be on the vascular pedicle alone or together with their associated skin, muscle or fascial carrier respectively. Usually the secondary defect will be directly closable.

For very large skin grafts or complex defects where a specialized tissue is needed, such as functional muscle or bone, innervated or hair-bearing skin, free flaps are indicated. These are based on a vascular pedicle and require microvascular anastomosis. Prefabricated (arterialized zone of specialized skin created by the implantation of a vascular pedicle so as to render it transferable as a free flap suitable to match a specific defect) and prelaminated flaps (the neovascularization of composite tissue around a vascular pedicle for subsequent transfer) are more sophisticated free flap indications. Tubed pedicle flaps are now rare but still find applications where there is an absence of recipient vessels at the defect site (Figure 2.3).

Design and application of flaps

Two concurrent considerations are critical to the successful planning of local flaps: (1) blood supply and (2) availability of adjacent mobile tissue (laxity).

Flap design and blood supply

The first consideration in designing a skin flap is to determine whether it will be viable. The vascular limitations on a flap's dimensions are not completely understood, though the principles to follow are helpful. Some flaps in some sites seem more predictable than others, despite the principles, and confidence in local flap surgery comes with trial and error, and ultimately experience with what works and what does not.

The simplest skin flaps are based on the subdermal plexus, the richness of which varies around the body (Figure 2.4). Vague length-to-breadth ratios govern the size of these so-called *random flaps*, with the only certainty being that a flap whose length equals its width will be viable anywhere on the body.



Figure 2.3 Tubed pedicle flaps are largely historic. This patient with osteomyelitis in his leg underwent a tubed pedicle flap from the abdomen using the hand as a carrier. Source: Dr Edwin J. Morrison. Reproduced with permission.

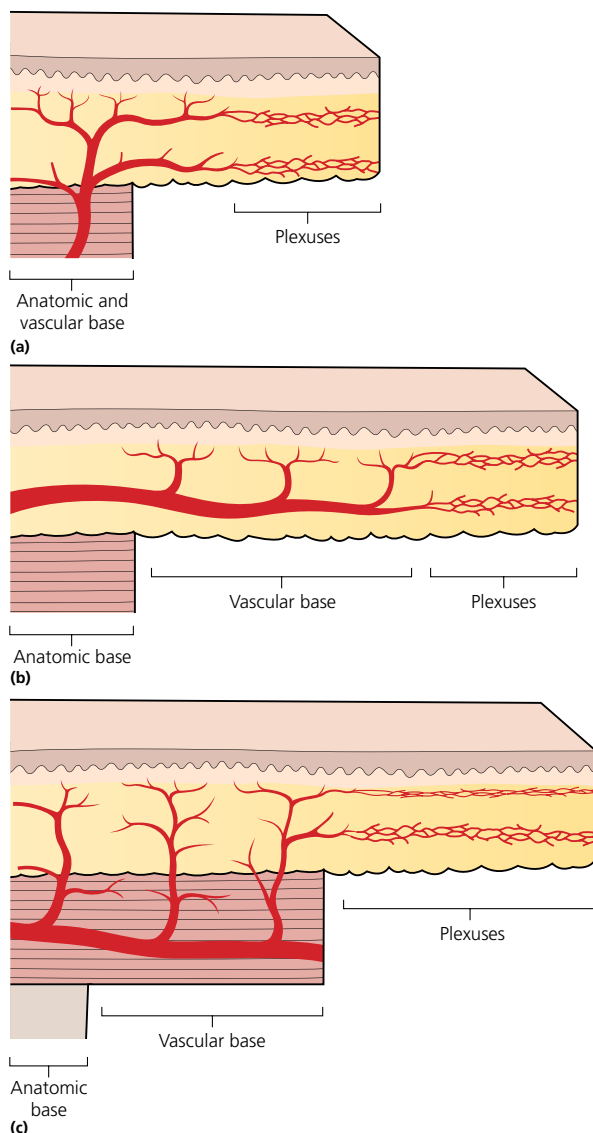


Figure 2.4 (a) Random flap; (b) axial flap; (c) musculocutaneous flap. Source: Dr Edwin J. Morrison, Department of Surgery, University of Melbourne. Reproduced with permission.

Because of the markedly increased vascularity of the face, such flaps can tolerate significantly higher length-to-breadth dimensions.

The area of conventional flaps can be increased by capture of an additional vascular component. Axial flaps, which have a known arterial pedicle coursing along their axis in the superficial plane (e.g. groin flap, forehead flap), may be further enlarged in a random fashion, or by incorporating some of the skin supplied by the next cutaneous perforator in the adjacent angiosome.^{20, 21} Skin flaps elevated with the deep fascia and therefore with the suprafascial plexus intact create a *fasciocutaneous flap*, which can also be elevated with significantly higher length-to-breadth dimensions. Musculocutaneous flaps are yet another variant.

Length-to-breadth ratios of random flaps and the volume of tissue in axial flaps can also be increased by harnessing the *delay phenomenon*. Although its mechanism is incompletely understood, *vascular delay* is a well-established surgical technique capable of improving the vascularity and reliability of a flap prior to its definitive elevation and inset.²² It involves the strategic division of the flap's blood supply so as to deliver a sublethal ischaemic insult. This is postulated to cause vessel dilatation, ischaemic preconditioning and neovascularization. It may also be beneficial in routine flap surgery on high-risk patients who are obese, smokers or who have undergone previous irradiation.

Flaps incorporating the horizontal component of the integument's blood supply are usefully classified as *flaps in-continuity*. For similarly obvious reasons, flaps that are circumferentially incised through dermis or deep fascia (or its equivalent) are *island flaps* whose vascular supply is through perforators (Figure 2.5).

Apart from the native vascularity, caution needs to be taken when flaps are folded to repair complex defects or pass over convex surfaces. Similarly, tight closure when flaps are too small and failure to recognize previous scars in the region may compromise vascularity. Haematoma is perhaps the greatest enemy of local flaps, because of increased tension and the inflammatory toxicity of the underlying blood clot over time.

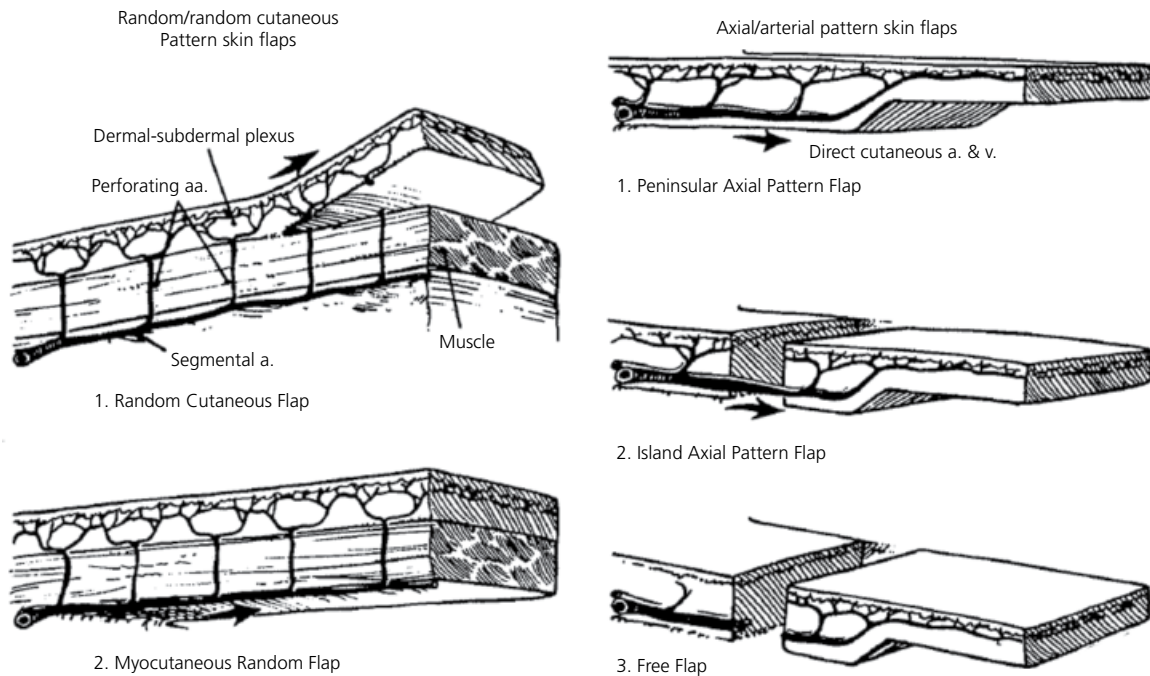


Figure 2.5 In-contiguity versus island flaps. Source: Daniel & Kerrigan, *Principles and Physiology of Skin Flap Surgery*. In: McCarthy, *Plastic Surgery*. Elsevier, 1990. Reproduced with permission.

Flap design and movement

Having determined that flap tissue is available and that it will be viable, the next question is how it can be moved to cover the defect. Flaps move by way of *transposition*, *rotation* or *advancement* though in reality their movement usually involves some combination of all of these and is often aided by initial partial direct closure of the defect itself.

The first principle of local flap design is to pinch up the skin immediately adjacent to the defect and serially march around its perimeter until the zone of maximally lax skin is detected. It is from this tissue that the flap will be elevated. Because the tissue is lax, it follows that the secondary defect should close directly.

Flaps in-contiguity

Most local flaps are totally elevated from their bed but retain a critical skin base for their vascularity. This especially pertains to transposition and rotation flaps.

Transposition flaps

Lax tissue immediately adjacent to the defect is elevated and transposed, sometimes across intact skin bridges. This creates a secondary defect that is usually closable directly because of the laxity.

The *rhomboid flap* is a particularly useful example of such a flap.²⁴ It is commonly used in skin cancer surgery and the principles can be applied to all transpositions. The anticipated defect is conceived in the form of a rhomboid (unequal parallelogram). A diagonal across the rhomboid is extended the same distance as the length of an adjacent side of the defect into the zone of laxity (a through b to c). A diagonal across the rhomboid is extended the same distance as the length of an adjacent side of the defect

into the zone of laxity ($ac = ce$). A line is then directed backwards from the limit of this point (e) parallel with the side of the defect (ef). The flap thus outlined is elevated and transposed into the defect. Preoperatively if point (f) is estimated to be able to approximate to point (c) then the secondary defect will close (see Figure 30.4a). Selecting the shorter diagonal generally facilitates flap closure because the flap requires less angulation. In some situations the adjacent tissue has not enough laxity to permit closure of the secondary defect and a second flap from the more lax tissue adjacent to this defect will close the tertiary defect (bilobed flap). When flaps are used to cover exposed bone or other non-graftable defects, transposition flaps must be used even if the adjacent zone will not permit direct closure, and here skin grafts are used for the secondary defect (Figure 2.6)

The other common application of the transposition flap is in association with the *Z-plasty*, a fundamental design concept in plastic surgery (Figure 2.7). Z-Plasties are used to release (lengthen) tight or webbed scars, redirect scars into a more favourable alignment and, in combination with linear access incisions, to prevent secondary linear contracture (e.g. Dupuytren disease). The traditional Z-plasty design involves an axial incision along the course of the scar. A back-cut extends from one end of the linear incision into the adjacent tissue at 60° for the same length as the axial incision. A second back-cut the same length is now made on the opposite side of the line, commencing at the other end of the line and directed to the first. This creates an equal-limbed Z incision with its diagonal in the axis of the original scar and flaps at 60° . By interdigitating (transposing) the two flaps created by the design across the midline, the diagonal will now change at right angles to the original



Figure 2.6 The rhomboid flap design, execution and outcome for a temple defect. Note the pinch test and the attempt to position the secondary scar in the natural crease lines of the forehead. Source: Grabb & Smith, 1991.²³ Reproduced with permission of Lippincott, Williams & Wilkins.

axis. This way the scar is partially redirected and, provided the planning is accurate, it should ideally fall within an existing crease line. It is estimated that with a 60° angle transposition the original length of the scar axis will increase by 75% (angle proportional to change in length).

To achieve maximum correction of tight scars it is essential to completely remove the entire underlying scar so that the contracture is fully released. Often it is unrealistic to close 60°

flaps, and smaller angles are required at the expense of lesser lengthening. In fact, when there is not much contracture, the flaps are used to redirect scars rather than increasing length, which is undesirable as it will simply create large redundant dog-ears.

As with all flaps, tissue is borrowed from one place to give to another. Z-Plasties lengthen at the expense of reduction in width. They are no different to other local flaps in that if there is adjacent redundant tissue they work well. This is particularly so

with contracted linear scars where the surrounding tissue is not involved. This tissue has been locked or gathered up in the scar but is not missing, and on release it is available for

transpositioning and scar lengthening. By contrast, where wide scarring has occurred, such as burns, the Z-plasty is essentially cheating by taking tissue from one direction only to create tightness in another. Here, imported skin is needed to replace what is lost either as a graft or more distant flap.

Rotation flaps

These are used in circumstances where there is no differential laxity in the immediate region of the defect. An exaggeratedly large flap is required, extending well beyond the zone of the defect to recruit tissue progressively over a large distance. Rotation flaps are typically employed over the scalp and upper cheek zones. Generally, in designing these flaps it is essential to understand the concept of the pivot point (see below). Their design involves triangulation of the defect, the shortest side being selected as the base (Figure 2.8).

The side of the triangle, which becomes the leading edge of the rotation flap, will advance to meet its opposite side by mobilizing all that tissue beyond the defect sufficient to allow movement. This is achieved by a sweeping curvilinear incision extending from in line of the triangle base over a wide expanse until it encroaches into the zone of laxity. The whole of the large flap is elevated completely up to its base and rotated around its pivot point until the incised edge of the flap rotates forward to close the defect. The base of the flap is that remaining bridge of tissue from the apex of the triangular defect to the point where the flap incision ceases. Most rotation flaps survive on a random blood supply but in the scalp known arterial axes should form the basis of the design and in these circumstances much narrower bases can be conceived.

Because of the spherical shape of the skull, skin flap design will need to accommodate changing lengths and mobility during flap rotation. The secondary defect should close directly by progressive recruitment of tissue along the whole course of the incision or if a back-cut was done to relieve tension on the closure, tissue from the opposite axis should be chosen.

Pivot point

In both transposition and rotation flaps the pivot point is a crucial technical consideration. It always lies at the base of the flap furthestmost from the defect and is the point at which the flap is

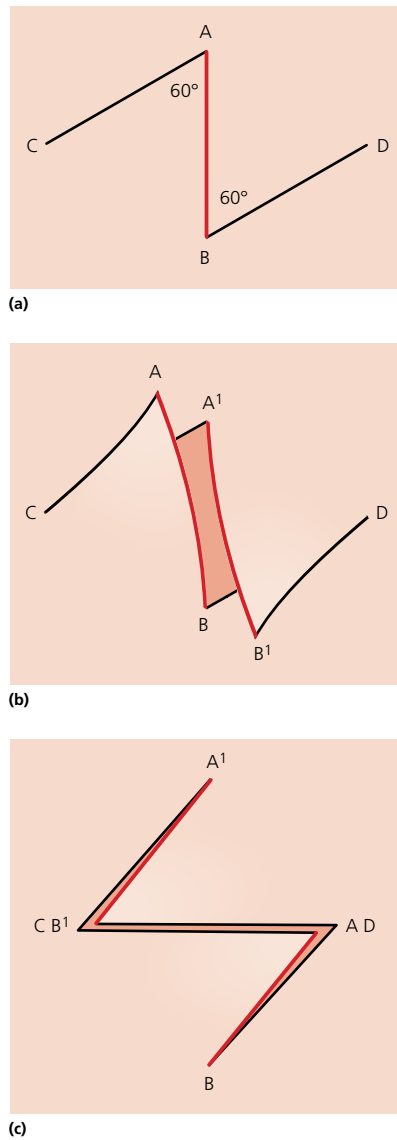


Figure 2.7 Z-Plasty.

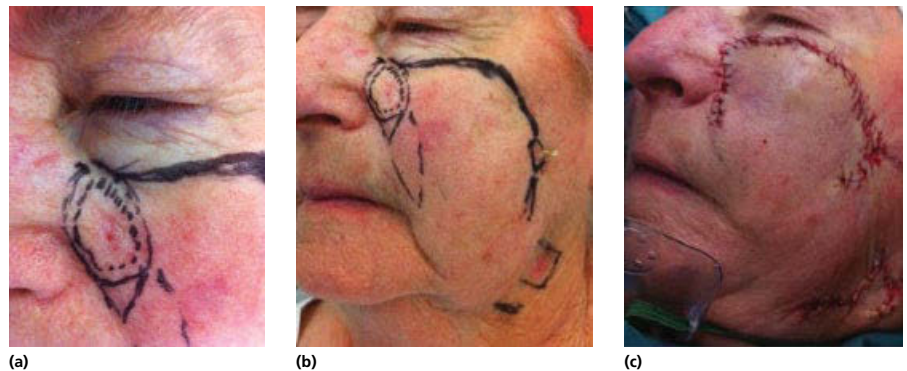


Figure 2.8 Cheek rotation flap.

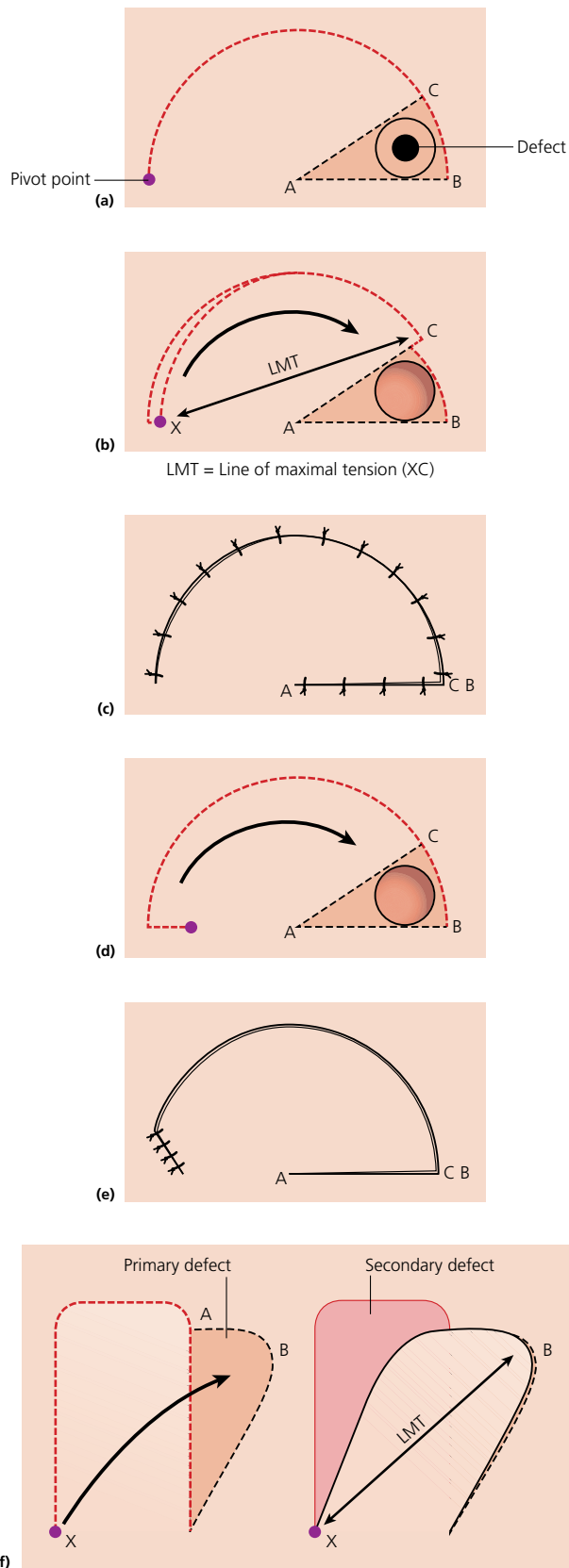


Figure 2.9 Pivot point of rotation and transposition flaps.

tethered, preventing further movement (Figure 2.9). Careful planning is necessary to avoid a flap being too small for the defect or becoming ischaemic with closure under tension. A *back-cut* can reposition the pivot point somewhat closer to the defect but comes at the expense of the flap's vascularity. *Planning in reverse* is a useful technique to determine the necessary dimensions of a flap preoperatively.

Advancement flaps

Lax adjacent tissue can be directly advanced into the defect. Realistically, tissue will not advance much more than its capacity for direct closure and to gain real movement requires islanding of the advancement flap. Such flaps then become dependent on subcutaneous or perforator blood supply.

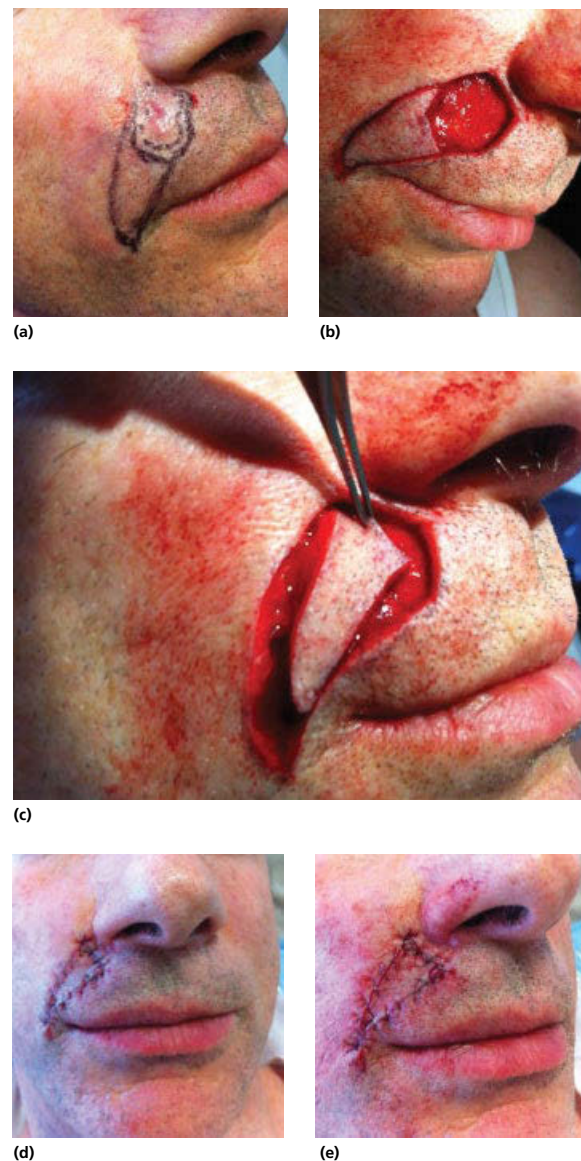


Figure 2.10 (a, b, c, d and e) V-Y flap to upper lip.

Island flaps are mostly advanced into the defect, though in some instances they may be rotated or transposed as in perforator flaps. The classic island flap design is the *V-Y configuration* (Figure 2.10). In general, island flaps tend to be more mobile compared with those in continuity. There are also varying degrees of islanding. Circumferential incision of the dermis alone leaves the deep fascia (or equivalent) intact and in the more mobile parts of the body, such as the face, blunt dissection of the subcutis will usually suffice to achieve the desired amount of movement. Where the overlying skin is more adherent to the deeper structures, as occurs in the limbs and trunk, judicious release of the deep fascia is required. Complete islanding of the suprafascial plexus may not be necessary in every case and a limited release of tethering structures should be trialled initially. A commitment to islanding requires certainty that a perforator lies at the base of the flap.

Finally, consideration needs to be given to the inset of the flap so as to camouflage its outline. To this extent, where possible the flap's axis and its margins should be parallel to crease lines and inset respecting cosmetic units. V-Y advancements along the nasolabial and glabella crease lines expound this concept. A flap design is less conspicuous with straight or gently curving margins than round flaps, as circular lines are unnatural on the face and are instantly recognizable. Respecting this principle it is often better to resect a circular skin tumour as a square rather than simply as a circle. Taken further, consideration can be given to resecting and replacing whole cosmetic units to improve cosmetic outcomes. Well-designed larger flaps are often preferable to circular flaps, which are prone to pin-cushioning as a result of cicatricial contracture. Scars crossing natural crease lines are conspicuous, and across hollows will cause tenting or webbing. A further principle of flap repair is that a single flap cannot realistically repair a defect that transgresses two separate planes (e.g. cheek-alar base). In these cases two flaps from different axes or a flap and a graft will be required.

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CHAPTER 3

Biomaterials and structural fat grafting

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General principles

Living cells respond to physical and biochemical cues (e.g. hormones, cell–cell signalling molecules and the physical environment) through the extracellular matrix (ECM), which enables adaption to changes in the physiological environment. Regulation of cell function (growth, migration, proliferation and differentiation) is dependent upon contact or adhesion between cells and the substrate (ECM). ECM has three key components: collagen (the structural framework), proteoglycans and adhesive matrix proteins (e.g. fibronectin, vitronectin). Initial contact of cells with a solid substrate is mediated by adsorbed adhesive proteins. Cell surface receptors form bonds with these, leading to cytoskeletal reorganization and progressive spreading of the cell on the substrate. These receptors transduce biochemical signals to the nucleus by activating the same intracellular signalling pathways that are used by growth factor receptors.¹ Next to biochemical cues, mechanical forces play an important role in cell adhesion, growth and possibly differentiation. These responses are mediated through internal cell receptors that respond to mechanical forces. Cell binding to ECM through specific cell–substrate contacts is critical to cell growth control through mechanical forces, resulting in alterations in cell shape and cytoskeletal tension.¹

A biomaterial is a non-pharmaceutical, non-viable material in intimate contact with living tissue used to treat, augment or replace any tissue, organ or function of the body. Ideal properties for a biomaterial include: biocompatibility, non-carcinogenicity, inertness, lack of allergenicity, cost-effectiveness and ease of handling. Biomaterials or tissue engineering scaffolds contain chemical and structural information that may control tissue formation, in a manner similar to cell–cell communication and cell–ECM interaction (Figure 3.1). An appropriate 3D scaffold should facilitate normal cellular organization and behaviour, define and maintain the desired tissue volume, and at the same time promote host integration and implant vascularization.²

Ultimately, the scaffold should undergo non-toxic degradation as it is replaced by the healthy host tissue. Hence, a

biodegradable scaffold is ideally used in tissue engineering. Understanding of the cell–biomaterial interface is important in the choice of scaffold due to a direct impact on cell adhesion – an important step in the survival of anchorage-dependent cells. Both cell proliferation and differentiation can be controlled by functionalization of the biomaterial surface. Multiple studies have shown that the interactions between cells and biomaterials occur at the nanoscale.^{3–5}

With the advent of nanotechnology, nanoparticles are being merged to synthetic scaffolds to develop nanostructured biomaterials and enhance interaction of proteins that control cell adhesion and, thus, tissue formation. Nanocomposites can be defined as multiphase solid materials where one of the phases has a dimension of less than 100 nm. Nanofibrous composite scaffolds are found to decrease immunogenicity, improve the capacity for cell interaction,⁶ and to harbour increased concentrations of fibronectin and vitronectin, the adsorption proteins that reduce apoptosis of transplanted cells.⁷ Biomaterials can be classified as bioinert, bioresorbable or bioactive. A *bioinert* material does not cause any toxic response from the body on implantation and is usually associated with fibrous encapsulation, whereas a *bioresorbable* material undergoes degradation in the body by hydrolysis, enzymes or osteoclasts. *Bioactive* materials produce a biological response from the body that results in a bond between the material and the host tissue. Type and site of biomaterial implantation governs tissue response: hard tissues (bone, enamel and dentin), soft tissues (breast and ocular implants), vascular implants (stents, heart valves and vascular grafts) and connective tissue (cartilage, tendons and ligaments).

A variety of possible scaffold biomaterials are available. Among these are biological polymers (e.g. collagen, elastin, chitosan, silk), biodegradable synthetic polymers (e.g. PGA, PLA, PDLA), synthetic biomimetic compounds (e.g. hydroxyapatite, calcium phosphates), allograft and xenografts, bioactive glasses or combinations of the above. When choosing and designing a biomaterial several factors should be taken into consideration. The most important feature of a biomaterial should be biocompatibility (lack of toxicity to the tissues). In addition, the

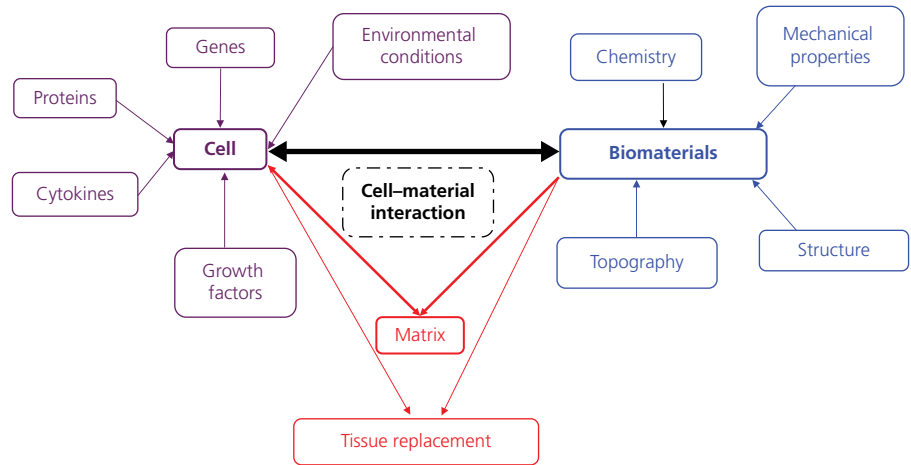


Figure 3.1 Cell and biomaterial interactions and factors leading to tissue regeneration.

mechanical properties of a material are important, as they provide structural support, influence cell behaviour, and determine the degree of wear and stress shielding. Geometric variables such as porosity, pore size and pore morphology are also important. A porous scaffold provides a large surface area for neovascularization, nutrient and waste exchange, cell migration and matrix deposition. Scaffolds can be made porous using several different methods. Porogens (i.e. salt or paraffin microspheres) could be incorporated in the polymer and subsequently leached out. Freeze drying of homogenized polymer solvents is another option, in addition to gel casting of organic scaffolds and sol-gel foaming. Surface properties, such as surface topography and chemistry, guide cellular behaviour and control protein adhesion and should be considered as well. In the case that a biodegradable material is chosen for implantation, the rate of resorption should match the speed at which the body replaces the missing tissue and its degradation by-products should be non-toxic. Finally, the biomaterial of choice should be economically viable and conform to the regulatory bodies.

Autologous

The word autologous means ‘related to self’ and refers to tissue that has been derived or transferred from the same organism. Autologous grafts, also referred to as ‘autografts’, can be harvested and transferred from several different tissues in the body (e.g. skin, bone, tendon, cartilage, etc.). The focus of this section is on the autologous transfer of skin, adipose tissue, bone and cartilage.

Skin autografts

From superficial to deep, skin is composed of epidermis, dermis and skin appendages. It has several functions, including physical protection, prevention of fluid loss, protection against ultraviolet radiation, regulation of body temperature, sensation, immunological surveillance and protection from microbiological organisms. Injured skin can heal itself through different phases of wound healing and epithelialization, whereby

epithelium is re-established across a wound. Different phases of wound healing consist of haemostasis, inflammation, proliferation and remodelling, whereas epithelial repair consists of mobilization and migration of epithelial cells, followed by mitosis and differentiation into stratified squamous epithelial cells. If the injury to the skin is grave or the area of skin loss large, this process may take several months and result in significant scarring. In this instance the use of a skin autograft can aid the healing process. Skin graft can either be split or full thickness. Split skin grafts (SSG) contain a variable amount of dermis and can be harvested from several sites (i.e. thigh, buttocks and scalp). The donor site heals through epithelial repair and does not require closure. Full-thickness skin grafts (FTSGs) contain the entire dermis and are usually taken from areas of the body with enough skin laxity to allow direct closure (i.e. groin, forearm, arm, post-auricular and supraclavicular areas).

Skin grafts heal in four phases:

- 1 **Adherence** of the graft to its bed via immediately formed fibrin bonds.
- 2 **Serum imbibition** Skin grafts swell in the first 2–4 days after application due to absorption of fluid.
- 3 **Revascularization** starts on about the 4th day and is a consequence of new vessel formation along new and existing vascular channels in the graft.
- 4 **Remodelling** is the process whereby the histological structure of the graft returns to that of the normal skin.

Skin grafts can fail to ‘take’ for several reasons (e.g. haematoma, infection, excessive shearing, an inappropriate bed, or technical errors such as placing the graft upside down on its bed or allowing it to dry before application).

The main drawback of skin grafts is the fact that they contract. Contraction of grafts is more pronounced with split-thickness skin grafts compared to full-thickness skin grafts.

Adipose tissue autografting

Although autologous fat transfer has gained popularity over the last two decades, Neuber first described the practice, using free autologous fat graft, in the medical literature in the

1890s.⁸ The injectable fat transfer was first described in the 1920s. In the 1950s Peer reported fat grafts losing approximately 45% of their weight and mass per year. He emphasized the importance of a good vascular recipient bed and meticulous haemostasis to ensure optimal graft survival.⁹ Autologous fat transfer can be regarded as the ideal filler. There is no host reaction or carcinogenic response. Being minimally invasive, the procedure is safe and fat is readily available and inexpensive.¹⁰ Limitations include unpredictable and variable rate of absorption, the need for overcorrection and the fact that it involves a surgical procedure. Practitioners must keep up to date with the latest development in harvesting, preparation and application of autologous fat transfer to ensure optimal graft outcome.

Donor site

Currently there is no objective evidence to determine the best donor site for autologous fat harvest. The most common site is the abdomen due to availability and ease of access while the patient lies supine. Other sites include the flanks, extremities (inner thighs, inner knees, inner arms, trochanteric area) and gluteal area. In one study¹¹ the viability of fat cells was analysed *in vitro* by using spectrophotometry from four different anatomical sites: abdomen, medial knee, flank and inner thighs. No difference was found in fat viability 5 h after harvest.

Extraction techniques

Vacuum extraction, syringe aspiration and direct fat excision are well-described techniques in harvesting fat tissue. Although syringe aspiration is the most popular technique, currently there are no objective studies to confirm the benefit of this technique compared to other methods of harvest. However, high negative pressure exerted on adipocytes in conventional liposuction has been reported to cause close to 90% adipocyte rupture.¹² Coleman, who has popularized autologous fat transfer, uses the syringe aspiration technique by using a 3 mm cannula connected to a 10 mL syringe, and aspiration is achieved by manual suction through drawing the plunger.¹³

Infiltration with local anaesthetic agents

Some clinicians do not use any local anaesthetic for fat harvest but most inject the donor site with short-acting local anaesthetic agents with or without adrenaline (epinephrine). A commonly used agent is lidocaine (lignocaine) with concentration dose ranging from 0.5% to 2% and adrenaline ranging from 1 : 80 000 to 1 : 200 000. Coleman recommends using 0.5% lidocaine with 1 : 200 000 adrenaline. A study done by Moore *et al.* on the impact of lidocaine on adipocyte growth in cell culture showed an inhibitory effect on their growth, but cell recovery was displayed by washing cells up to 10 days post exposure.¹⁴ A major clinical advantage of infiltration with local anaesthetic combined with adrenaline is to reduce potential donor site pain, blood loss and bruising.

Preparation

Centrifugation is a popular method of fat graft preparation. The process separates the non-viable components, including blood, oil and lidocaine, leaving behind the adipocyte-rich segment for transfer. Coleman advocates 3 min of centrifugation of aspirated fat at 3000 rpm. This process separates the fat into a bottom layer of tumescent lidocaine and blood, a middle layer of usable fat graft and a top layer of oil from ruptured adipocytes.¹⁵ Other clinicians also advocate the use of centrifugation at different spinning rate and duration,¹⁶ but there is no objective evidence to support which is the best centrifugation method. Rohrich *et al.* have recently challenged the process of centrifugation and found no quantitative difference between the viability of processed and unprocessed fat graft viability¹¹ and Ramon *et al.* using a nude mouse model have demonstrated that fat cell survival after 16 weeks between centrifuged and non-centrifuged fat grafts in terms of weight and volume was the same. Histological analysis showed non-centrifuged fat grafts had less fibrosis.¹⁷

Washing harvested fat grafts as a form of preparation has also been described using lactate Ringer's solution to improve viability.¹⁸ The rationale is to reduce the inflammatory mediators from the graft and minimize an immune response at the recipient site. Currently there is no objective evidence in the literature to suggest washing the harvested fat graft improves overall graft survival.

Application

To maximize graft survival the adipocytes must be in close vicinity to a blood supply. Some suggest that fat placed within 2 mm of an arterial supply will survive and beyond that the risk of necrosis and fibrosis increases.¹⁹ Thus the surface area of fat graft in contact with a vascular recipient bed is essential to improve graft survival. The 'fanning out' technique is one method of injection that ensures deposits of fat graft are spread over a large surface area. Other commonly used injection methods, such as linear threading; serial puncture and cross-hatching techniques, can also be used to achieve the desired outcome. Curved microcannula 2–3 mm in diameter or blunt tip needle 14–19 gauge is commonly used for autologous fat transfer.

Complications

In general, autologous fat transfer is a safe procedure with a low risk profile. However, patients must be warned about bruising, swelling, contour irregularity and infection, along with fat necrosis and calcification. Rare cases of severe complications such as fat embolism of cerebral artery and retinal artery, leading to stroke and blindness, respectively, have been reported in the literature during soft tissue augmentation of the nasolabial fold and periorbital area.^{20,21}

During the consent procedure it is important to cover the common and rare risks of fat transfer along with the limitation of the procedure, which is mainly the unpredictability of the graft take and partial and complete absorption over time.

The rise in popularity of autologous fat transfer in addressing contour deformity and volume restoration over the last few decades has brought this technique under the spotlight. Many questions are still left unanswered on the most optimal donor site, the best method of harvest and preparation. Further objective randomized clinical trials need to be carried out to address these questions. However, this established technique that was first described over 100 years ago is here to stay and serves as a powerful tool in the armamentarium of practitioners in this field.

Bone autografts

The success of bone autografts in the treatment of non-unions is well established. In the tibia, union rates of more than 90% have been reported using iliac crest bone graft at a mechanically stable site.^{22–24} Similar success rates have been documented in the treatment of diaphyseal non-unions.^{25,26} In posterior cervical fusions, success rates in 92% to 100% of the patients have been reported when using autologous iliac bone grafts.²⁷ In addition, bone grafting has been successful in treating recalcitrant^{28,29} and infected^{30,31} non-unions as well as aiding the healing at the docking site of non-unions treated with distraction osteogenesis.³² Over and above the mechanical properties and volume effect, the biologic properties of bone autografts are of great importance.³³

Autologous bone graft continues to represent the gold standard for management of bone defects and non-unions. Bone grafts heal by the following mechanisms: (1) adherence to the surrounding tissue (i.e. incorporation), (2) creeping substitution, also referred to as osseointegration, a process whereby the bone graft acts as a scaffold along which angiogenesis occurs and new bone is formed by progenitor cells, and (3) osseointegration, the differentiation of mesenchymal stem cells into osteocytes. Autologous bone grafts possess biological advantages over allografts and synthetic bone grafts as a result of their excellent combination of osteogenic, osteoinductive and osteoconductive properties.³³

Autologous cancellous bone grafts

The trabecular structure of cancellous bone results in a large surface area. This allows for a high number of cellular components (mesenchymal stem cells and osteoblasts) to be incorporated and explains its excellent osteogenic and osteoinductive properties.³³ Additionally, the trabecular structure allows for easy revascularization and incorporation at the host site.³⁴ On the other hand, one of the limitations of cancellous bone is its poor mechanical properties. The formation of new bone at the site of a necrotic cellular structure weakens the construct within the first weeks. However, increased stability is achieved within months due to the biological capacity of trabecular bone graft to induce new bone formation, once it is incorporated.³⁴

Autologous cortical bone grafts

Cortical bone autografts have a more limited biological profile than cancellous bone autografts. Although cortical bone has fewer osteoblasts, osteocytes and growth factors, and less surface

area per unit weight, the structure of which constitutes a barrier to vascular ingrowth and remodelling, it provides good initial mechanical strength and stability.³⁵ Osteoclastic activity with resorption of the cortices and loss of bone starts at two weeks after the grafting.³⁶ This results in transient weakness with reduction of mechanical strength of up to 75%.³⁷ Experimental studies have shown that cortical grafts have significantly reduced strength at six weeks that remains low through 24 weeks but returns to normal by 48 weeks post implantation.³⁶

Autologous vascularized bone grafts

Autologous vascularized cortical bone grafts have favourable biologic attributes as compared with standard autologous non-vascularized autografts. In addition, they are mechanically superior during the initial 6–12 months after the grafting procedure. Despite their many advantages, their main limitation is that they are more technically difficult to harvest and their implantation requires orthopaedic and microvascular skills. They can also cause significant morbidity at the donor site. Vascularized cortical autografts heal rapidly at the graft-recipient interface as the healing/remodelling process is similar to that of normal bone.³⁴ It has been proposed that more than 90% of the osteocytes may survive the transplantation, if patent vascular anastomoses and stability of the graft are achieved.³⁷ Osteoneogenesis by graft and host can lead to expeditious incorporation of the graft, while the residual weakness of the construct remains minimal.³⁸

As a result of their excellent and cost-effective combination of biologic and mechanical properties, bone grafts continue to be an important tool in the management of bone defects and non-unions.

Cartilage autografts

Cartilage does not possess the regenerative capacity of bone and other connective tissues, due to its lack of vascularity. Cartilage is composed of chondroitin sulfate, a gelatinous ground substance, in which collagen and elastic protein fibres are incorporated. It is flexible, very durable and resistant to compression forces. There are three types of cartilage: hyaline, elastic and fibrocartilage. The most abundant type is hyaline, found in the nose, ear, trachea, larynx, smaller respiratory tracks, costal cartilages and the articular surfaces of synovial joints. It is the precursor to bone in most of the embryonic skeleton. Injury to or congenital deformity of cartilage can result in pain, loss of mobility and significant deformity. The management of moderate to severe cartilage defects has been difficult owing to the lack of its intrinsic ability to self-repair.^{39,40}

Autologous cartilage grafts

Autologous cartilage grafts are commonly used in the reconstruction of congenital ear deformities, augmentation or revision rhinoplasty, and nipple reconstruction post mastectomy. They can be harvested from the costal cartilages, septum of the nose and the ear. Autologous cartilage grafts can provide

excellent results, but are associated with significant donor site morbidity and the final aesthetic result is dependent on the carving skills of the operating surgeon.

When reconstructing injured joint surface cartilage, transplants of osteochondral plugs have become popular because they offer the chance at true hyaline cartilage resurfacing, can be performed in a single procedure using reusable equipment, and do not require outside laboratory assistance. However, osteochondral grafts are not always amenable to arthroscopic technique and may require an arthrotomy. A recent 10-year follow-up study showed superior clinical results of osteochondral grafting compared with microfracture in young athletes with focal osteochondral defects.⁴¹ Disadvantages of osteochondral grafting include limitations on donor site availability and morbidity.⁴² The space between cylindrical grafts may impair the quality of the repair as poor integration of full-thickness gaps were shown in experiments in goats.⁴³

Autologous chondrocyte implantation

Autologous chondrocyte implantation (ACI) aims to provide hyaline cartilage for repair of injured articular cartilage. Over the last 20 years, it has gained significant popularity. This cell-based technique takes place in three stages: (1) 200–300 mg cartilage is harvested arthroscopically from a less weight-bearing area (intercondylar notch, superior ridge of the femoral condyles). Chondrocytes are isolated after enzymatic removal of the matrix, grown *in vitro* for 4–6 weeks, and then applied to the damaged areas as the patient undergoes a second procedure.

A clinical study of ACI showed an improvement in symptoms in 14 out of 16 patients with femoral condylar lesions at 2 years follow-up.⁴⁴ Another study with a 2- to 9-year follow-up showed good to excellent clinical results following ACI, especially in patients with isolated femoral condyle lesions and osteochondritis dissecans.⁴⁵ At 10–20 years after ACI, 74% of 224 patients reported improvements in symptoms.⁴⁶ Equivalently, matrix-induced ACI resulted in good to excellent clinical outcomes in 12 out of 14 patients after 2- to 8-year follow-up. On the other hand, in a 5-year long-term randomized controlled clinical trial, ACI results were comparable to those of microfracture. Subgroup analysis showed that patients with a time of symptom onset of less than 3 years had better outcomes with ACI than with microfracture.⁴⁷ ACI is technically more challenging, with high reoperation rates of 9–20% and is associated with higher costs.⁴⁸ It also requires *in vitro* expansion of chondrocytes, which theoretically increases the chance of introducing infection, is dependent upon an outside lab, is very expensive, requires an arthrotomy, and necessitates two operations, typically at an interval of two weeks.

Allografts and xenografts

Allografts can be defined as graft of tissue obtained from a donor of the same species as, but with a different genetic make-up from, the recipient. In recent years natural ECM have been

procured, processed and marketed for clinical use as patches to reinforce tissue repair. ECM may provide temporary mechanical support with inherent biological properties that can influence host cell attachment, proliferation and differentiation and ultimately integration into the host tissue.

Bone allografts

Bone allografts can be applied to aid healing of the bone injured either by trauma or due to iatrogenic causes. Survival of bone grafts depends on systemic factors that also affect wound healing (e.g. age, nutritional stage, immunosuppression, diabetes, obesity). In addition, bone grafts placed in an orthotopic position are less prone to absorption. Scarring and infection and previous radiotherapy at the site of the recipient bed also adversely affect graft survival. Allografts lose some of their biologic properties through sterilization and synthetic bone substitutes have only osteoconductive properties if not combined with human growth factors or autograft. If heterologous or synthetic bone is combined with growth factors, these composite grafts may have equal biologic properties to autologous bone grafts, but further research is required to determine their efficacy and cost-effectiveness.

Fascia allografts

An engineered fascia device may be applicable for ligament repair, extension of rotational muscle transfers, grafting lacerated muscles, periosteal coverage, correction of eyelid ptosis and wound healing. Fascia lata or fascia from the iliotibial tract is most commonly used as allografts. An *in vitro* study comparing the histological features, tensile strength to failure, creep and stress-relaxation properties of allograft iliotibial bands with those of tibialis anterior tendons and anterior cruciate ligaments showed they shared a similar structure made of aligned collagen fibres.⁴⁹

No significant difference was observed between allograft iliotibial bands and tibialis anterior tendon ultimate tensile strength, creep and stress-relaxation viscoelastic properties. Anterior cruciate ligament tensile strength was shown to be lower than that of the other two alternatives, although during creep and stress-relaxation testing it was shown to be superior. Allograft iliotibial bands showed low cytotoxicity and good biocompatibility *in vitro*, with a somewhat higher propensity to favour cell attachment and infiltration over time. A clinical study using banked fascia lata allografts for abdominal wall defects showed there were no major signs of recurrence, laxity, infection of grafts or other related pathologic symptoms.⁵⁰ Neofascial tissue appeared stable until 3–6 months, later being gradually degraded and replaced by the host tissue.

Lyophilized or freeze-dried fasciae share similar characteristics of the other alloplastic materials and have several advantages: biocompatibility, ability to be replaced by the host tissue, good mechanical properties and ease of handling.⁵¹ Although lyophilized dura and autogenous fascia have been used for similar purposes, dura has been reported to cause Creutzfeldt-Jakob disease. The major concern with stored fascia is that it is

prone to resorption (up to 10%). In a report, it was pointed out that fascia resorbed in 14 of 127 cases observed for a four-month period.⁵¹

Dermal matrices

Integra

Integra® (Integra LifeSciences Corp., Plainsboro, NJ, USA) is a dermal regeneration matrix consisting of bovine collagen, chondroitin-6-sulfate and a silastic membrane. It has gained widespread use in the clinical treatment of deep partial-thickness and full-thickness burn wounds, full-thickness skin defects, chronic wounds and in soft tissue defects.^{52–55} The bovine collagen dermal component integrates with the patient's cells and the temporary epidermal silicone is peeled away as the dermis regenerates. A very thin autograft is then grafted onto the neodermis.^{53,56}

Heimbach *et al.* showed that Integra was superior to autograft, allograft or xenograft in terms of wound healing time.⁵⁵ However, with regard to wound infection and graft take, Integra did not produce as good a result.^{55,57} Soft tissue coverage with Integra has been successfully used in reconstructive and burn surgery to increase the thickness of the resulting skin and to cover underlying structures like bone or tendon.⁵⁸ However, it requires a two-stage procedure before final defect coverage to allow for adequate vascularization of the template.⁵⁹ Integra provides an immediate donor site coverage independent of wound size.⁶⁰ Additionally, it creates a gliding surface between the underlying structures (e.g. paratenon or nerves and the split-thickness skin graft). Long-term outcomes regarding functionality and cosmesis after radial forearm flap donor site coverage with Integra and split-thickness skin show aesthetic and functionally successful defect coverage.⁶¹

AlloDerm

AlloDerm® (LifeCell Corporation, Branchburg, NJ, USA) is fabricated by a decellularizing cadaveric skin. After processing, the skin is reduced to a basement membrane and properly oriented dermal collagen matrix that theoretically will not be rejected. It ultimately transitions into the host tissue. It has been used extensively in patients with burns to provide biological coverage of third-degree burns and as a framework for split-thickness skin grafts^{62,63} and for intraoral resurfacing after tumour excision.⁶⁴ AlloDerm has been shown in clinical trials to be well tolerated. It becomes integrated into the host skin, is invaded by fibroblasts, and undergoes neovascularization and neoepithelialization, without any evidence of rejection or adverse reaction to the implant by the patient.

Its ability to support neovascularization and tissue ingrowth permits persistent volume correction. This gives AlloDerm an advantage over other injectable forms of collagen. A significant drawback of AlloDerm sheets is the requirement of an incision for placement. An injectable form of AlloDerm (Micronized AlloDerm) has been created by homogenizing AlloDerm sheets

cut into strips. The product is injectable-sized particles of AlloDerm that maintain the ultrastructure of the dermis and can pass easily through a 26-gauge needle.

Several large published series of patients undergoing breast reconstruction utilizing acellular dermal matrices have reported very low rates of capsular contracture.⁶⁵ In addition, a primate study reported the absence of capsule around implants where human acellular dermal matrix was placed.⁶⁶ The absence of a capsule has been observed clinically at the time of implant exchange and was recently confirmed histologically in a clinical study where biopsies were obtained at the time of implant exchange.

Strattice

Strattice™ (LifeCell Corporation, Branchburg, NJ, USA) is an acellular reconstructive tissue matrix derived from porcine dermis, which undergoes proprietary processing that removes cells and significantly reduces the risk of xenogeneic rejection response. The presence of the α -Gal epitope on porcine tissues has been associated with rapid rejection. The enzymatic processing of Strattice significantly reduces the amount of α -Gal epitope in the absence of damage to the matrix integrity (collagen and elastin architecture).⁶⁷ In addition, glycosaminoglycans are still present, indicating that Strattice retains key markers for the presence of relevant biochemical components. The tissue matrix is provided in two forms – pliable and firm. Performance of Strattice has been evaluated by implantation into the abdominal wall of a primate excisional repair model. The animals tolerated the implants well and exhibited no clinical signs of inflammation, laxity, hernia or visceral tissue adhesion. Histological evaluation revealed marked host fibroblast repopulation and neoangiogenesis as early as two weeks post implantation, reaching a plateau at three months. Immunological evaluation of immune cell infiltration demonstrated an early mixed cellular inflammatory response at two weeks, which was transient and diminished to baseline levels by three months. A study comparing three non-cross-linked, commercially available ECM scaffolds (Strattice, Veritas and XenMatrix) showed that the intrinsic denaturation temperature for Strattice, Veritas and XenMatrix was 51°C, 38°C and 44°C, respectively. In addition, the susceptibility to collagenase degradation was increased in Veritas and XenMatrix. These findings suggest more ECM modifications in the Veritas and XenMatrix scaffolds.⁶⁸ In an *in vivo* primate model, Strattice permitted cell repopulation and was remodelled over six months. Veritas was unstable at body temperature, absorbed rapidly and associated with moderate inflammation. XenMatrix caused severe inflammation.⁶⁸

The use of Strattice for repair of incisional hernias in Yucatan pigs compared to primary closure showed that primary closure caused more scarring and bone hyperplasia along the incision line. Strattice elastic modulus and tensile stress were similar to fascia and it provided a more complete wound healing response compared with primary closure.⁶⁹

Clinically, Strattice may enhance soft-tissue support in patients with poor-quality mammary soft-tissue support, especially in implant-based breast reconstruction post mastectomy and in certain patients undergoing augmentation mammoplasty. It has been associated with reduced implant-related complications, including capsular contracture, rippling, palpability and malposition.⁷⁰

Alloplastic

Metals

An ideal metal implant is resistant to corrosion, has a high strength and is biocompatible. Advantages of metallic biomaterials include lack of donor site morbidity and availability. Disadvantages include eliciting host reaction to the material, and their high cost. Metals are used in osteotomy/fracture fixation (plates, screws and wires), cranial plates and artificial joints. Most metals used as biomaterials are alloys. An alloy is a mixture of two or more metals in a single material. Commonly used alloys include stainless steel and alloys incorporating cobalt, titanium or gold.

Stainless steel

Stainless steel is an alloy composed mainly of iron, chromium and nickel. It is highly corrosion resistant in the physiological environment compared to other formulations. Chromium forms a strong adherent surface oxide layer thus rendering it resistant to corrosion. Despite the favourable anticorrosive properties of stainless steel, the leaching of metallic ions can lead to inflammatory reactions in the surrounding tissues and may cause pain.

Cobalt alloys

Vitallium is an example of a cobalt alloy composed of cobalt, chromium and molybdenum (Co–Cr–Mo). This alloy has similar strength to stainless steel but is lighter and less resistant to corrosion.⁷¹

Titanium (Ti)

Titanium is highly resistant to corrosion and has excellent biocompatibility. It has the capacity to form direct chemical bonds with mineralized bone matrix, leading to osseointegration⁷² and providing high stability of these implants. Osseointegration allows for a solid incorporation between the soft tissue, implant and bone. Osseointegrated percutaneous implants have a wide application in plastic surgery, particularly in prosthetic reconstruction. In the first stage the titanium implant is placed into bone and covered by periosteum or bone for at least three months. Titanium has a unique property in that it forms direct chemical bonds with mineralized bone matrix. The histological appearance shows calcified matrix in direct apposition to the metal surface without intervening fibrous tissue. It is this interaction that provides unparalleled stability for titanium implants.

At the second stage the implant is uncovered and a permanent prosthesis is made for the fixation to the implant. Maxillofacial prosthetics largely rely on the support of titanium osseointegrated implants.

Combined with aluminium and vanadium alloy, titanium becomes less malleable. There is also less scatter artefact on radiographic images compared to cobalt alloys and stainless steel.⁷³ Titanium mesh is used in orbital floor reconstruction and for calvarial reconstruction in cranioplasty. Titanium microplates are routinely used for bony internal fixation.

Gold (Au)

Gold has limited application in plastic surgery. Although resistant to corrosion, gold lacks the appropriate mechanical properties and is costly. It still has a role in facial palsy patients by creating iatrogenic ptosis through gold lid loads in the upper eyelid and addressing lagophthalmos.

Ceramics

Ceramics are solid materials composed of inorganic non-metallic substances. Ceramic materials may be amorphous or crystalline. In 1969 L.L. Hench discovered that various kinds of glasses and ceramics could bond to living bone.

They are excellent heat and electric insulators and are heat and corrosion resistant. In addition, they are hard and brittle (fracture without deformation), strong in compression, but weak in shearing and tension. Examples of ceramic materials are porcelain, cements, pottery and glass (amorphous). Chemically, ceramics are normally formed by a hybrid of covalent and ionic bonds and the atomic repeating order can be short range (e.g. glass) or long order (e.g. crystals). The advantages of ceramics are that they can be chemically inert in many environments or designed to be bioactive in the body. They have high wear resistance, high modulus (stiffness) and compressive strength. They are aesthetic and can be used for dental applications. Their composition can be similar to the mineral component in bone. Their disadvantages are brittleness (low fracture resistance, flaw tolerance), low tensile strength (with the exception of fibres) and poor fatigue resistance.

Bioinert ceramics

Bioinert ceramics include oxide ceramics (Al_2O_3 , ZrO_2), silica ceramics, carbon fibre and synthetic diamond. Alumina (Al_2O_3) and zirconia (ZrO_2) are the two most commonly used structural bioceramics. They are primarily used as modular heads on femoral stem hip components, but can also be used for dental restorations – crowns, bridges, brackets. They have the tendency to wear less than metal components, and the wear particles are generally better tolerated. In addition, they have good mechanical and aesthetic properties.

Bioactive ceramics

Bioactive ceramics include tricalcium phosphates, synthetic hydroxyapatite substitutes and bioactive glass (e.g. 45S5 Bioglass®). Bioactive glasses are osteoproliferative and encourage

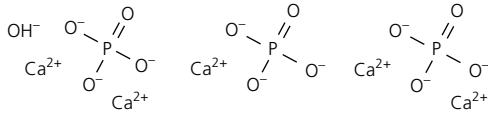


Figure 3.2 Hydroxyapatite (HA).

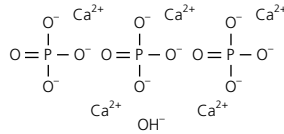


Figure 3.3 Tricalcium phosphate.

bone cells to produce matrix. They bond to soft tissue and hard tissue and are known to stimulate genes in osteoblasts. Bioglass (composition =46.1% SiO₂, 24.4% NaO, 26.9% CaO, 2.6% P₂O₅ (mol%)) was the first material seen to form an interfacial bond with the host tissue. The strength of the bond between Bioglass and cortical bone was shown to be similar to the strength of the host bone.⁷⁴ Bioglass particulate has been in clinical use since 1993 as Perioglas®, used to fill periodontal defects. Hydroxyapatite (HA) and tricalcium phosphate are the most commonly used ceramics in plastic surgery. HA is a naturally occurring mineral form of calcium apatite. It is the hydroxyl end part of the apatite group. Carbonated calcium-deficient HA is the main mineral of which dental enamel and dentin are constituted. By a hydrothermal exchange process of carbonate for phosphate it becomes identical to bone. The material comes as blocks or granules and can bond with adjacent bone without an inflammatory response. It provides a scaffold for bony ingrowth and is not resorbed (Figure 3.2).

Tricalcium phosphate is a synthetic material, more porous than HA and prone to resorption. Its porosity ranges from 30% to 80%. It is usually supplied in blocks or granules, similar to HA (Figure 3.3). Calcium phosphate cement is a synthetic HA cement that when mixed with water and a drying agent (sodium phosphate) forms a paste that hardens to form a microporous HA.⁷⁵ The cement improves contour adaptability and eliminates the need for prefabrication. It is used in calvarial and chest wall defects. It is biocompatible and associated with minimal reabsorption and inflammatory response.⁷⁶

Polymers

Polymers are long-chained molecules composed of small repeating units. Synthetic polymers are found in facial implants, breast prosthesis, sutures, implantable fabrics and joint replacements.

Silicone

Silicon (Si) is a semi-metallic element found abundantly in nature as various forms of silicon dioxide (silica) or silicates. Silicones are mixed inorganic-organic polymerized siloxanes. The basic repeating unit of silicone polymers is siloxane [R₂SiO]_n, where R is an organic group such as methyl, ethyl or

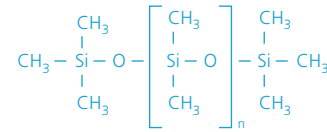
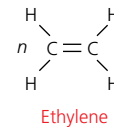
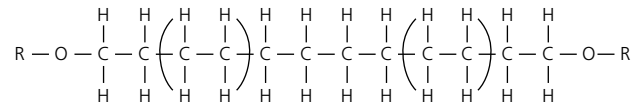


Figure 3.4 Poly(dimethylsiloxane) (PDMS).

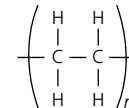


Ethylene

↓ Polymerization



Or more simply



Polyethylene

n = a very large integer

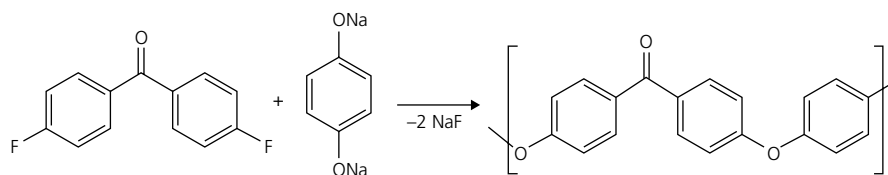
Figure 3.5 Polyethylene.

phenyl. The most commonly used formulation of silicone in medicine is poly(dimethylsiloxane) (PDMS) (Figure 3.4). Medical silicone is available as a liquid, gel or rubber by altering the length and cross-linking of the PDMS chains.⁷⁷

Medical grade silicone is biocompatible, non-allergenic and non-absorbable, which has led to many medical applications including catheters, shunts and intraocular lenses. In plastic surgery, silicone implants have been used in craniofacial surgery for malar and chin augmentation. In hand surgery, silicone rods are routinely used for two-stage tendon reconstruction and as spacers for arthroplasty.

In breast surgery, silicone has been used as prosthesis since 1963. It is estimated that over 2 million women worldwide have received silicone gel-filled implants. Among a variety of biomaterials available, silicone seems to have become the favoured option, especially in adipose tissue replenishment and small joint replacements. It was first introduced as a relatively inert biomaterial, thought to cause minimal inflammation. However, long-term studies are now revealing that silicone affects wound healing and causes capsule formation as a foreign body reaction. Capsular contraction of silicone implants can lead to pain, distortion and hardness. Although alternatives, such as porous polyurethane implants, allow better tissue fixation and are reported to reduce the incidence of capsule formation, they feel unnatural and there is a perceived risk of carcinogenesis. It is true that silicone is relatively inert, however, its tendency to fragment into miniscule particles limits its interfacial biocompatibility, and this holds true for polyurethanes as well. If subjected to repetitive loading and deformation silicone can

Figure 3.6 Poly(aryl-ether-ether-ketone) (PEEK).



fragment into small particles, leading to silicone granulomas caused by a reaction of the immune system.

On the other hand, the fibrous capsule that forms around the implant helps to keep the implant in position and makes subsequent removal easier. There is no evidence that silicone implants are associated with any major diseases, such as connective tissue disorders, cancer or neurological disorders. Silicone breast augmentation is not a contraindication to breastfeeding.⁷⁸

Polyethylene

Polyethylene is the simplest carbon polymer ($-C_2H_2-$) composed of a carbon backbone (Figure 3.5). Pore sizes range from 100–250 μm , which allows tissue ingrowth and stabilization of the implant. In plastic surgery, high-density polyethylene (HDPE) (Medpore[®]) is used for its biocompatibility and tensile strength. The material is porous, allows tissue ingrowth, and is commonly used in ear reconstruction, facial and cranial augmentation. It is available in sheets and blocks that can be sculpted intra-operatively. The host forms a thin capsule around the material with overall low infection rates,⁷⁹ although extrusion is an infrequent problem. Polyethylene can be modified by adding a methyl group to each monomer to form poly(1-methylethylene) or polypropylene (Prolene[®]). This material is commonly used as a non-absorbable suture and can be woven into a fabric mesh, used for chest wall, pelvic floor and abdominal wall reconstruction.

Replacement of the hydrogen atoms in polyethylene with fluorine yields polytetrafluoroethylene (PTFE, Teflon[®]). This material is known for its non-stick properties and good biocompatibility. Rapidly stretching PTFE under certain manufacturing conditions results in formation of a strong microporous material known as expanded PTFE (ePTFE, Gortex[®]). The micropores allow ingrowth of soft tissue and stability of the implant. It has been used as vascular graft since the 1970s and also in the chest wall, abdominal and pelvic floor reconstruction.⁸⁰ The drawback of PTFE vascular grafts is the high incidence of stenosis when small in calibre.

PEEK

Poly(aryl-ether-ether-ketone) or PEEK is a relatively new family of high-temperature thermoplastic polymers, consisting of an aromatic backbone molecular chain, interconnected by ketone and ether functional groups. The chemical structure of polyaromatic ketones confers resistance to chemical and radiation damage, biocompatibility, inertness and radiolucency.⁸¹ The material has been gaining greater popularity over time for its

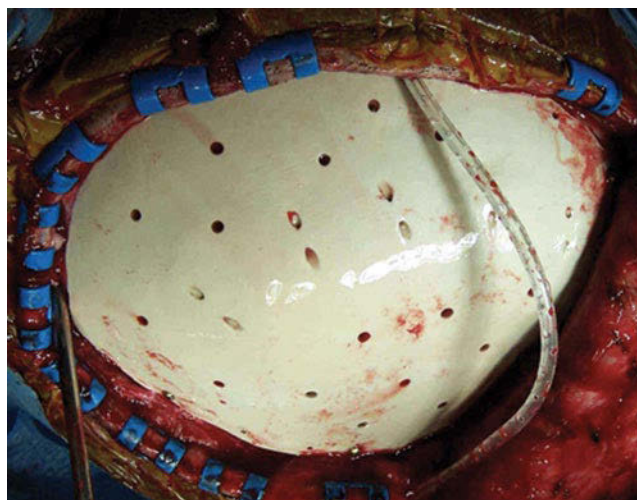


Figure 3.7 PEEK implant applied in cranial vault reconstruction.

medical application. PEEK is being used in facial and cranial reconstruction and its application extends to the field of plastic surgery. It has similar mechanical properties to native bone and is biostable. As a radiolucent material it produces minimal artefacts during imaging (Figures 3.6 and 3.7).

Conclusion

Many alternative biomaterials are available for the practising surgeon; all have their specific benefits and limitations. They have become increasingly popular over the years for their long 'shelf life', ease of use, safety and availability.

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CHAPTER 4

Local anaesthetics

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Introduction

Local anaesthetic agents cause reversible blockade of the neuronal sodium (Na^+) channel, thereby blocking transmission of nerve impulses from nociceptive afferents.

Mechanism of action of local anaesthetics

In the unionized state, local anaesthetics are varying lipid soluble, thereby penetrating the phospholipid cell membrane into the axoplasm. Here, re-ionization of the molecule takes place. It is this re-ionized molecule that then enters and blocks sodium channels, preventing sodium influx into the neuron during depolarization, and thus halting propagation of an action potential.¹

Local anaesthetics have also been shown to exert far-reaching effects on other receptors, independent of their action on sodium channels. These include inhibition of the local tissue inflammation that results in nociceptor sensitization, leading to pain and hyperalgesia.² They may also be involved in priming of descending inhibitory pain pathways, which could show clinical benefit in the reduction of neuropathic pain.³ Furthermore, local anaesthetics have been shown to have antithrombotic, antimicrobial and neuroprotective actions.^{4,5}

Nerve fibre characteristics and differential blockade

Several nerve fibre characteristics have been shown, *in vitro*, to govern the sensitivity of nerve fibres to local anaesthetics:⁶

- Smaller nerve fibres with shorter axons are more easily blocked compared to larger nerve fibres with longer axons.
- Smaller diameter nerve fibres (e.g. 0.4–1.2 μm C-fibres which transmit pain, temperature and touch) are blocked at lower concentrations compared to larger diameter nerve fibres.
- Myelinated fibres (e.g. A- α fibres, which convey motor and proprioception) are more easily blocked than unmyelinated nerve fibres (e.g. C-fibres), as local anaesthetic tends to pool within the myelin sheath.

These principles do not, however, consistently correlate with the clinical observations. When blocking peripheral nerves, there is usually an order in the disappearance of sensing modalities: pain, temperature, deep pressure and finally motor function loss.

Physiochemical properties of local anaesthetics

Chemistry

Local anaesthetics are weak bases.¹ Their basic molecular structure comprises a lipophilic aromatic moiety and a hydrophilic amino domain (Figure 4.1).

Depending on the chemical linkage between the respective lipophilic and hydrophilic groups, local anaesthetics can be classified as either amides or esters. This linkage has implications on drug metabolism and allergenicity (Table 4.1).

Potency

The potency of local anaesthetics correlates well with lipid solubility and molecular weight *in vitro*.⁸

Other factors affecting potency include tissue distribution and direct vasodilator actions of the local anaesthetic.

Duration of action

Local anaesthetics are classed as 'short', 'medium' or 'long' acting. The duration of action positively correlates with the degree of protein binding: the more protein bound a local anaesthetic, the longer the duration of action, with the 'unbound' portion of the drug being biologically active.⁸ However, local anaesthetic molecules remain bound to sodium channels for seconds only. Given such a high dissociation rate, of greater relevance are factors that maintain a high concentration of local anaesthetic molecules around the nerve. These factors are:

- local anaesthetic lipid solubility (there is a direct correlation between lipid solubility and protein binding);
- tissue vascularity (depends on regional blood flow, and the addition of vasoconstrictors); and
- the intrinsic vasodilatory properties of local anaesthetics.

Local anaesthetics cause vasoconstriction at very low concentrations and vasodilatation at higher, clinically useful, concentrations.¹ Considerable variability exists however. Moreover, ester local anaesthetics are more potent vasodilators compared to amides. The exception to this rule is cocaine, which causes vasoconstriction by blocking reuptake of noradrenaline (nor-epinephrine) at sympathetic nerve endings. The greater the intrinsic vasodilatory action of a local anaesthetic, the more pronounced the effect of the addition of vasoconstrictors.

Onset of action

The faster the diffusion of local anaesthetic across a nerve sheath, the faster the onset of action. The rate of diffusion is determined by:

- the site of local anaesthetic injection,
- the concentration of local anaesthetic used,
- the degree of ionization of local anaesthetic molecules.

The latter is related to a physiochemical property of a drug known as the pK_a . The pK_a is the pH at which 50% of the drug is present in an ionized form.¹ For basic drugs, such as local anaesthetics, a greater proportion of drug is present in the ionized form at a pH below the pK_a ; at a pH above the pK_a , a greater proportion of the drug will be unionized. The reverse is true for

weak acids. Therefore, local anaesthetics with a higher pK_a will have a greater drug fraction present in the ionized state, resulting in a slow onset of action.¹ The most clinically relevant comparison is the faster action onset of lidocaine (lignocaine) (pK_a 7.9) compared to bupivacaine (pK_a 8.1).

Pharmacokinetics of local anaesthetics

Absorption

Absorption of local anaesthetics depends primarily on:

- the site of injection,
- the addition of vasoconstrictors, and
- the physiochemical properties of the local anaesthetic agent itself.

On reaching the systemic circulation, local anaesthetics are taken up by distant organs according to vascular density.⁹ Highly vascular organs, such as the brain, heart, lung, liver and kidneys, will have greatest exposure to the peak concentration of unmetabolized local anaesthetic. Uptake is determined by the tissue–plasma partition coefficient.

Distribution

Local anaesthetic molecules can be ‘free’ or bound to plasma proteins. Alpha-1 acid glycoprotein binds the weakly basic local anaesthetic molecules with high affinity. However, albumin represents a greater fraction of plasma proteins, and as such has a high capacity (but comparatively low affinity) for binding to local anaesthetic molecules. Local anaesthetic protein binding is pH-dependent, and decreases as the pH decreases.⁹ This has important clinical relevance. For example, in local anaesthetic systemic toxicity (see below), a metabolic acidosis may ensue secondary to tissue hypoxia. Thus as the plasma pH decreases, the proportion of ‘free’ drug increases, potentiating the toxicity. Furthermore, intracellular acidosis increases conversion of the local anaesthetic molecule into the cationic state, rendering the molecule unable to cross the cell membrane. This ‘ion trapping’ exacerbates the toxicity further.

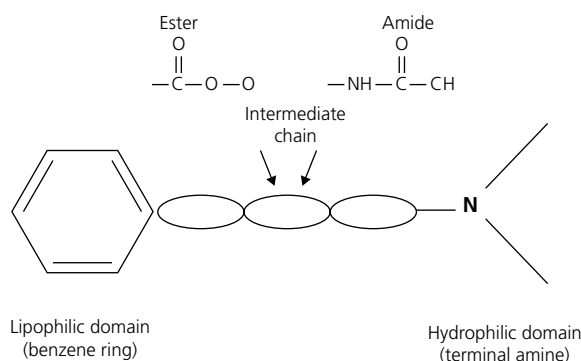


Figure 4.1 Chemical structure of a local anaesthetic.

Table 4.1 A comparison of the main characteristic differences between amide and ester local anaesthetics

Class	Examples	Allergenicity ^a	Pharmacokinetics	Stability
Amides	Lidocaine Mepivacaine Bupivacaine Ropivacaine Levobupivacaine	Rare ⁷	Extensively protein bound Hepatic metabolism by amidases; slower than ester metabolism thus potential for accumulation	Long shelf-life and heat-insensitive unless mixed with glucose
Esters	Procaine 2-Chloroprocaine Tetracaine (Amethocaine) Benzocaine Cocaine	Rare, but more common than with amides, especially with procaine Cross-reactivity may exist with other esters Implicated metabolite: <i>p</i> - aminobenzoic acid	Minimally protein bound Hydrolysis by plasma cholinesterase and other esterases	Short shelf-life due to spontaneous hydrolysis

^aAntioxidant additives, or preservatives such as metabisulfite or parabens may be implicated in causing non-anaphylactic reactions.

Clearance and metabolism

Esters (except cocaine) undergo rapid hydrolysis by plasma cholinesterases, and other non-specific esterases.¹ Their metabolites are inactive, but may be associated with hypersensitivity reactions. Amides are metabolized in the liver by the action of amidases.¹

Hepatic clearance is dependent on the hepatic blood flow, and the hepatic extraction ratio, which depends on the ratio of unbound to protein-bound drug. Drugs that are comparatively less protein bound (e.g. lidocaine) have a higher hepatic extraction ratio.¹⁰ Clearance is thus only limited by hepatic blood flow. Thus factors reducing hepatic blood flow (e.g. heart failure) may increase toxicity. Conversely, the clearance of drugs that are more protein bound (e.g. bupivacaine) is more dependent on free drug concentration, rather than hepatic blood flow.⁹ Intrinsic liver disease may, in turn, affect free drug concentration (e.g. a reduction in liver enzymes or reduced plasma protein production).

Patients with liver disease and cardiac disease can receive 'normal dose' single shot blocks. However, catheter infusions or repeated blocks should be given at a reduced dose.¹¹

Renal failure associated with severe uraemia can be associated with reduced clearance and more rapid absorption of local anaesthetic, leading to higher peak plasma concentrations.

Some local anaesthetics may undergo first-pass metabolism by lung extraction due to the lower pH of the lungs compared to the plasma, resulting in an 'ion trapping' effect. This effect is lost in patients with right-to-left cardiac shunts, thus increasing the risk of toxicity.¹²

Local anaesthetics in clinical practice

Table 4.2 summarizes the pharmacokinetic and pharmacodynamic properties of the most commonly used local anaesthetics.

Concentration of local anaesthetics

A 1% solution of a drug contains 1000 mg of drug per 100 mL of solution. Thus a 1% solution of lidocaine contains 1000 mg of lidocaine per 100 mL (10 mg/mL). Similarly, a 0.5% solution of bupivacaine contains 500 mg of bupivacaine in 100 mL of solution (5 mg/mL).

Clinically useful adjuncts

Vasoconstrictors

Adrenaline (epinephrine) is the most commonly used vasopressor. It slows the rate of absorption of local anaesthetics, thereby prolonging the duration of action¹¹ and reducing the risk of toxicity. Furthermore, it can help decrease surgical blood loss, and may identify inadvertent intravascular injection. Absolute contraindications to use include injection close to tissues with inadequate collateral circulation (ankle, wrists,

digits and penis) and intravenous regional anaesthesia. Relative contraindications to use include the presence or risk of tissue ischaemia, cardiac disease, hypertension and concomitant use of other sympathomimetic drugs (e.g. tricyclic antidepressants).

The maximum safe dose of adrenaline is 4 µg/kg; concentrations in excess of 1 : 200 000 should not be used (Table 4.3).

Sodium bicarbonate

Sodium bicarbonate (NaHCO₃) raises the pH of the local anaesthetic solution, thus increasing the fraction of unionized drug able to traverse the neuronal membrane.¹¹ It may also reduce the pain of injection.¹³ Furthermore, the speed of onset of the local anaesthetic is theoretically increased. However, the rise in pH seen is only modest, and limited by the solubility of the base form of the drug. This translates to only modest decreases in block onset time.¹⁴

The recommended dose is 1 mL of 8.4% sodium bicarbonate per 10 mL of local anaesthetic.¹¹

Topical local anaesthetics

Topical local anaesthetics are available in a variety of formulations and presentations. Their advantages include avoidance of first-pass metabolism, and avoidance of the risks of more invasive local anaesthetic techniques.

The 'ideal' topical local anaesthetic formulation will maximize permeability and absorption via the stratum corneum, while limiting systemic absorption hence toxicity.

Disadvantages of topical local anaesthesia include unpredictable absorption, uneven distribution or spread of the agent during application, and a failure of the occlusive dressings to stick to the skin. These agents should not be applied to broken, irritated or infected skin.

Topical anaesthesia lends itself well to procedures such as skin biopsy and removal, electrocautery and superficial laser therapy. It also enables painless needle insertion.

Cutaneous agents

The most commonly used topical agents are summarized in Table 4.4.

Mucosal agents

Examples of suitable ointments include cocaine 4%, benzocaine 5–20% and lidocaine 2–5%. Lidocaine is also available as an aerosol preparation.

Transdermal local anaesthetic delivery

In order for a drug to passively diffuse across the stratum corneum, it must be lipophilic, non-ionized and have a low molecular weight (<500 Da). Various technologies are evolving in further accelerating transdermal drug delivery.¹⁶

Table 4.2 Summary of local anaesthetics most commonly used in clinical practice

Class	Amides				Esters			
	Lidocaine	Mepivacaine	Prilocaine	Bupivacaine	Levobupivacaine	Ropivacaine	Procaine	Amethocaine
Local anaesthetic								
Onset of action	Fast	Fast	Fast	Medium	Medium	Medium	Slow	Slow
Relative potency	2	2	2	8	8	8	1	8
Duration of action (h)	Medium	Medium	Medium	Long	Long	Long	Short	Long
Maximum dose (mg/kg) ^a	3	5	6	2	2.5	3	7	1.0
Maximum dose with adrenaline (where applicable) (mg/kg)	7	7	9	2.5	3	4	10	1.5
Additional notes	Relative lack of vasodilatation Risk of methaemoglobinaemia				S-enantiomer of bupivacaine; modestly less cardiotoxic and neurotoxic	Pure S-enantiomer Lower propensity for motor block compared to bupivacaine Marked vasoconstriction at low concentration	Local vasodilatory action Lower propensity for motor block compared to bupivacaine Marked vasoconstriction at low concentration	Blocks reuptake of noradrenaline, thus can provoke hypertensive crisis and arrhythmias Potent vasoconstrictor

^aQuoted maximum 'recommended 'safe doses' vary. Most available data is extrapolated from animal studies. Other factors, including the site and speed of injection, are of greater relevance (see text).

Iontophoresis

Iontophoresis involves the application of a constant low-voltage direct current to actively transport drugs across a biological membrane.¹⁶ As ions move between positive and negative electrodes, they are actively transported through the skin. Drug molecules that are hydrophilic and ionized, thus ordinarily being skin-impermeable, are ideal for delivery by iontophoresis.

In the United States, the first Food and Drug Administration (FDA)-approved iontophoretically delivered local anaesthetic patch was LidoSite® (lidocaine hydrochloride 10% and adrenaline 0.1%). Pretreatment with sonophoresis may work synergistically to enhance drug delivery.¹⁶

Microporation technology

Microporation produces transient microchannels in the skin in order to form a conduit through which drugs may pass.¹⁶ Examples include the Painless Laser Epidermal System® (Pantec

Biosolutions AG, Liechtenstein). High-velocity, high-pressure 'needleless jet' sprays can deliver local anaesthetic in a similar way.

Sonophoresis

Sonophoresis employs the use of low-frequency (20–100 kHz) ultrasound waves to cause acoustic cavitation within the stratum corneum, resulting in the non-linear formation of acoustic microjets. The addition of surfactant material to the skin works synergistically to enhance the flux of hydrophilic molecules with large molecular weights that would ordinarily not traverse the skin membrane.¹⁶ Ultrasound can have a thermal heating effect, and induce convective transport, both of which may contribute to enhancing lipid permeability.¹⁶

Magnetophoresis

The application of a magnetic field causes an increase of the oil: water partition coefficient of drugs, making them move across biological membranes such as the skin by 'magnetokinesis'.¹⁶ Patch systems that employ this technology are currently in development.

Electroporation

Electroporation employs high-voltage (>100 V) pulses for microsecond bursts, causing formation of aqueous channels within the stratum corneum, providing a conduit for the diffusion of molecules. The time between the high-voltage pulses

Table 4.3 Equivalent drug concentrations for standard drug solutions

Drug solution	Drug concentration (µg/mL)
1 : 10 000	100
1 : 100 000	10
1 : 200 000	5
1 : 400 000	2.5

Table 4.4 Commonly used topical local anaesthetic preparations

Local anaesthetic	Trade name (pharmaceutical company)	Formulation	Application time (minimum–maximum)	Maximum dose/area	Particulars
Lidocaine 2.5% w/w + prilocaine 2.5% w/w	EMLA 5% (Astra Zeneca)	Cream + occlusive dressing	1–5 h	Adults: 60 g + 600 cm ² Children: 6–11 years: 20 g + 200 cm ² for 1–5 h 1–5 years: 10 g + 100 cm ² for 1–5 h 2–11 months: 2 g + 20 cm ² for 1 h <2 months: 1 g + 10 cm ² for 1 h	Not for use if <37 weeks gestation Not for use in infants (<12 months) taking methaemoglobin-inducing agents
Tetracaine 4% w/w	Ametop (Smith & Nephew)	Gel + occlusive dressing	30–45 min	Adult + child >5 years: max 5 tubes (1 tube = 1 g) at separate sites at single time Child <5 years: max 1 tube at single time	Not recommended for infants <1 month
Lidocaine 7% + tetracaine 7%	Pliaglis (Galderma)	Cream	30–60 min	1 mm layer (1.3 g of cream per 10 cm ²)	Not recommended if age <18 years Apply with spatula only Peel off skin prior to procedure
Lidocaine 4% w/w	LMX 4 (Ferndale)	Liposomal + occlusive dressing	30 min–5 h Child: 3–12 months: 4 h. <3 months: 1 h	Adult + child >1 year: 2.5 g Child <1 year: 1 g	

allows the skin to depolarize, thus avoiding interference with current flow.¹⁶

Future drug delivery

An evolving area of research in local anaesthetic delivery is that encapsulation matrices, where the properties of carrier molecules are used to enhance drug delivery and action.¹⁶

Liposomes are lipid vesicles that act as a drug reservoir for local anaesthetic. A lipid-soluble molecule is carried in the outer lipid bilayer, and aliphatic or hydrophilic molecules are contained in an aqueous interior. Liposomes are biodegradable, and can be administered by various routes (intravenous, topical, intramuscular, subcutaneous, nasal and oral). However, consistency in their manufacture is challenging, posing the risk of drug leakage of toxic metabolites.¹⁶

Other areas in development include microemulsions, hyaluronic acid-containing hydrogels, microspheres and nanospheres, injectable liquid polymers and bioadhesive films.

Local anaesthetic systemic toxicity

Local anaesthetic systemic toxicity (LAST) is a spectrum of neurological and cardiovascular signs and symptoms that result from the direct effect of local anaesthetics on neuronal activity within the central nervous system (CNS), autonomic ganglia and myocardium. The manifestation of these symptoms is related to the plasma concentration of the local anaesthetic, which is in turn dependent on:

- the dose of local anaesthetic administered,
- the rate of local anaesthetic absorption, distribution, metabolism and excretion, and
- the vascularity of the injection site.

Toxicity can present in a variety of ways, hence a high index of suspicion is paramount.

Symptoms and signs

Raised plasma concentration of local anaesthetics characteristically causes initial excitatory phenomena, secondary to suppression of inhibitory neuronal circuits. This is followed by nervous system and cardiovascular depression.¹⁷

CNS effects

The patient may complain of circumoral tingling, a metallic taste in the mouth, diplopia, dizziness and tinnitus. They may appear confused, anxious or agitated, or show signs of muscle excitability and twitching. Convulsions may occur.^{17,18}

CNS depressant effects manifest as apnoea and a reduced level of consciousness or coma.

Cardiovascular effects

Cardiovascular effects of LAST may initially stimulate sympathetic activity, resulting in hypertension and tachycardia. As plasma concentrations rise, this can rapidly evolve into ven-

tricular arrhythmias: ventricular tachycardia, including polymorphic ventricular tachycardia (torsades de pointes) and ventricular fibrillation.¹⁷ Consequently, refractory hypotension, heart conduction blocks or complete loss of cardiac output may occur.¹⁷

Toxicity varies between local anaesthetics, with more potent agents such as bupivacaine being more cardiotoxic compared to, for example, lidocaine. The clinical relevance of this is that cardiotoxicity with more potent agents is less likely to produce premonitory CNS signs, implying a narrower safety margin.¹⁷ Moreover, once malignant arrhythmias occur, they typically require prolonged resuscitation.

All local anaesthetics are arrhythmogenic and, with the exception of cocaine, have direct myocardial depressant effects.

Prevention of LAST

Facilities, including Local Anaesthetic Toxicity Toolkit, together with local departmental algorithms for treatment of LAST must be available in every theatre and recovery complex. All practitioners must be familiar with the location and usage of this apparatus.

Risk factors for LAST

Variables that may affect the risk of LAST include:¹⁷

- site of injection and vascularity,
- low cardiac output state,
- liver disease,
- pregnancy,
- acidaemia,
- concomitant use of sedatives, as these may mask symptoms or decrease the seizure threshold.¹⁹

Reducing the risk of LAST

The following practices reduce the risk of LAST:

- The use of the minimum dose of local anaesthetic required and adherence to maximum safe dosages.¹⁷ Note that the concept of 'maximum safe dose' is based mainly on extrapolations from animal data. It is clinically irrelevant unless the site, speed and route of injection are taken into consideration.
- Regular aspiration prior to injection (every 4–5 mL), ensuring slow, incremental injection.¹⁷
- Aspiration tests repeated each time the needle is moved. A negative aspiration test does not exclude intravascular injection, particularly if the needle is placed in a small vessel or a catheter technique is used.
- Use of pharmacological markers (e.g. adrenaline) to aid detection of inadvertent intravascular injection.¹⁷ Injection of a 2–3 mL local anaesthetic solution containing adrenaline 1 : 200 000 would be expected to induce a sympathetic response within 1–2 min should the needle be placed intravascularly.
- Regular communication with the patient, observing for symptoms of LAST.

- Routine monitoring of the patient during the recovery phase, in case of a delayed presentation of LAST.
- Use of real-time ultrasound-guided peripheral nerve blockade.
- Use of more dilute local anaesthetic solutions.
- Reduced doses of local anaesthetic in high-risk patients.

Management of LAST

Management of LAST is underpinned by three principles:

- restoration of the circulation to maintain tissue oxygenation,
- control of seizure activity, thus reducing oxygen consumption, and
- mitigating the systemic effects of local anaesthetics, particularly with respect with the cardiovascular system.

Management requires the expertise of the acute resuscitation team, usually led by an anaesthetist or intensive care physician.

All resuscitation team members, including surgeons, must be familiar with the resuscitation algorithm:²⁰

- 1 Stop injection of local anaesthetic and call for help. If in doubt, call the cardiac arrest team.
- 2 Start resuscitation (as per national protocols):
 - (a) Airway: maintain patency using chin lift/jaw thrust and airway adjuncts; intubate the trachea once skilled persons available.
 - (b) Breathing: ventilate both lungs with 100% oxygen; hyperventilating the patient (respiratory rate >20) may help to alleviate ensuing metabolic acidosis.
 - (c) Circulation: establish intravenous access. Control seizures by bolus administration of an intravenous benzodiazepines, barbiturates and propofol.
- 3 Start treatment:
 - (a) Start intravenous 20% lipid emulsion therapy:
 - Give 1.5 mL/kg bolus over 1 min and simultaneously start infusion at rate of 15 mL/kg/h.
 - Repeat bolus dose up to two times, with 5-min intervals between each bolus, should cardiovascular stability not be restored. In this instance, the background infusion rate should also be doubled to 30 mL/kg/h (after 5 min of starting the infusion).
 - The maximum cumulative dose of 20% lipid emulsion should not exceed 12 mL/kg.
 - (b) Treat arrhythmias as per nationally endorsed resuscitation guidelines. *Arrhythmias in LAST are typically refractory to treatment. Resuscitation may be prolonged.*^{17,20}
 - (c) Consider cardiopulmonary bypass in cases refractory to treatment.
- 4 Post-resuscitation management:
 - (a) Patients must be managed in a high dependency unit.
 - (b) Where 20% lipid emulsion has been administered, there remains a theoretical risk of acute pancreatitis for up to 48 h. Thus regular clinical assessment and serum lipase assays must be considered.

The standard practice of administering 1 mg of adrenaline, as advocated by most Advanced Life Support Algorithms, may exacerbate the arrhythmogenic state induced by LAST. For this

reason, general consensus is that adrenaline should be given at a reduced dose of <1 µg/kg.¹⁷

Methaemoglobinaemia

Methaemoglobin is formed when the the iron contained within haemoglobin is present in the ferric (Fe³⁺) rather than the reduced ferrous (Fe²⁺) state. The result is an inability to carry oxygen.²¹

Local anaesthetics implicated in the development of methaemoglobinaemia are, namely, prilocaine (which is metabolized to *o*-toluidine, which oxidizes haemoglobin to methaemoglobin), benzocaine and lidocaine.²¹

Methaemoglobin levels exceeding 1% are deemed abnormal. Symptoms manifest with methaemoglobin levels in excess of 10%, and include a slate-grey discoloration of the skin, fatigue, shortness of breath and generalized weakness, progressing to cardiac dysrhythmias and coma. Risk factors include pre-existing congenital methaemoglobinaemia, the administration of drugs with a high oxidative stress (e.g. phenytoin, sulphonamides) and age under six months.^{1,21} The latter is due to reduced level of NADH reductase enzymes.¹

Management of methaemoglobinaemia is with high concentration oxygen and the administration of a reducing agent such a methylene blue (1 mg/kg intravenously).

Regional techniques of relevance to plastic surgery

There are many regional techniques that lend themselves well to plastic and reconstructive surgery. The advantage of successful regional techniques compared to local infiltration is a more predictable area of anaesthesia, with minimal risk of tissue distortion.

The majority of regional techniques are carried out by anaesthetists. The use of ultrasound-guided blocks has become increasingly popular.

The following criteria must be fulfilled prior to the commencement of any regional anaesthetic technique:

- 1 Patient consent.
- 2 Exclusion of contraindications to block:
 - Absolute contraindications: patient refusal, local anaesthetic allergy (rare)
 - Relative contraindications: localized infection; coagulopathy
- 3 Confirmation, both verbal and written, of the site and side to be blocked
- 4 Patient monitoring
- 5 Resuscitation equipment and drugs
- 6 Presence of a trained assistant
- 7 An alternative management plan should the regional technique fail.

Intravenous regional anaesthesia (Bier block)

Indications

Surgery on the wrist or hand lasting less than 1 h (e.g. foreign body removal, release of tendon contractures).

Principle

Local anaesthetic is injected into the forearm previously exsanguinated by use of a tourniquet. Local anaesthetic diffuses from the venous circulation into surrounding nerve endings, providing anaesthesia below the level of the tourniquet. Prior venous exsanguination prevents dilution of the local anaesthetic, thus maintaining a sufficient concentration gradient for diffusion to occur.

The duration of the block is limited by tourniquet pain, which typically occurs after 30–45 min. Patients invariably require conscious sedation.

Technique

- 1 An intravenous cannula is inserted into the opposite arm ('systemic circulation' access).
- 2 A 22-gauge intravenous (IV) cannula is also inserted into the hand of the limb to be blocked ('regional circulation' access).
- 3 With the patient supine, the arm to be blocked is passively exsanguinated by elevation for 2 min.
- 4 A double-cuffed tourniquet is placed proximal to the limb that is to be blocked. Adequate padding between the skin and the tourniquet is essential to prevent skin necrosis. It is imperative that both tourniquet cuffs are checked for leaks.
- 5 An Esmarch bandage is applied to enhance limb exsanguination. At this point the distal cuff is inflated, followed by the proximal cuff. Once the proximal cuff is fully inflated, the distal cuff can be deflated. The cuffs are inflated to approximately 100 mmHg above systolic blood pressure.
- 6 The Esmarch is removed, and the distal limb is checked for pallor and the absence of a distal pulse. Local anaesthetic is then injected into the IV cannula of the exsanguinated limb.
- 7 For the upper limb, 12–15 mL of 2% lidocaine or 30–40 mL of 0.5% lidocaine is a suitable regimen. Onset of anaesthesia typically takes 5–10 min.

Complications

- *Tourniquet pain* typically occurs after 30 min. It is alleviated by inflating the proximal cuff and deflating the distal cuff, which may afford a further 30 min of operating time.
- *Local anaesthetic toxicity* can be avoided by ensuring correct function and application of the tourniquet, with clear labelling of the 'distal' and 'proximal' cuffs. At the end of surgery, the tourniquet should be released in a graduated manner, preventing a sudden washout of local anaesthetic into the systemic circulation.
- *Haematoma* the smallest possible gauge IV line should be used. Should there be failed attempts at IV cannulation,

pressure should be applied until bleeding stops, prior to limb exsanguination.

- *Engorgement of the extremity* occurs where arterial blood enters the limb in spite of the tourniquet, and venous blood is unable to escape. It can be avoided by ensuring good tourniquet function with a pressure of at least 100 mmHg above systolic.

Nerve blocks for the face

General considerations

- All three anatomical landmarks discussed below can be identified by imagining a line drawn vertically parallel to the midline of the face, intersecting a centred pupil.
- The eyes must be protected from cleaning solutions.
- The needle should never be inserted directly into the relevant foramina, as this may cause damage to the neurovascular bundle. If immediate paraesthesia is encountered on slow injection, the needle should be withdrawn and redirected.
- Topical anaesthesia should be considered for intraoral blocks.
- Commonly used agents include 1% lidocaine or 1% mepivacaine.
- A 23- or 25-gauge 25 mm intradermal needle attached to a 3 mL or 5 mL Luer syringe can be used.

Table 4.5 summarizes the relevant sensory innervation of important branches of the trigeminal nerve. For a more detailed description of trigeminal nerve anatomy, please refer to an anatomy textbook.

Surpraorbital nerve block

The supraorbital and the supratrochlear nerves can be blocked by a single injection technique.

Anatomy

The supraorbital nerve exits the supraorbital notch. At this junction, the nerve and its branches approximate with the orbital periosteum, underneath the obicularis and frontalis muscles. After giving off palpebral filaments to the upper eyelid, the nerve divides into medial and lateral branches, which fan out in a cephalad direction.

Landmark

Supraorbital notch (in a smaller percentage closed to form a foramen), palpated at the junction of the lateral two thirds and medial one of third of the inferior border of the supraorbital ridge; 2–3 cm lateral to the midline of the forehead.

Technique

The needle is inserted 1 cm medial to 0.5 cm immediately superior to the supraorbital notch. The needle is then withdrawn a few millimetres and redirected medially towards the junction of the orbital rim with the nasal bone; 2–3 mL is injected.

Table 4.5 Summary of sensory innervation of important branches of trigeminal nerve

Trigeminal nerve division	Nerve	Branch	Sensory supply (ipsilateral side)
Ophthalmic (V1)	Frontal	Supraorbital nerve	Upper eyelid Forehead Scalp (until lambdoid suture)
		Supraorbital nerve	Conjunctiva Lower medial forehead Skin of upper eyelid Frontal sinus mucosa
Maxillary (V2)	Infraorbital		Lower eyelid Side of nose Upper lip Facial gingiva maxillary premolars and anterior teeth
Mandibular (V3)	Inferior alveolar	Mental nerve	Anterior chin Lower lip to mentolabial fold Lower teeth

Supratrochlear nerve block

Anatomy

The supratrochlear nerve exits the orbit 1 cm medial to the supraorbital nerve. It passes above the trochlea of the superior oblique muscle, and courses cephalad, giving off several small terminal branches.

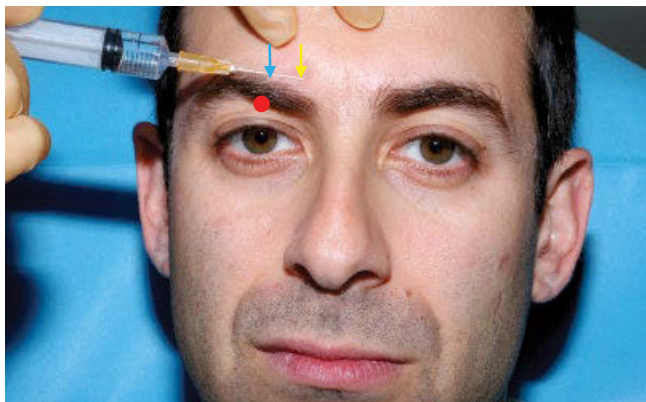
Landmark

The supraorbital notch (see above), as the supratrochlear notch is difficult to palpate, or else absent in some people.

Technique

The needle is withdrawn a few millimetres and redirected (along the line of the supraorbital ridge) approximately 1 cm medially towards to the junction of the orbital rim with the nasal bone; 1–2 mL of local anaesthetic is injected into the subcutaneous tissue in a fan-like distribution.

Application of mild pressure to the upper eyelid via a finger or roll of gauze while administering the aforementioned blocks may help to prevent palpebral oedema and/or ecchymosis (Figure 4.2).



(a)



(b)



(c)

Figure 4.2 (a) Supraorbital foramen (red dot); approximate positions of needle insertion for supraorbital (blue arrow) and supratrochlear (yellow arrow) nerve blocks. (b) Intraoral approach for infraorbital nerve block. (c) Intraoral approach for mental nerve block.

Infraorbital nerve block

Anatomy

The infraorbital nerve is the third branch of the maxillary (V2) division, which divides off within the pterygopalatine space, passing through the infraorbital fissure, along the floor of the orbit to enter the infraorbital canal. On exiting the canal, it gives off three terminal branches: the nasal, palpebral and labial nerves.

Landmark

The infraorbital foramen, located along the inferior border of infraorbital rim.

Technique

The cheek is retracted and the infraorbital foramen is continually palpated. The needle is inserted into the mucosa directed approximately 0.5 cm above the upper second bicuspid tooth, aiming cephalad and laterally towards the lower border of the foramen; 2–3 mL of local anaesthetic is injected.

Mental nerve block

Anatomy

The mental nerve is one of two terminal branches of the inferior alveolar nerve. It exits the mental foramen, an anterior opening of the mandibular canal. As it passes underneath the depressor anguli oris muscle, it divides into three further branches.

Landmark

The mental foramen, which is normally located between the two lower premolars in the adult, approximately 1 cm below the gum-line. In children, the foramen is at the junction of the first and second primary molars. There is some variability with the position of the foramen, however.

Technique (intraoral approach)

The cheek is retracted. The needle is inserted 0.5 cm above the mental foramen, and directed caudally; 2–3 mL of local anaesthetic is injected.

Where the foramen is not palpable, the nerve can be targeted by injecting the buccal mucosa at the junction of the two lower premolar teeth.

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CHAPTER 5

Lasers

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Introduction

Albert Einstein first proposed the concept of stimulated emission in 1927 and the first laser (light amplification by the stimulated emission of radiation) was developed in 1959 (ruby). Lasers were first used on human skin in 1963.¹

The use of these early lasers was crude, and damage to cutaneous tissues, and hence scarring, was common. It was not until the introduction of the concept of selective thermolysis by Parrish and Anderson in the 1980s² that the use of cutaneous medical lasers became commonplace. This concept has led to the development of lasers that can act on specific targets or 'chromophores' in certain layers of the skin, allowing for targeted therapy with minimal collateral damage in the form of scarring.

Laser physics

Laser light is produced by the release of a series of coherent, unidirectional photons of light. These photons are generated by an external light source or electromagnetic field, which is used to excite a specific medium. Different media, which can be in solid, gaseous or liquid state, are selected to produce different lasers.

The atoms in the medium *absorb* the energy from the external source, thereby exciting some of their electrons into higher energy levels. Upon relaxation back into resting state, the energy lost is released as a photon of light in a process known as *spontaneous emission*. These photons are incoherent and go on to excite other electrons into higher energy levels, resulting in multiple photons being spontaneously emitted throughout the material. This process continues until the number of electrons in the higher energy levels outnumbers those in the lower energy levels, resulting in a situation called *population inversion*. When this occurs, the high-energy electrons will cause the photons to drop down into resting orbit, emitting a further photon of the same frequency and direction which is *in phase* with the

incident photon without absorbing the incident photon. This process is called *stimulated emission* of photons and each photon released contains a specific amount of energy that is consistent throughout the material.

By placing the medium between two mirrors this process can amplify itself by allowing light that is produced at right angles to the mirrors to pass back and forth through the medium, producing increasing numbers of unidirectional, coherent photons which are then released, by transiently removing one mirror, as a laser beam.

Laser light has specific therapeutic properties that are exploited in medical use:

- **Monochromatic** – Lasers have a specific wavelength and so have the ability to selectively target chromophores.
- **Coherent** – Alignment of light waves allows high intensity to be focused over small area.
- **Compressible** – The high temporal coherence of waves allows for ultra short pulses that deliver localized energy.
- **Collimated** – Transmission of parallel rays of light without divergence allows for high localized intensity.

Principles of laser therapy in cutaneous treatment

Selective photothermolysis predicts that selective thermal damage of a chromophore will result when sufficient fluence (energy delivered per unit area – J/m²), at a wavelength preferentially absorbed by that target is delivered during a time (pulse duration) equal to or less than the thermal relaxation time of target.²

The use of laser in cutaneous treatment involves the selection of a specific wavelength that is preferentially absorbed by a specific target or chromophore in the skin. This specific target can be oxyhaemoglobin (vascular lesions), melanin (pigmented lesions) or water (skin resurfacing). Since the surrounding epidermis is spared, the treatment is specific with little injury to normal skin. The laser effect is influenced by a number of laser

parameters, including spot size, divergence/convergence of laser beam, wavelength, pulse duration and fluence.

The first two factors (spot size and divergence/convergence) are believed to be optimized in all modern lasers. The *spot size* of a laser affects the optical penetration of light if its radius is equal to or less than the distance for which light is free to diffuse within the tissues. Larger spot diameter will allow greater laser penetration due to greater internal reflection. Furthermore, a larger surface area can be treated faster. Most lasers therefore use a spot size that is greater than the penetrative depth of the laser (>4 mm).

The efficacy of laser treatment also depends on the *convergence* of incident light and the uniformity of irradiance. Convergence of laser light allows for less external reflection from the skin surface and therefore deeper penetration and more selective target injury.

Different chromophores in the skin are targeted by lasers with different *wavelengths*. Wavelength also affects the depth of penetration, with laser light with a longer wavelength penetrating deeper into the dermis.

The most selective target heating is achieved when energy is deposited at a rate faster than the rate of cooling of the target chromophore. Longer *pulse duration* will achieve more efficient heating but if the pulse duration is too long, there will be insufficient time for heat to dissipate, causing an undesirable temperature rise that will lead to damage to surrounding epidermis and dermis. These injuries could result in scarring or other cutaneous side effects. The time taken for heat to dissipate from a particular chromophore (the thermal relaxation time) in seconds is approximately equal to the square of the target's diameter in millimeters.³ In treating vascular malformations, where a variety of abnormal vessel diameters are present at different depths under the epidermis, the setting of an ideal pulse duration for treatment becomes difficult.

In addition to pulse duration, sufficient *fluence* is also required to achieve a damaging temperature in a target chromophore (therapeutic threshold). The use of higher fluence will increase the risk of developing skin complications during treatment.

Laser modality

A laser can be classified as operating in either continuous or pulsed mode. A *continuous* wave laser simply means that the laser emits a constant source of light without interruption, whereas a *pulsed* wave laser emits laser light at a predetermined duration and repetition rate. In a *Q-switched* laser, pulses are only emitted at peak intensity and for extremely short durations.

Most medical lasers are used in pulsed mode, as the targeting of chromophores becomes more specific with less thermal damage to the surrounding tissues due to the short thermal relaxation time of chromophores allowing heat dissipation

between pulses. A pulsed laser is used when the destruction of the target chromophore relies on heat denaturation or coagulation.

In a Q-switched laser, short pulses of high peak power laser light are emitted. Hence, it is used in conditions where a shattering effect is needed to destroy the target chromophores (i.e. pigmented lesions and tattoos).

Laser safety

Lasers can be broadly classified into four classes (Table 5.1). Most medical lasers used are class 4 and hence suitable precautions need to be taken while undertaking laser procedures.

The MHRA (Medicines and Healthcare Products Regulatory Agency) has produced a guideline on the safe use of lasers.⁴ All personnel who are associated with the purchase, supply, installation, use and maintenance of medical lasers and intense pulsed light (IPL) systems should be familiar with the document.

In addition, any hospital that undertakes laser treatment should have local safety rules, with a trained laser safety officer. The local safety rules should be specific to each clinical application and for each laser, and all personnel involved in laser use should be familiar with it. It involves the recommendation that all staff are appropriately trained for the use and maintenance of lasers, safety precautions and adverse events reporting.

Specific safety precautions

- *Eye protection* – Suitable eye protection should be used for each specific laser.
- *Smoke/plume extraction* – Smoke plume may contain hair particles, viable cells, bacteria, viruses, prions and other hazardous materials. Numerous toxic and carcinogenic gases will also be given off. It is therefore essential that precautions are taken to reduce such risks. A well-fitting, high-filtration-efficiency face mask (e.g. particulate respirators that filter particles of 0.1 µm in size) should be used. A standard surgical face mask is not sufficient to act as the primary method of particle filtration. This should be supplemented by a dedicated smoke evacuator. A standard medical vacuum system (e.g. an operating theatre wall suction system) is not suitable for smoke plume removal.

Table 5.1 Classification of lasers

Class 1	Lasers that are not capable of emitting hazardous levels of radiation under normal conditions
Class 2	Lasers that have visible wavelength lasers with lower power output, which will not cause permanent damage to the eye in the 0.25 s it takes to blink
Class 3	Lasers which, if viewed directly, can cause permanent damage to the eye
Class 4	Lasers that produce a hazardous emission, either through direct or scattered radiation. Suitable eye protection must be worn. There is also a potential fire risk

- **Fire hazard** – Flammable substances such as alcoholic skin prep should not be used in areas where lasers are used. Fire extinguishing equipment should be readily available. Care should also be taken when performing laser treatments near nasopharyngeal or endotracheal tubes.

Types of laser

The main types of lasers are divided according to the chromophores they target – oxyhaemoglobin (vascular lesions), melanin (pigmented lesions and hair removal lasers) or water (resurfacing lasers). *Ablative lasers* are resurfacing lasers, such as CO₂ and erbium:YAG lasers, which remove the epidermis. *Non-ablative lasers* selectively target the chromophores within the epidermis or the dermis only. Most lasers that are used to treat vascular, pigmented lesions or hair, and the IPL systems fall into this group. Commonly used lasers along with their wavelengths and target chromophores are listed in Table 5.2.

Fractionated lasers

With the advance of laser technology, fractionated lasers have been developed in the last decade to improve the safety profiles of the traditional ablative lasers. First used in 2004,⁵ fractionated lasers deliver laser beams that produce tiny columns of thermal injury called microthermal treatment zones (MTZs), interspersed with normal epidermis and dermis. As a result, healing is faster than the traditional ablative lasers, with less potential side effects. Both ablative and non-ablative fractionated lasers are available. The advantage of this system is that it allows the resurfacing of non-facial areas and also it allows deep penetration of the laser beam into the dermis, thus encouraging collagen remodelling.

Ablative fractionated lasers can either be based on erbium:YAG for superficial treatments or CO₂ for deeper treatments.

Intense pulsed light

Intense pulsed light (IPL) systems work on the same principle as the non-ablative laser systems. The main difference is that the system delivers a range of wavelengths between 500 and 1200 nm in each pulse of light (non-laser) rather than just one wavelength.

IPL systems work well for vascular and pigmented lesions and also improve mild rhytids. The proposed mechanism of action is thought to be thermal denaturation of dermal collagen, leading to inflammatory changes and subsequent collagen synthesis and remodelling.

When an IPL system is used to treat vascular lesions, no postoperative purpura is seen due to the longer wavelength resulting in more even heating of vessels. Its wide range of pulse width also allows treatment of large and small vessels.

Cutaneous side effects of laser treatment

Temporary

Cutaneous blistering and serous crusting are common after laser treatment. These normally resolve within a few days with no long-term sequelae. Patients are advised to keep the treated area clean.

Skin hyper- and hypopigmentation can occur and is more common in patients with darker skin types. The melanin in the skin competes with other chromophores in absorbing the laser energy, resulting in postinflammatory changes. If it occurs, it normally resolves with time, with or without application of topical skin depigmentation treatment.

Long term

Scarring can occur in severe cases of burn due to laser treatment. There is so far no indication that repeated laser treatment will result in any carcinogenic harmful effects to the skin. To reduce the incidence of cutaneous side effects, it is common for most modern lasers to have an incorporated cooling device. This can either be cold air, cryogen spray or contact cooling device.

In addition, clinicians have to ensure that patients' skin colour is at its lightest when treatment is undertaken. This reduces the melanin load of the skin which can compete with skin chromophores in absorbing laser energy. This can be accomplished by ensuring that patients undergoing treatments are not tanned during treatment, or by temporarily depigmenting the skin before laser treatment with topical products such as hydroquinone.

Table 5.2 Commonly used medical lasers

Medium	Laser	Wavelength	Chromophore
Solid	Alexandrite	755 nm	Melanin
	Nd:YAG	1064 nm (deep lesions)	Tattoo pigment
	KTP	532 nm (epidermal lesions)	Melanin
	Copper vapour	578 nm	Tattoo pigment
	Erbium:YAG	2940 nm	Oxyhaemoglobin
Liquid	Pigmented lesion dye	510 nm	Water
Gas	Carbon dioxide (CO ₂)	10 600 nm	Melanin
			Water

Laser treatment by lesion type

Vascular lesions

Vascular lesions that are responsive to laser treatment include port wine stain, haemangiomas, facial telangiectasia, rosacea, spider naevus, cherry angioma, erythematous scars and warts.

The gold standard *pulsed dyed laser* has a wavelength of 585 nm, and causes vaporization of blood, local rupture of microvessels and purpura. The efficacy of treatment of vascular lesions depends on vessel size, depth and wall thickness. Clinically, dark red or purple lesions tend to be larger and deeper and respond less favourably than the more superficial,

lighter coloured lesions. In treating port wine stain, a poorer response has been observed in lesions found on the extremities. The reason for this is unknown but it may be due to the effect of gravity or deoxygenation on the vasculature or possibly the increased vessel wall thickness of more distal vessels.

After treatment, purpura develops immediately and resolves in 1–2 weeks. Other side effects include scaling (12%), crusting (4%) and scarring (<1%).

Technical tips

- If a smaller spot size is used then higher fluence is needed to achieve similar clinical results.
- The multipass technique has been shown to achieve better results.
- Tissue cooling is important to reduce the incidence of epidermal injury caused by stacking pulses (treating the same spot by firing two pulses sequentially).
- Avoid using 755 nm and 1064 nm lasers around the eyes without metal guards protecting the globe. As they have deeper penetration they can injure the eyes by targeting the retina and iris and blindness has been reported.
- Larger spot size allows deeper tissue penetration.

Other types of laser helpful in treating vascular lesions

- *KTP* (potassium titanyl phosphate; frequency-doubled Nd:YAG laser) – Longer pulse width avoids rupturing target vessels, and hence avoiding purpura.
- *V beam* – Variable pulse width, and can be used to treat telangiectasia without as much purpura by using longer pulse width.
- *Alexandrite laser (755 nm)* – Targets oxyhaemoglobin, and hence effective for bluish/nodular/hypertrophic port wine stains.
- *Long pulsed Nd:YAG* – Longer wavelength, with deeper penetration, and hence more effective in deeper vessels (i.e. veins).
- *Sequential lasers* (e.g. Cynergy) – The first laser (pulsed dye) converts haemoglobin to methaemoglobin, which is then targeted by the Nd:YAG laser. This allows the use of a lower fluence of Nd:YAG.
- *Fractionated lasers* – Can be used to treat resistant port wine stains which has very little chromophore left as well as nodular port wine stains.

Pigmented lesions

Q-switched lasers are most commonly used to treat pigmented lesions. They target melanosomes, causing rapid heating, expansion and rupture. Q-switched frequency-doubled Nd:YAG (532 nm) and the pigmented lesion dye (510 nm) lasers are most useful for superficial epidermal lesions such as freckles (ephelides) and solar lentigines, whereas ruby (695 nm) and alexandrite (755 nm) lasers are of benefit in deeper lesions such as café-au-lait spots and naevi of Ota and Ito.

When using lasers to treat pigmented lesions, patients are always warned that sun exposure can cause the lesions to recur.

This is because the melanocytes that produce the pigmentation are not destroyed by laser treatment. Posttreatment hypopigmentation and hyperpigmentation can occur.

Care should be taken when treating melasma and postinflammatory hyperpigmentation. These conditions generally get worse after treatment.

Tattoos

Q-switched lasers are used to treat tattoos. The laser energy is absorbed by pigment particles, leading to fragmentation of the particles, which are then ingested by phagocytes and transported to the regional lymph nodes.

Black and blue ink absorb all wavelengths well and hence can be treated by a number of different lasers. Different coloured inks absorb different coloured wavelengths so different lasers are indicated to treat different colours: KTP for red, alexandrite for green, and 510 nm pigmented dye for red, orange, purple or yellow.

In general, 8–15 treatments are needed. Patients should be counselled that it may not be possible to completely clear the tattoos. This will result in a ‘ghost tattoo’ where only part of the tattoo is visible. Some patients may find this more unsightly than the actual tattoo itself.

The operator should be aware that in some circumstances ink darkening (reduction of ferrous to ferric oxide or titanium oxide) can occur. This is especially important if the tattoo is of ‘flesh’ colour. Allergic reactions from laser-induced release of tattoo antigens (e.g. chromium-containing red or yellow inks) have also been reported.

Sunblock should be removed prior to treatment as it may contain metal-containing oxides and salts (e.g. titanium dioxide) that may be flammable after exposure to high-energy laser pulses.

To improve results, recently Q-switched lasers with shorter pulse duration (picoseconds) have been used with encouraging results. Such short pulses in high fluence have been reported to be successful in generating a tremendous amount of pigment shattering.^{6,7}

Aesthetic applications

There are three main factors that contribute to an ageing face:

- gravitational effects that give rise to jowls and eyebrow ptosis, which are best dealt with by surgery,
 - reduction in volume, which can be corrected by injection of synthetic fillers or autologous fat; and
 - photodamage, a collective term to describe the effect of sun damage to skin, resulting in damage to collagen and elastin.
- Photodamage leads to the formation of fine wrinkles (rhytids) and poor skin texture and the formation of brown spots due to pigmentary dyschromia, and facial red spots or thread veins due to damage to superficial blood vessels. Facial fine lines can be improved by removing the top layer of skin by chemical peel,

dermabrasion or laser resurfacing. Laser resurfacing is better as it gives precise depth of ablation and controlled thermal damage with the added benefit of skin tightening. In laser resurfacing CO₂ and erbium lasers are used. Energy is absorbed by intracellular water, causing rapid heating and vaporization of tissue.

Most published series have reported an overall 90% improvement in rhytids following laser resurfacing. Most improvement can be expected in the perioral area and least in areas of dynamic wrinkling such as the glabella. The results decrease with time, probably due to delayed resolution of postoperative oedema (down to 31% in glabella in 2 years in one study).

In acne scarring, the maximum improvement that can be expected after one treatment is 30–50%. If resurfacing is used to treat pigmentary changes or lesions, there is a chance of recurrence.

CO₂ lasers

The CO₂ laser has a wavelength of 10 600 nm and is highly absorbed by water. It was first introduced in 1964, in continuous mode for cutting and coagulating tissues. The development of a high-energy pulsed mode in the 1990s expanded its use and made it the gold standard in skin resurfacing.

A conventional CO₂ laser deposits energy in the upper 20 µm of skin (after one pass) due to strong water absorption. There can be 0.2–1 mm of residual thermal damage, leading to prolonged downtime with long periods of wound healing. Re-epithelialization usually takes about 7 days with prolonged erythema that can last more than six weeks. Such thermal damage also leads to a high incidence of scarring and skin pigmentation changes. The thermal damage has an additional benefit of causing skin tightening.

In the mid 1990s, to reduce thermal damage and its associated side effects, modification was made to the CO₂ lasers so that only pulses that are shorter than the thermal relaxation time (1 ms) were used. This was achieved by either the use of high-energy, short-pulsed (<1 ms) CO₂ lasers (Ultrapulse), or a focused beam with rapid scanning resulting in contact time of <1 ms (Silktouch/Feathertouch/Sharplan).

Erbium:YAG lasers

The erbium laser was first used in 1996. It has a wavelength of 2940 nm and hence has 16 times higher water absorption compared to the CO₂ lasers. It penetrates 1 µm of skin (versus 20 µm for CO₂) and offers more precise skin ablation. It causes less thermal damage than the CO₂ lasers, and hence has a lower incidence of skin complications such as prolonged erythema and skin pigmentary changes. Due to the less significant thermal effect it offers poorer haemostasis and less skin tightening when used as a resurfacing tool.

Fractionated CO₂ lasers

To minimize the thermal side effects of the CO₂ lasers, fractionated CO₂ lasers were recently developed. They deliver evenly spaced thermal injury, surrounded by healthy skin, which

enables rapid epithelialization. Depending on the fluence used, re-epithelialization usually occurs between 3 and 6 days, and the side effects associated with thermal injury, such as prolonged erythema and skin pigmentary changes, are less common than with non-fractionated lasers. In addition, because intact skin is interspersed with ablated skin, the depth of ablation can be increased without the fear of scarring. This is useful in upregulating skin collagen production, which is most useful in skin tightening⁸ and improving atrophic scars.⁹

Histological examination of fractionated CO₂ laser-treated skin shows columns of thermally damaged skin with immediate inflammatory response, followed by reorganization of dermal matrix and collagen induction.¹⁰

Fractionated CO₂ lasers also allow resurfacing of areas outside the face, which is not possible with traditional resurfacing lasers. It also allows treatment of darker skin types without a high risk of skin complications.

There is some evidence that fractionated CO₂ lasers improve skin hypopigmentation and hyperpigmentation, as seen in conditions such as melasma. This is achieved by facilitating migration of melanocytes from neighbouring skin, and changing the optical characteristic of skin by increasing production of collagen.¹¹

The effects of fractionated CO₂ laser resurfacing depend on the depth of laser penetration, dermal coagulation and spacing. Normally 2–4 treatments are needed, at intervals of 6–12 weeks.

Preoperative consultation

It is important to elicit the patient's expectation from treatment, as well as their acceptance of treatment downtime and side effects before starting treatment. In preoperative history taking, the patient's skin type and scarring history must be noted. Roaccutane should be avoided for 9–12 months prior to treatment as it may compromise skin healing. If the patient has a history of cold sores, antiviral therapy should be started prior to laser resurfacing (aciclovir 400 mg three times daily from 24 h prior to treatment, and continued for 5 days afterwards). Treatment should ideally be offered when the patient has not had any recent significant sun exposure. Treatment of tanned skin may result in unfavourable pigmentation.

Patients should be warned of possible complications, including acne flares, prolonged oedema, erythema, postinflammatory skin hyperpigmentation and skin hypopigmentation.

Postoperative care

I favour the open technique, where no dressing is used to the treated area. The patient is encouraged to wash the area as normal in the shower, and keep the area clean. Skin oedema usually peaks at 48 h, and resolves over 6–8 weeks. Elevation and application of ice will help. Some patients experience pruritus, usually in the first 5 days. A cold compress or emollients will help. Erythema usually resolves gradually over 2–5 months, but may last for more than 3 months in 12–31% of patients. Patients should be encouraged to use total sunblock during this period of time.

Complications

- Infection (1–2%) – highest risk days 2–10. *Pseudomonas* (41%), *Staphylococcus aureus* (35%), *Staph. epidermidis* (35%), *Candida* (24%).
- Pain and itching.
- Follicular occlusion leading to acne or milia (up to 84%).
- Hyperpigmentation (30–40%), usually occurs 2–6 weeks post op, only lasting more than three months in 18% of cases. The incidence is increased with darker coloured skin (86–100% skin type IV and V). Sunblock/depigmenting agents can be used.
- Hypopigmentation (8–19%) – may not be evident until seven months post op. This reflects the degree of pretreatment photodamage and bronzing, as well as aggressiveness of resurfacing.
- True hypopigmentation – due to decreased melanogenesis.
- Pseudohypopigmentation – relative lightening of skin compared with surrounding untreated sun-damaged skin.
- Scarring (2–3%).

Laser hair removal

Laser hair removal is now the standard in long-term hair control.¹² Hair removal lasers with a wavelength of approximately 700 µm direct laser light precisely to each melanin-containing hair follicle. Several lasers are effective: the ruby laser (694 nm), alexandrite laser (755 nm), Nd:YAG laser (1064 nm) and the IPL source (590–1200 nm). Although the mechanism of follicular injury is likely to be thermal, the precise contributions of photo-mechanical damage or thermal denaturation to follicular injury are unknown. It is possible that after absorption of radiant energy, the large temperature difference between the melanosomes and their surroundings produces a localized rapid volume expansion, leading to microvaporization or ‘shock waves’, which cause structural damage to the hair.

Studies on the efficacy of laser hair removal show that long hair-free periods are achievable with all of the above lasers. Several treatments are needed, usually at 4–6 weekly intervals when the hair has regrown. The hair-free period is variable after a course of 6–8 treatments, but usually ranges from 3 to 6 months. Permanent hair removal is rare. Complications from treatment are usually uncommon, especially if a test patch is performed prior to full treatment, and is more common in darker coloured skin. The most common complication is redness after treatment, which lasts a few hours. Less commonly patients can expect crusting of skin, and skin hypo and hyperpigmentation. Most of these complications are temporary, with skin pigmentation changes usually recovering in 6–9 months.

The use of the Nd:YAG laser is more beneficial in patients with darker coloured skin. The absorption of laser light by melanin is low at 1064 nm, and hence will cause less heating to the epidermal melanin. The low melanin absorption is

somewhat counteracted by the deeper penetration of the laser light at longer wavelength, and the use of longer pulsed duration ensures the longer period of heating of the melanin in the hair follicles.

Other indications for laser therapy

- *Resistant warts* – Flat warts can be treated with the pulsed dye laser. Thick warts can be treated with ablative lasers. In the case of recurrence, topical treatment or pulsed dye laser can be considered.
- *Keloid scars* – Erbium laser ablation, followed by regular Adcortryl injection.
- *Scar resurfacing* – Fractionated CO₂ laser as per aesthetic resurfacing.
- *Cutaneous malignancies and premalignancies* – Premalignant conditions such as actinic keratosis and Bowen’s disease respond well to laser ablation, especially with the erbium laser. Erbium laser is also useful in treating superficial basal cell carcinoma, especially in patients with Gorlin syndrome, where repeated surgery can be disfiguring. The CO₂ or erbium lasers are useful in treating superficial in-transit melanoma.
- *Kaposi’s sarcoma* – Pulsed dye laser, photodynamic therapy, Nd:YAG and CO₂ lasers have been used with varying success to treat Kaposi’s sarcoma.

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CHAPTER 6

Tissue expansion

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Background

The process of tissue expansion in reconstructive surgery was first reported by Charles Neumann, who described its application for auricular soft tissue reconstruction in 1957.¹ Neumann simply applied concepts found in both normal physiological as well as pathological processes of the human body. Examples of this phenomenon include pregnancy, where the abdomen increases in size to accommodate the enlarging fetus, and rapid neoplastic growth, which may result in soft tissue expansion, deformity and eventual skin breakdown. African tribes have also adopted tissue expansion as part of their cultural background, enlarging facial structures such as the lip or the ear.^{1,2}

Nearly half a century later, tissue expansion was repopularized by Radovan for use in postmastectomy breast reconstruction,³ although its applicability soon became evident in other branches of plastic surgery. Tissue expansion enables soft tissue coverage of large defects secondary to burns, trauma, congenital malformations and cancer excision. It involves the insertion of an implant adjacent to a wound or defect that needs to be resurfaced. The implant is then incrementally enlarged by injection of isotonic saline, resulting in stretching of the overlying soft tissue. The expanded skin can then be used to resurface a defect or incorporate permanent prostheses, including those used for postmastectomy breast reconstructions. Tissue expansion exploits the principles of controlled mechanical skin overstretch in order to generate new tissues with ideal compliance and almost perfect skin colour, texture match and sensibility to the resurfaced defect.⁴

However, this useful tool on the reconstructive ladder does not come without its complications. In this chapter, we review the effects of tissue expansion on soft tissues and explore the tenets that govern its safe and effective use in the patient.

Viscoelastic properties of skin

The structural components of skin can be altered by expansion as demonstrated by several biomechanical studies. Its viscoelasticity means that the mechanical response to loading involves both a viscous (time-dependent) and an elastic (time-independent) component.^{5–7} Skin is mainly composed of dermis and epidermis. The predominant proteins in the dermal layer are collagen types I and III, which are produced by fibroblasts. In the relaxed state, their triple helical structure results in a wavy and convoluted alignment. Upon stretching, the collagen fibres straighten and realign parallel to each other.⁴ The skin's elasticity, essential for joint movements, is due mainly to superficial thin bundles of microfibrils associated with progressively larger amounts of amorphous elastin to form elastic fibres.^{8,9} Once strain is released, elastin fibres return collagen to its relaxed coiled posture.¹⁰ If stretched excessively, elastin is prone to fragmentation, resulting in a loss of recoil.¹¹ Viscoelastic properties change with age and repeated mechanical forces, resulting in skin laxity.⁵

Skin is a dynamic organ and acclimatizes to constant mechanical forces applied to it due to its viscoelastic properties (Table 6.1). On a tissular and cellular level, constant mechanical stress induces changes termed mechanical creep and biological creep. These phenomena interact to restore resting tension of stretched tissues to baseline – stress relaxation (Figure 6.1).¹² For example, a wound which was under tension intraoperatively but relaxed at outpatient follow-up is an example of stress relaxation.⁴

Creep is defined as the stretching of skin under a constant load over time. In order to explain the biomechanics of skin during the process of tissue expansion, a distinction was made between mechanical and biological creep.¹³ Mechanical creep

*These authors contributed equally to this chapter.

Table 6.1 Summary of the viscoelastic properties of skin

Mechanical creep	The elongation of skin under a constant load over time Collagen fibres stretch out and become parallel Elastin undergoes microfragmentation Interstitial fluid is displaced (e.g. during intraoperative tissue expansion and skin suturing under tension)
Biological creep	The generation of new tissue secondary to a persistent chronic stretching force (e.g. seen during pregnancy)
Stress relaxation	The tendency for this resistance of skin to a stretching force to decrease when held at a given tension over time

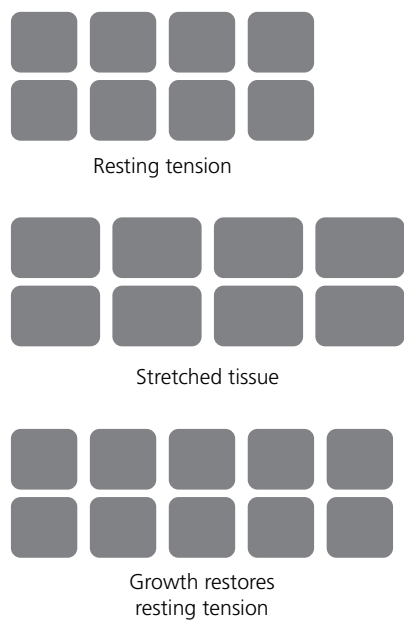


Figure 6.1 Principle of tissue expansion. Source: De Filippo *et al.*, 2002.¹² Adapted with permission of Lippincott Williams & Wilkins.

describes the clinically observed elongation of skin beyond the original stretch when placed under sudden constant tension and is thought to be attributable to the morphological changes (stretching) of the dermal framework in response to stress. Upon stretching, the formerly convoluted collagen fibres straighten and realign parallel to each other, resulting in the extrusion of fluid from the collagen network and microfragmentation of elastic fibres, thus making the skin more viscous.¹³

Biological creep occurs with the formation of new tissues as a result of a persistent chronic stretching force in an attempt to relieve the stress (stress relaxation). It underlies the mechanism of gap junctional disruptions, which results in cellular proliferation. Several studies have confirmed that the gain in skin surface area after expansion was caused by new skin formation rather than simply stretching the pre-existing skin. Over a period of 6–12 weeks, a multitude of growth factors, cytokines, adhesion molecules and signal transduction proteins are induced to stimulate epidermal thickening, with concurrent dermal thinning

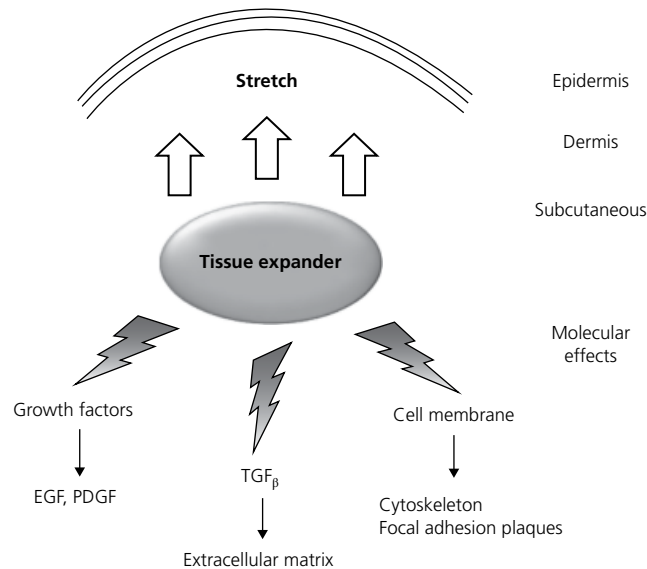


Figure 6.2 Effects of tissue expansion on surrounding tissues. Stretching of surrounding tissues induces growth factors, which mediate cell proliferation and increased angiogenesis. Factors such as TGF- β influence extracellular matrix production while membrane-bound protein kinases, focal adhesion points and the cytoskeleton influence intracellular signalling cascades. Source: Takei *et al.*, 1998.¹⁴ Reproduced with permission of Lippincott Williams & Wilkins.

and collagen fibril alignment of the newly expanded tissues (Figures 6.2 and 6.3).¹⁴ In fact, a study conducted by Vander Kolk *et al.* demonstrated a significant increase in collagen production in expanded skins compared to control skins.¹⁵ Additionally, growth factors including epidermal growth factor (EGF), transforming growth factor beta (TGF- β) and platelet-derived growth factor (PDGF) are upregulated to stimulate neoangiogenesis of the expanded tissues. Deformational forces acting on the cell membrane and disruption of integrins reduce cell-matrix adhesion and induce conformational changes of membrane proteins. Calcium channels open, leading to signal transduction and cytoskeletal microfilament contraction. The actin cytoskeleton then transmits mechanical forces intracellularly, resulting in increased gene expression and mitosis via interaction with protein kinases, second messengers and nuclear proteins.^{16–19} Cellular proliferation then results in tissue enlargement.

Enhanced vascularity is among the most noticeable advantages of expanded tissues, exhibiting both higher vessel numbers and larger calibres compared to non-expanded counterparts.^{20–23}

Pietramaggiore *et al.* demonstrated that cyclic mechanical forces applied to the skin of living rats stimulated cell proliferation and vascular remodelling.²⁴ Additionally, a recent study by Erba *et al.* supported the angiogenic role of vascular endothelial growth factor (VEGF) in cyclic dermal stretching.²⁵ Stretched skin presented with more densely packed vessels as well as a significantly increased thickening. However, the most important change during tissue expansion is tissue proliferation secondary

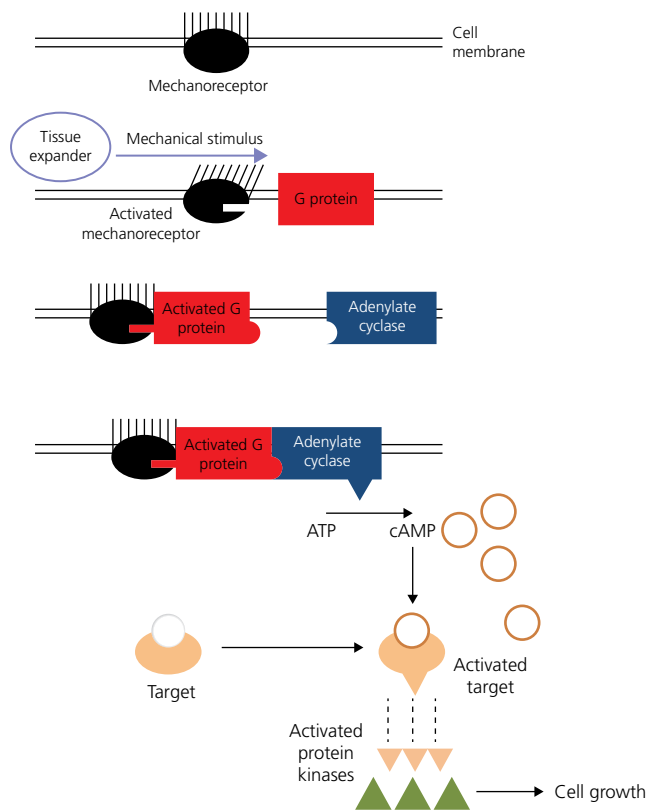


Figure 6.3 Molecular cascade in response to tissue expansion resulting in cellular proliferation. Source: De Filippo *et al.*, 2002.¹² Reproduced with permission of Lippincott Williams & Wilkins.

to stress-induced cellular changes (biological creep). Sheng *et al.* demonstrated that transplantation of angiogenic growth factor-secreting cells into expanding skins of rats resulted in accelerated tissue expansion, enhanced neovascularization of the expanded skin and promoted cell proliferation.²⁶ Expression of EGF, VEGF and basic fibroblastic growth factor (bFGF) were significantly enhanced in treated animals compared to control animals. Growth factor supplementation may well play an important role in the future of tissue expansion, both in reducing hospitalization times and flap retraction after expansion. Current research aims at evaluating the best possible source of growth factors. Wang *et al.* demonstrated increased tissue regeneration efficiency and reduced skin shrinkage when expanding tissues were exposed to bone marrow mesenchymal stem cells (BMMSCs).²⁷ The gene expression of important growth factors such as VEGF, bFGF, EGF and stromal cell-derived factor (SDF) was significantly enhanced in exposed tissues compared to sham treated tissues. Local administration as opposed to systemic infusion of BMMSCs even further enhanced growth factor expression, which is supportive of the substantial role these factors play in tissue regeneration following expansion. Because of their relative inaccessibility within the bone marrow and the invasive nature of BMMSC harvest, scientific research looks to alternative sources of growth factor-secreting stem

cells. Adipose-derived stem cells (ADSCs) are ubiquitously available and have several advantages over other sources. They are easily accessible in large quantities, can be obtained via a minimally invasive harvesting procedure and their isolation yields a high amount of stem cells. Several studies demonstrated that wound supplementation with ADSCs significantly accelerated regeneration.^{28–30}

Tissue changes during expansion

In order to fully understand the mechanics involved in tissue expansion, we will first familiarize ourselves with the structural components of skin. Skin is a multilayered structure and has three well-defined anatomical regions.^{31,32} The outermost layer, the epidermis, forms a thin, uninterrupted and protective layer across most of the body's surface but contributes little to the skin's mechanical properties.⁷ The bottom layer, the hypodermis, is mainly made of fatty tissues and serves as an insulating layer. The middle dermal layer holds most of the skin's adnexae, including nerve endings, sweat and oil glands and consists of three main structural components that underlie the skin's strength and elasticity: collagen, elastic fibres and extrafibrillar matrix. The dermis can be further divided into papillary and reticular layers. The papillary dermis contains thin collagen fibrils which are packed together into thicker collagen fibres (Figure 6.4). Only about 15% of the dermis is type III collagen, most of which is found in the papillary layer. Collagen type I, the most abundant building block in vertebrates, is the principal protein providing tensile strength to the skin.^{33,34}

Austad *et al.* assessed the effects of tissue expansion on the mitotic activity in the epidermis in order to address the question whether there is a net gain in epidermal tissue.¹³ Their studies confirmed that tissue expansion caused a marked increase in epidermal mitotic activity in a guinea-pig model. Studies on 17 human volunteers supported this finding as Pasyk *et al.* demonstrated a significantly thickened epidermis of expanded human skin compared to non-expanded skin.¹⁴ Although expanded tissues maintain their phenotypic characteristics, mechanical strain induces an increase in DNA synthesis and therefore cellular proliferation.^{7,15} Biochemical pathways involved in this process include cytoskeletal, extracellular enzyme, membrane and cytosolic components that converge together to convert the pathway of mechanotransduction seen in tissue expansion into a cellular response, ultimately leading to cellular proliferation.¹⁵

The dermis, in contrast, becomes thinner upon stretching, with hair follicles moving further apart. It may take up to 2 years for the thickness of this layer to return to normal. In keeping with animal models, Pasyk *et al.* assessed the effect of expansion on the dermis in 14 patients and identified significant thinning of the dermis.¹⁴ This, however, was not found to be rate-dependent. Furthermore, dermal thinning after expansion was noted to be equal in children and adults.

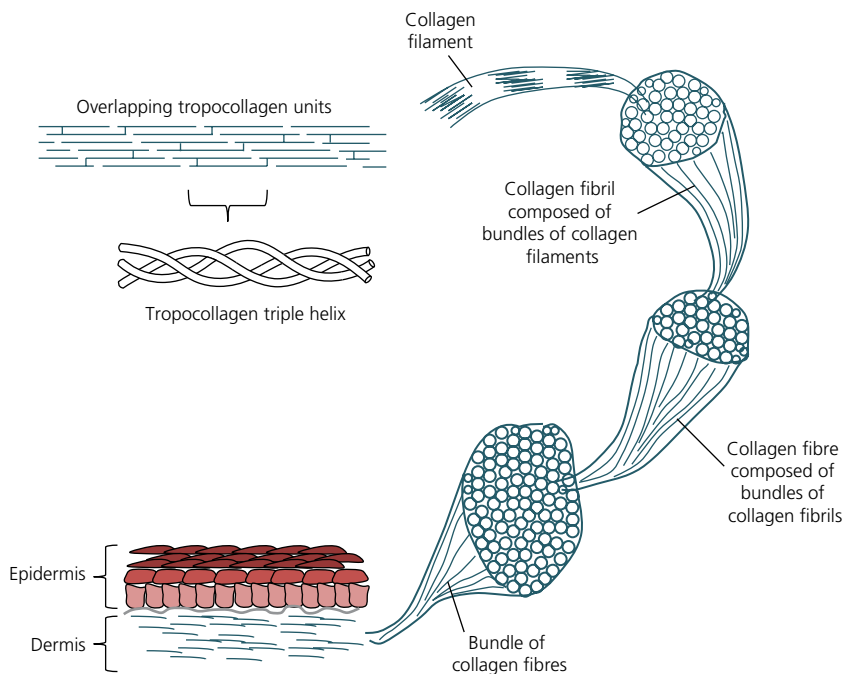


Figure 6.4 Schematic illustration of the composition of collagen in the dermal matrix.

Subcutaneous fat tissues were also found to thin after expansion. Adipocytes were flattened, surrounded by fibrosis, or disappeared altogether during the expansion process. Similarly, skeletal muscles are known to undergo atrophy after expansion resulting in the so-called 'bath-tub depression'. Histologically, the atrophic muscle cells are surrounded by fibrous tissue and demonstrate abnormally arranged sarcomeres, large amounts of sarcoplasm and also numerous large mitochondria.¹⁴ As opposed to the changes in muscle tissues, the loss of subcutaneous fat is permanent.

Enhanced angiogenesis in expanded tissues versus non-expanded counterparts is of clinical significance and presents an advantage in flaps raised on such skin. Improved survival of flaps raised on expanded skin shows properties similar to a delay phenomenon. This was confirmed by Cherry *et al.* who, using a pig model, elevated random pattern flaps in expanded tissues or non-expanded tissue to determine whether these flaps survived for the same time intervals.¹⁷ Flaps raised on expanded tissue had the greatest increase in surviving length, averaging 117% over control flaps, which was in large part due to an increased vascularity as well as vasodilatation in the angiograms of skins expanded over a period of five weeks.¹⁷ The pressure and mechanical stresses applied to expanding tissues resulted in tissue hypoxia, which induced the formation of an increased microcirculation in expanded tissues.¹⁸ Sasaki *et al.* also demonstrated that flaps based on expanded tissue survive to a greater length than non-expanded or delayed as undermined bipedicle flaps.¹⁹ Increased vascularity is thought to result from increased gene expression and significantly higher levels of angiogenic factors such as VEGF in expanded tissues compared to non-expanded controls.^{20–22}

Rapid rates of expansion have further been demonstrated to increase flap microvasculature compared to slowly expanded

tissues. However, it should be noted that rapidly expanded tissues may become profoundly hypoxic up to 48 h which may compromise flap survival.¹⁸ In addition, significant patient discomfort accompanying rapid tissue expansion may limit the rate of expansion.

Types of devices

Each tissue expander needs to be carefully selected for its desired purpose, making tissue expansion a highly individualized procedure. Depending on the size and shape of the defect to be covered, different expander types may be selected (Figure 6.5). The basic design is an inflatable silicone elastomer reservoir.² Isotonic saline is delivered in a controlled fashion via a valve port system which is either integrated into the expander or connected to the prosthesis via silicone tubing (Figure 6.6). An integrated system offers the advantage of undermining a single pocket for expander placement, but also places the implant at risk of perforation by a misplaced needle. Internally placed remote ports remove the danger of expander perforation, but introduce potential complications, including kinking of the port, tube obstruction and migration. To avoid these complications, the undermining for the port pocket is minimal, the port is placed over firm, supporting tissue and, if need be, the port position can be fixed with sutures.³⁵ Externally placed portals have proved particularly useful in the paediatric population as they may alleviate pain and anxiety associated with frequent injections²⁴ and the safety of such ports has been demonstrated repeatedly.²⁵

The shape of the expander is governed by the anatomical location and may, if necessary, be custom made to fit any size or

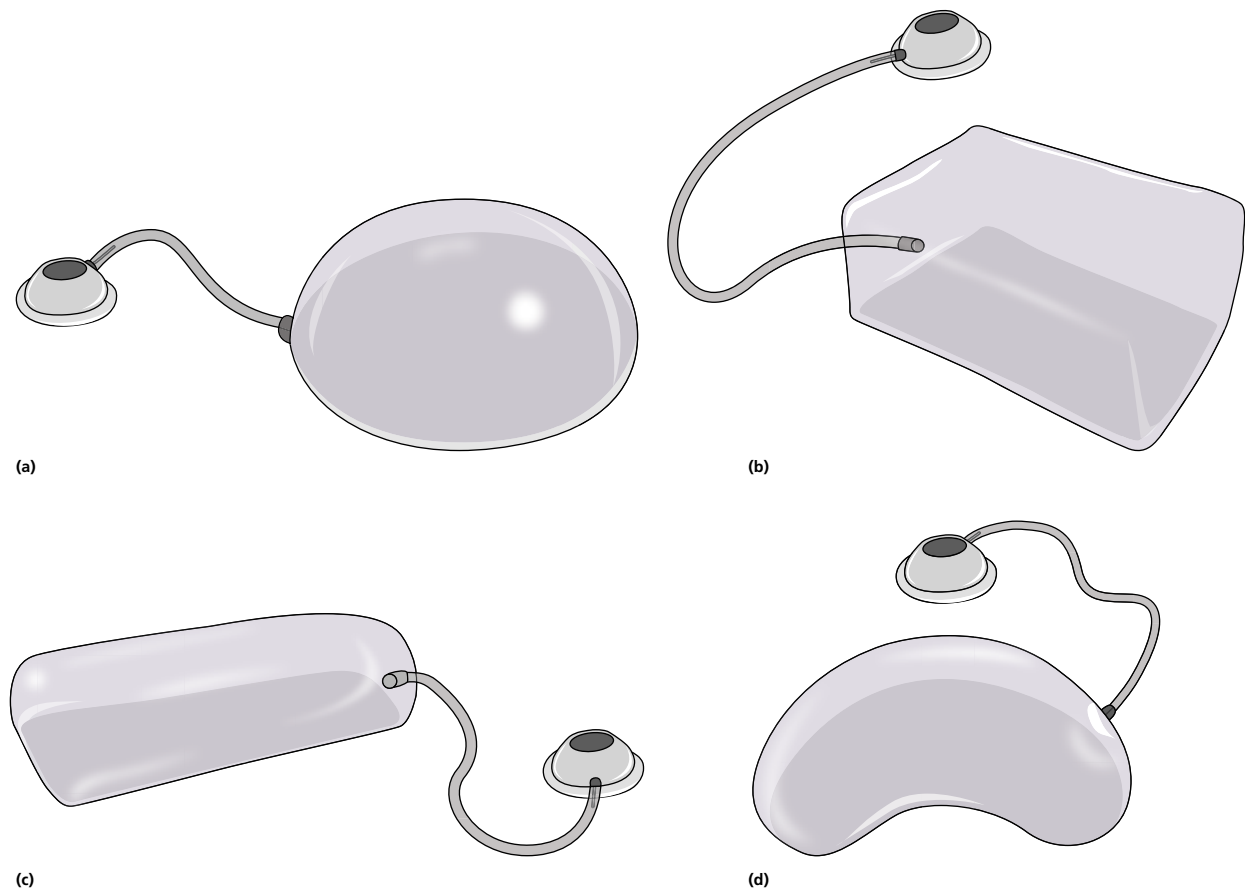


Figure 6.5 Variation in shape of tissue expanders: (a) spherical; (b) rectangular; (c) cylindrical; (d) crescent.

shape. Circular expanders are best suited to areas such as the breast, whereas crescentic and rectangular shapes have been described for the scalp and lower limbs, respectively.² A further parameter governing tissue expansion involves directional expansion in order to impart a certain contour to the expanded pocket.⁸ This may be of importance if a specific shape is required, such as for breast reconstructions.

In 1982, Radovan successfully applied a temporary tissue expander for postmastectomy breast reconstruction.³⁶ Such procedures, however, require a second surgical intervention to remove the expander and replace it with the final breast implant. Two years later, Becker described a permanent expander for breast reconstruction where a modified inflatable breast implant enables a reservoir to be attached and detached at a side-filling valve.³⁷ The implant therefore functioned both as a tissue expansion device and a permanent implant upon removal of the reservoir. In a more recent study, the Becker implant was compared prospectively to the two-stage expansion process in a randomized study and patient satisfaction was measured.³⁸ Seventy patients were randomized to receive either a round permanent Becker implant ($n = 35$) or undergo a two-stage reconstruction with a crescent-shaped expander ($n = 35$), later replaced by an anatomical implant. Thirty patients were excluded and therefore only 20 patients per group were evaluated. A

significant 70% of patients in the Becker implant group underwent revision surgery secondary to upper pole fullness and poor ptosis. It was therefore concluded that a two-stage procedure achieves a better aesthetic outcome.

In an effort to minimize these traditional disadvantages of conventional tissue expanders, a new generation of pressure-sensitive self-expanding devices was first described in 1982. Austad and Rose created a permeable silicone balloon filled with a hypertonic NaCl solution which drew in body fluids down the osmotic gradient.³⁹ Despite being described as having some value for soft tissue reconstruction, these osmotic expanders are rarely in use nowadays due to major disadvantages.⁴⁰ First, there were relatively long inflation periods of 8–14 weeks. Second, high incidences of balloon rupture during the early stages resulted in necrosis of the overlying tissues.⁴¹ More than a decade after its initial conception, Wiese developed an osmotically active expander that was completely independent of the use of saturated saline. The implants exploited the osmotic forces of hydrogels, which consist of two components: a polymer network with constant quantity and an aqueous component that is variable.⁴² The gel is placed subcutaneously and absorbs body fluids from the extracellular space. Water uptake then causes gradual swelling of the hydrogel. The theoretical advantages of self-inflating expanders, including less frequent hospital visits and

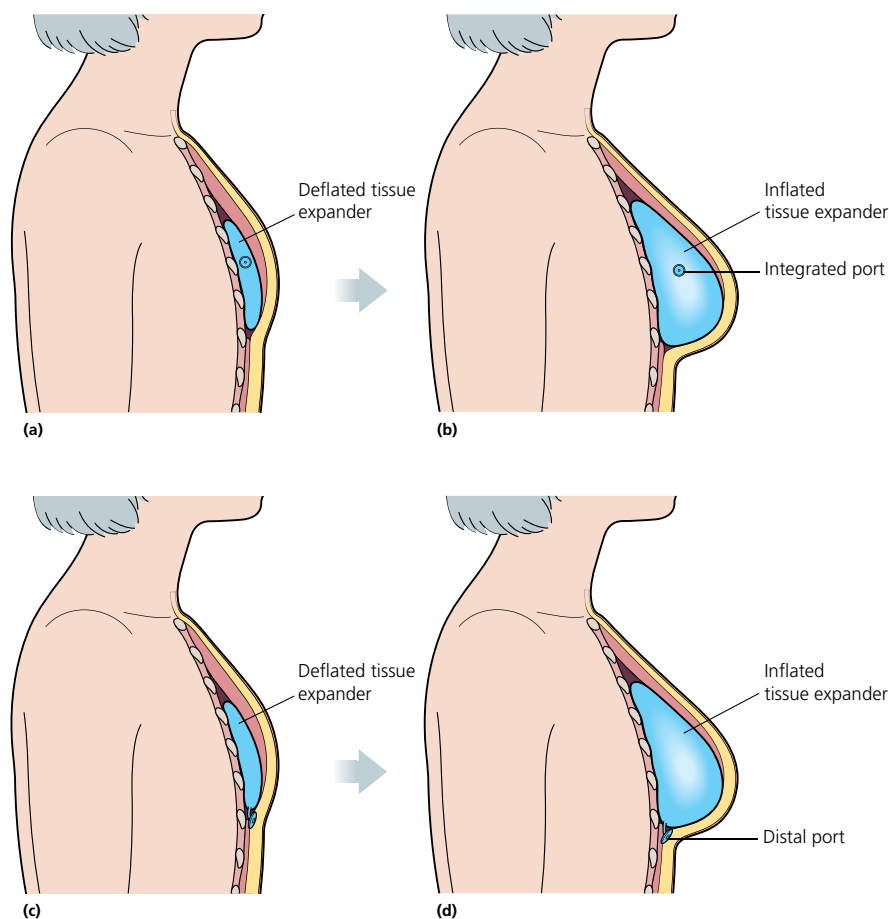


Figure 6.6 Schematic illustration of submuscularly placed tissue expanders. (a) An integrated injection port system allows saline injections directly into the expander whereas saline is injected into a distal port in (b).

injections as well as reduced anxiety and pain, led to their commercialization and distribution by Osmed (Ilmenau, Germany).

However, a recent case series using Osmed self-expanders conducted by Chummun *et al.* demonstrated high incidences of expander infection among the paediatric population (2 out of 7, 28.5%), device migration and in some cases tissue ischaemia and necrosis.⁴⁰ The fact that a small study population and single-centre study was selected should be taken into account before prematurely dismissing self-expanding implants, however.

Basic incision and design plan

Tissue expansion requires meticulous preoperative planning, careful placement of the expander and suitable incremental inflation. Without these sequential steps the final result would be compromised.^{43,44} Placement is dictated by where the proximity of the defect is situated and this must be determined in advance after careful consideration of all possibilities. Easily recruitable sites of high tissue laxity should be determined before selecting skin of similar colour, texture and hair-bearing quality to the recipient site.⁴⁴ Tissue expansion may take up to 3–6 months depending on the anatomical site and the size of the expander.² Breast and abdominal tissues can tolerate large expansion volumes in one sitting, while forehead and scalp

expand more slowly.² The characteristics common to ideal expander placement are: (a) easy accessibility through a small incision, (b) gradual enlargement over a relatively short time period, (c) no painful inflation spikes, (d) good long-term biocompatibility with the body, (e) resistance to infection and extrusion, and (f) no need for revision procedures.⁴⁵

Selection of the ideal type, size and shape of the expander requires some foresight in terms of the timetable for the inflation process, which must be individualized to the patient in question.¹⁵ Radovan advised that the expander base should have the same size as the defect to be reconstructed as this allows for doubling of the surface area, enabling eventual coverage of both the donor and recipient sites.³ In contrast, other investigators postulate the use of as large a tissue expander as possible, however without empirical evidence.^{43,46}

In an effort to prevent arbitrary expander selection, several mathematical models have been developed to precisely calculate necessary dimensions. Early computational programs required the implant base diameter and the desired surface area gain to estimate the capacity of a spherical tissue expander.⁴⁷ More sophisticated programs are able to calculate the volumes of rectangular tissue expanders after feeding the length and breadth of the lesion to be repaired, considering them the size of the base of the expander. Raposio *et al.* incorporated a 'correcting factor' by multiplying the surface requirements by 3/2 to account

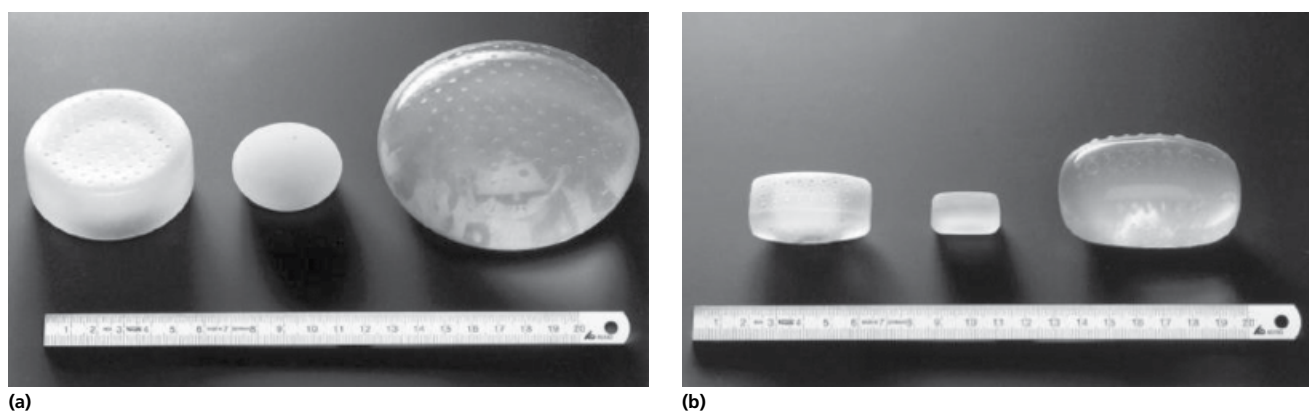


Figure 6.7 Round (a) and rectangular (b) osmotic tissue expanders. The hydrogel (middle) is wrapped inside a loosely fitting silicone membrane (left) that contains pores for finely tuned water uptake.

for potential tissue loss during tissue transfer.⁴⁰ However, software-based predictions of tissue expander size and shape are complicated by factors such as the surface on which the expander is placed upon. If placed over rigid flat surfaces, predicting the outcome of tissue expansion is straightforward. The difficulty occurs when expanders are placed over soft tissues with the tendency for inward compression or in areas of concavity, since the prediction of tissue expansion is less reliable. Due to such complexity, mathematical models failed to cross over to clinical routine where the trained surgeon's eye remains the ultimate, still arbitrary, judge.^{37,38}

The primary incision is best placed perpendicular to the direction of expansion to avoid tension across the incision, which could result in wound dehiscence and broadening of the scar line.⁴⁸ The ideal type of initial incision is still a matter of debate. However, no debate surrounds the issue of the ideal size; regardless of the type of incision, it should be as small as possible. Incisions can be made either at the junction of normal tissue and scarred area (i.e. paralesional incision) or remotely, so healthy tissue approximates healthy tissue. Paralesional incisions offer the advantage of minimal undermining of tissues for the insertion of expanders and their rates of infection, wound dehiscence and implant exposure do not seem to be statistically different to those in remote incisions. The latter, however, may offer better healing. There is, as yet, no final consensus as to which type of incision is definitely superior. This calls for controlled, randomized studies on site-specific expansions.

Another important consideration is the plane into which the expander is placed, as minimizing stress on overlying skin flaps aids during procedures such as mastectomies and increases the flexibility in choosing the final breast volume⁴⁸ (Figure 6.7). Before placement of the expander into the pocket, meticulous haemostasis is warranted and a closed system suction drain is put in place. Irrigation with povidone-iodine and/or gentamycin/cefazolin is commonly performed to minimize the chance of infection, followed by intravenous administration of antibiotics effective against skin flora.^{43,49}

After expander placement, the expansion process is usually delayed to allow the wounds to heal and reduce the chances of wound dehiscence.⁵⁰ This also allows for any swellings to subside and problems with the skin flap to reveal themselves.⁴³ The expander is filled until the patient feels some discomfort or the overlying skin blanches, but excessive tension on the expander flap should be avoided as tissue necrosis may ensue. Finally it should be noted that despite meticulous planning and appropriate technique expansion may not be successful. Success is also dependent on patient comorbidities, nutritional habits and local tissue factors.² Finally, patient cooperation to the expansion procedure is vital, as the process requires several interventions and involves many visits to the outpatient clinic.

Advantages and disadvantages of tissue expansion

Tissue expansion has many notable advantages to other plastic and reconstructive techniques. The expanded tissue usually provides sufficient soft tissue coverage for both the recipient as well as donor sites. Tissues from locally expanded sites allow same colour, texture, sensation and quality skin to be used in resurfacing a defect, thus avoiding the use of split-thickness skin grafts which are associated with more recipient and donor site morbidities^{15,51} (Figure 6.8a,b,c). Additional advantages include the significantly higher vascularity of expanded tissues providing more robust skin coverage.

Disadvantages of tissue expansion include the long timeframe of up to several months for the expansion process to finish and multiple outpatient visits. Most commonly, a two-stage procedure is necessary – one to place the expander in and a second one to replace the temporary implant with a more permanent one. Significant complications may result in multiple interventions. Further disadvantages include the frequent outpatient visits for incremental tissue expansion which can be particularly uncomfortable on the forehead and distal extremities. There is also a



(a)



(b)



(c)

Figure 6.8 (a) Expansion process used to reconstruct a large defect secondary to vascular malformation. (b) Explanted expanders in same patient. (c) Final result after advancing expanded tissues and excision of vascular malformation. An optimal colour and skin quality match has been achieved.

psychological impact of tissue expansion as there is a temporary disfigurement that can last for many months before the expander is removed.² Finally, tissue expansion is not without risks, and many large case series have noted major complications such as infection or implant exposure. A summary of associated complications is given below.

Complications

Tissue expansion, like any surgical procedure, comes with its own set of complications. In 1984, Manders *et al.* summarized their experience with 41 expanders in 35 patients and divided the observed complications into major and minor categories.⁴⁶ Major complications include infection, implant extrusion and flap ischaemia and may interrupt the expansion process. Minor complications, including seroma formation, scar widening, dog ear formation, transient pain and neuropraxia, may resolve without failure of the procedure.

Antonyshyn *et al.* performed a critical review of their results on tissue expansion over a 5-year period.⁵⁰ Sixty-six expansions were performed on 66 patients. Complications were identified and analysed based on their anatomical location, time of onset and detrimental results on the final outcome. Interestingly, the authors of this study noted some common sequelae of tissue expansion which, strictly speaking, were not complications. Transient pain following implant inflation was a commonly observed side effect, which was particularly associated with expansions of the distal extremities, dorsal trunk and forehead. Clinically significant complications were most frequently observed with head, neck and lower extremity expansion (39% in their case series) but generally did not affect the final outcome and excellent results were noted in 83% of cases. More serious complications tended to occur at the final stages of expansion with implant failure, neuropraxia, bone resorption and late implant exposure being highlighted. Haematoma formation requiring prompt evacuation was observed in only one patient. Similarly, cellulitic infection of the skin covering

the reservoir dome was observed in one patient only and was successfully treated with intravenous antibiotics. More severe complications, including infections involving the implant cavity and implant exposure secondary to incisional dehiscence, erosion through inadequate soft tissue cover and pressure necrosis of soft tissues, were observed more frequently.

Pandya *et al.* assessed complication rates of expansion at non-limb versus limb sites in a retrospective case series of 88 patients.⁵² The overall incidence of complications was higher in the limb group (43%) compared to other sites (27%). Lower limb complications were also more frequently observed than upper limb complications (47% versus 30%). They noted that exposure or infection of an implant on the extremity was not necessarily a sign of preceding failure. In these circumstances, expansion was suspended, appropriate antibiotics were administered and the area of exposure was allowed to heal. If the latter steps were successful, tissue expansion was restarted. The anatomical and physiological differences in the extremities could explain the higher complication rates. First, the extremities are in frequent motion and the underlying musculature can exert disruptive forces to the expander. Second, the flaps designed on the extremities are more difficult to encompass a curvilinear biconvex surface necessary for successful tissue expansion.

Fischer *et al.* examined significant preoperative risk factors associated with failed tissue expander placement in breast reconstruction.⁵³ In their analysis they utilized the American College of Surgeons National Surgical quality improvement program data sets from 2005 to 2010 to determine perioperative risk factors associated with early expander loss. A multivariate regression analysis demonstrated several preoperative patient conditions that are associated with early device loss: age >55 years, obesity and active smoking. Deep wound infection was the strongest predictor of loss (odds ratio (OR) 9.1, $p < 0.001$).⁴⁸ Patients with burns injuries have also been found to have greater complication rates during tissue expansion.⁵⁴

Future of tissue expansion

Expansion of skin and subcutaneous tissues adjacent to the pathological skin to be replaced is probably the only method of reconstruction that creates almost normal structure, anatomy and function in reconstructive surgery. This, however, may lead to the technique being restricted to patients with normal tissues surrounding the pathological defect. Furthermore, complications, especially in certain anatomical areas, are frequent, leading to further restrictions in terms of patient eligibility. In the future, as the underlying cell biology and molecular basis of tissue expansion is elucidated, former restrictions and complications may be overcome. We are now witnessing a revolution in the field of regenerative medicine and tissue-engineered expanders will surely follow. For example, the expander's material could be used as a carrier to deliver signals that 'manipulate' cell proliferation, vascularity and function. This may shorten the expansion time and increase the available expanded tissues.

In summary, tissue expansion is an important reconstructive tool in the armamentarium of plastic surgeons. It can overcome tissue shortages in challenging scenarios, providing an excellent colour, quality, sensitivity and tissue match for the defect site. However, as discussed in this chapter it is not without its complications and patients undergoing this procedure should be appropriately consented about the timeframe and what to encounter during the process.

Ten golden rules of expanders

- 1 Select a donor site where the consistency of the soft tissues are identical to those required to close the defect.
- 2 The access incision for the tissue expander should be placed at the margin of the defect and ideally through unscarred and non-radiated tissues.
- 3 Rectangular expanders give the most tissue expansion. Rectangular expanders increase surface area by 38% in contrast to round, which only give 25%.
- 4 The amount of fluid injected into the expander should be individualized to the patient and governed by tenseness, blanching of the overlying skin and pain. Care in preventing overexpansion will reduce the chances of tissue ischaemia and necrosis of the skin flaps.
- 5 The tissue expander should be sited on a base that resists mechanical deformation and directs the force of expansion outwards and towards the overlying soft tissues (e.g. bone).
- 6 The pocket which is dissected for tissue expander placement must be larger than the dimensions of the expander.
- 7 Delay implant inflation for approximately two weeks until sufficient time has passed for the wound to heal and oedema to settle. This will reduce the chances of incisional dehiscence and will allow time for problems with the flap to reveal itself.
- 8 One must recall that the capsule around the expander is highly vascular and capsulectomy can compromise the vascularity of the overlying skin.
- 9 Complication rates vary in tissue expansion depending on anatomical location. Extremity lower limb expansion has been shown to have higher rates of major complications in comparison to the scalp and the breast.
- 10 An exposed expander can be salvaged and expansion resumed in some cases.

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CHAPTER 7

Tissue engineering

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Nanotechnology meets tissue engineering

More than 30 years ago, scientists at the Massachusetts Institute of Technology (MIT) first began researching into 'living skin equivalents' and vascularized scaffolds, leading the way into the era of tissue engineering (TE).¹ The main driving force behind the progression of TE to date is the prevailing organ donor shortage but it was not until 1987 that the term 'tissue engineering' was defined at a National Science Foundation meeting as 'the application of the principles and methods of engineering and life sciences towards the fundamental understanding of structure–function relationships in normal and pathological mammalian tissues and the development of biologic substitutes that restore, maintain, or improve tissue function'.² The pivotal strategies underlying TE have been identified as the use of (a) isolated cells or cell substitutes, (b) tissue-inducing substances and (c) cells placed on or within matrices.¹ This rapidly emerging field combines the principles of bioengineering, cell transplantation and biomaterials engineering to create platforms that resemble as much as possible the body's own native tissues in order to stimulate tissue and organ regeneration for transplantation. The amalgamation of biological and artificial components in such a way as to achieve active repair, structural maintenance and targeted reconstruction is the ultimate goal of TE. Basic building blocks including cellular components such as stem cells and scaffolds which may act as inert structural supporting frameworks and/or delivery vehicles for biologically active molecules may be superimposed in various different ways to create suitably specific scaffolds (Figure 7.1).

Several experimental studies have used TE approaches to successfully regenerate skin,^{3,4} bone,^{5,6} cartilage,^{7,8} musculature^{9,10} and fatty tissues^{11,12} and applied to surgical problems, TE has a clear potential to make a substantial difference by facilitating surgical procedures, accelerating postoperative wound healing and developing new surgical strategies. Current TE techniques mostly rely on the use of porous scaffolds which act as supporting structures for initial cell attachment and subsequent tissue ingrowth.^{13–15} Although relatively successful at providing basic platforms for cell

adherence, their usually macroscopic internal architecture lacks submicroscopic environmental cues which are normally found within natural extracellular matrix (ECM) (Figure 7.2), thus limiting the cells' inert capabilities to stimulate regeneration of relatively large or complex tissues.¹⁶ In order to generate constructs capable of inducing and enhancing natural cellular behaviours, researchers sought to develop structures that no longer served only as cellular adhesion platforms, but could also operate as independent agents capable of interacting with their environment to actively stimulate and guide tissue regeneration.¹⁷

The emergence of nanotechnology and its integration into the medical arena carries the field above and beyond former limitations, thereby igniting a wave of interest for the exploration of research into biotechnological systems at the nanometre scale. With regards to TE, the emergence of nanotechnology ('nano-engineering') has revolutionized the approach to fabricating tissues and organs, since the manufacture of novel biosynthetic scaffolds with enhanced material properties enables scientists to develop controllable and application-specific biomaterials. Novel biomaterials of nanometre dimensions are particularly attractive as they can closely mimic the molecular structure of tissues and organs, thus providing an ideal environment for cellular interactions and tissue regeneration. Scaffolds with nanostructured surfaces capable of incorporating bioactive agents have thus been developed to enable finer control over cellular positioning, organization and interactions.¹⁸

This chapter focuses on the main determinants for successful engineering of tissues and whole organs. Particular attention will be paid to the importance of stem cells as well as physical parameters of scaffolds, including material dimensional surface characteristics (micro- versus nanoscale features) and mechanical properties that, when orchestrated correctly, can act in synergy to recuperate lost function and aesthetics. Further, the ability to exploit the fundamental principles of nanotechnology will be discussed within the realms of TE to create hybrid, biologically active organ replacements based on the synergistic interplay between composite and nanocomposite materials and cellular or bioactive components. A new generation of silica nanocomposite

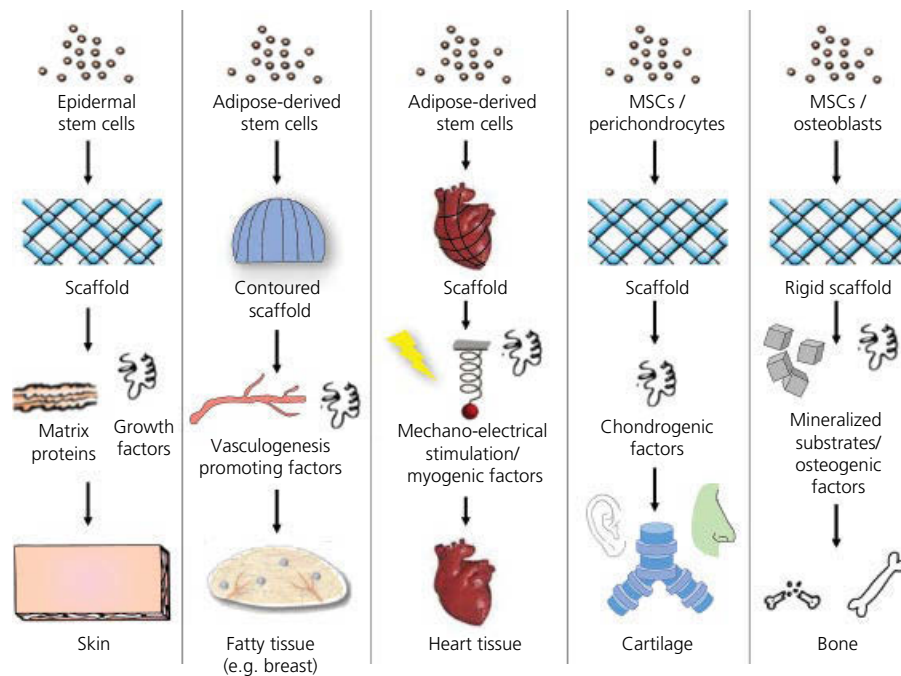


Figure 7.1 Tissue engineering strategies for plastic and reconstructive surgery. The basic principles of tissue engineering are applied to obtain fully functioning organ and tissue replacements.

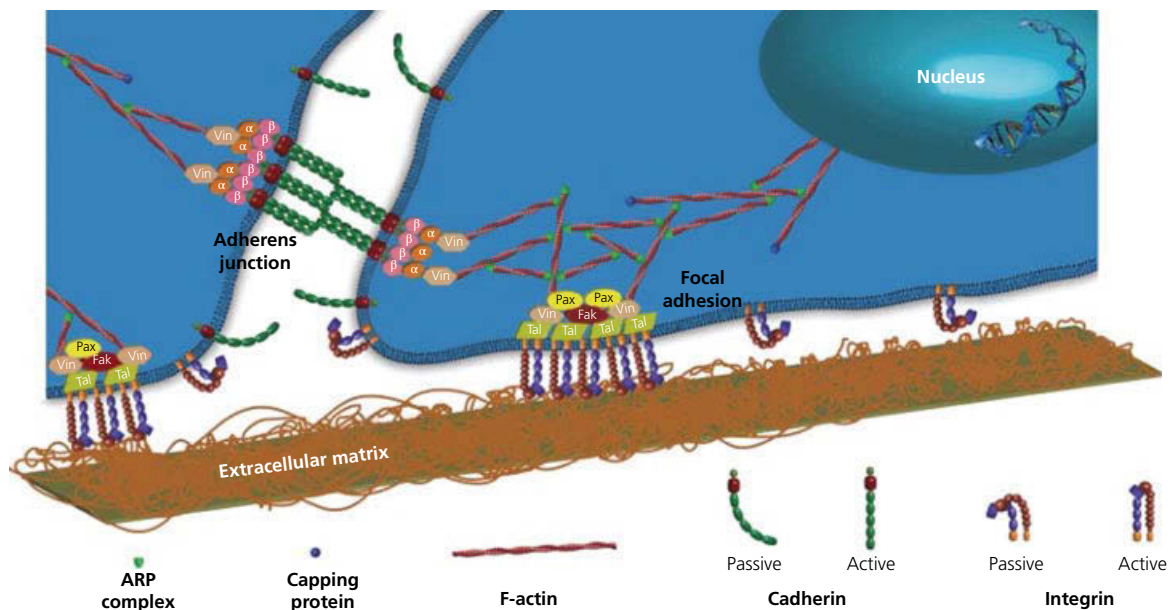


Figure 7.2 A schematic illustration of cell-cell and cell-matrix interactions. Source: Girard *et al.*, 2007.⁸ Reproduced with permission of the Royal Society of Chemistry.

scaffolds based on polyhedral oligomeric silsesquioxane (POSS) nanoparticle-integrated poly(carbonate urea)urethane (POSS-PCU) will be critically debated in the context of their materials properties and their immense clinical translational potential, exemplified via the world's first in-human transplantation of a fully synthetic tracheal replacement and several more applications.

Pluripotent stem cells in tissue engineering

With the recent discovery of adult somatic stem cell reprogramming, the enticing prospect of theoretically limitless self-renewal, pluripotency, as well as differentiating capacity of induced pluripotent stem (iPS) cells catapults the field of

TE above and beyond theoretical model systems, as any cell type may now theoretically be induced in virtually limitless amounts. The notion of creating genetically designed and patient-specific pluripotent stem cells for research or clinical application has already progressed to tentative reality in the cases of human iPS cells derived from amyotrophic lateral sclerosis (ALS) and inherited spinal muscular atrophy patients which were successfully differentiated into motor neurons, the cell type destroyed in these two conditions.^{19,20} Further diseases with realistic potential to benefit from iPS cell technology include Parkinson's disease, Huntington's disease and Down syndrome among several more.²¹ In particular, the area of regenerative medicine could be revolutionized with the use of iPS cells, as scientists, doctors and patients would no longer be reliant on donated organs but could populate three-dimensional scaffolding structures with the patient's iPS cells and differentiate those into the required cell lineage.

Although theoretically brilliant, several substantial hurdles must be overcome prior to successful implementation of pluripotent stem cell-derived tissues in the clinic:²²

- The genetic stability of pluripotent stem cell lines must be thoroughly assessed and maintained throughout long-term culture and differentiation conditions to eliminate the potential for transformation and teratoma formation.
- The differentiation potential of iPS cells must be fully elucidated and a robust method for consistent differentiation into the relevant and functional tissue types must be determined.
- The molecular and functional characteristics of cells derived from pluripotent stem cells must be elucidated.
- The immunological determinants and engraftment potential of allogeneic pluripotent stem cell-derived cells are yet to be ascertained.
- Ethical issues regarding pluripotent stem cells, particularly those derived from human embryonic stem (hES) cells must be resolved.
- Finally, the progenitor or differentiated cells must be able to self-assemble or 'sense' directional cues within a 3D tissue engineered construct with appropriate cell–cell and cell–matrix interactions.

Despite such barriers, recent efforts to combine promising pluripotent stem cell technologies and TE concepts have gained valuable insight into cardiovascular,^{23–26} bone,^{27,28} cartilage,²⁹ nerve³⁰ and liver³¹ regeneration.

To begin with, the different sources of human stem cells within the body will be presented before highlighting the important role of local environmental cues in directing these stem cells down the predetermined lineage. The prior notion that cells isolated from specific tissues may only renew and differentiate into lineages of its host tissue has now been challenged, suggestive of considerable plasticity and ability to cross-over lineage restriction boundaries to give rise to cells of other lineages.³²

Human embryonic stem cells – an ethical dilemma

In 1998, James Thomson first harvested hES cells from the inner cell mass of blastocyst-stage embryos (from day 5 post fertilization) and subsequently cultured these on a mouse embryonic fibroblast feeder layer.³³ It was found that highest yield derivation was achieved by harvest and culture of the entire inner cell mass of the blastocyst. Subsequent studies have demonstrated the feasibility of harvesting embryonic cells prior to the blastocyst stage, during the morula stage (day 3–4 post fertilization). Rates of successful derivations, however, remained lower compared to those of blastocyst-stage derived cells.^{34,35} Further predicting factors for poor hES cell yield include embryo storage conditions and quality prior to derivation attempts. Prematurely arrested or highly fragmented embryos rarely derived cell lines, whereas those which have achieved blastocyst stage represent a solid source of hES cells.³⁶

Due to ethical concerns regarding the use of hES cells, efforts at stem cell derivation without embryonic destruction have been made and several different cell lines have been obtained using single-cell biopsies from morula-stage embryos.³⁷ Despite ongoing efforts, currently available protocols remain labour-intensive and are thus unlikely to become widespread.

Blastocysts may also be generated from unfertilized oocytes via asexual fertilization – a process termed parthenogenesis, which relies on chemical or electrical stimuli to induce development. Parthenogenesis may prove particularly useful in regenerative therapies as HLA homozygous hES cell lines could be derived from HLA heterozygous donors, possibly rendering this technique suitable for generating and banking HLA-matched hES cell lines.^{38,39} It remains to be seen whether hES cell lines will serve as renewable cell sources in the future since prohibitively large ethical concerns bar in-depth research using embryo-derived components.

Human induced pluripotent stem cell sources

With the seminal discovery 'that mature cells can be reprogrammed to become pluripotent' a new era of stem cell research with theoretically unlimited potential has begun. The ability to extract adult somatic cells and induce these to express genes important for maintaining properties of hES cells has already proved a useful tool for drug development and *in vitro* disease modelling. In 2007, Thomson and Yamanaka independently demonstrated the feasibility of reprogramming embryonic and adult human fibroblasts using only four transcription factors each. Takahashi *et al.* retrovirally transduced the four genes *Oct4*, *Sox2*, *KLF4* and *c-Myc* to revert differentiated adult human dermal fibroblasts into a pluripotent state⁴⁰ based on previous studies of factors known to reprogram murine somatic cells to induced pluripotent stem (iPS) cells.^{41–44} Yu *et al.* used lentiviral transduction of *Oct4*, *Sox2*, *Nanog* and *Lin28* to reprogram human foreskin fibroblasts and achieve pluripotency.⁴⁵ It was demonstrated that these iPS cells expressed cell markers characteristic of hES cells, including Ssea-3, Ssea-4, Tra-1–60, Tra-1–81, alkaline phosphatase, Nanog, Oct3/4 and Sox2.

Additional similarities to hES cells such as high telomerase activity,⁴⁶ histone methylation patterns,⁴⁷ embryoid body formation,⁴⁸ directed differentiation to cells in each of the three germ layers⁴⁹ and teratoma formation in mice⁵⁰ were observed in iPS cells. Slight variations in overall gene expression patterns were deemed minor and in line with regular line-to-line variations observed between hES cell lines.⁵¹

Despite such fundamental progress in stem cell science, prohibitive barriers, including the use of the oncogene *c-Myc* and an integrating retrovirus for gene transfer, had to be eliminated prior to adoption of iPS cells into the field of TE and regenerative medicine. Retroviral integration of transcription factors pose concerns due to possible transgene reactivation events and insertional mutagenesis of genes at integration sites which can alter the cell's subsequent biological behaviour and result in tumorigenicity, as observed in some patients who underwent gene therapy for X-linked severe combined immunodeficiency (X-SCID).⁵² It is therefore deemed crucial to generate iPS cells using non-integration methods.⁵³ In 2008, Okita *et al.* successfully induced pluripotent stem cells using non-integrating plasmid transfection but observed substantially lower iPS cell generation efficiency compared to transduction with retroviruses.⁵⁴ It remains to be determined whether a lower transgene expression with plasmid transfection is the underlying reason for relatively lower efficiency in iPS cell generation. Simultaneously, Zhou *et al.* reprogrammed murine embryonic fibroblasts to a pluripotent state using direct protein transduction.⁵⁵ Valproic acid and recombinant Oct4, Sox2, KLF4 and *c-Myc* proteins were fused to polyarginine protein transduction domains to obtain reprogrammed cells. Although this method has not yet been demonstrated in human cells, a similar approach is likely to be effective.

Stem cell plasticity

Poor iPS cell generation may not only depend on degrees of transgene expression but seems to be fundamentally affected by the somatic cell source. It has been shown that reprogramming efficiency was higher with human keratinocytes compared to human fibroblasts, with fewer viral integrations in keratinocyte-derived iPS cells, supportive of a higher degree of reprogramming efficiency and less genomic damage.⁵⁶ Besides the somatic cell source, other factors affecting differentiation efficiency of iPS cells include donor age and the specific method used for reprogramming. As evidenced by numerous studies, human iPS cells may be obtained from a multitude of somatic cell sources. Studies using transgenic mice containing the four reprogramming transcription factors have demonstrated iPS cell harvest from different cell types, including neuronal progenitor cells, keratinocytes, hepatocytes, B cells and fibroblasts of murine tail tips, kidneys, muscles and adrenal glands.⁵⁷ Such diverse origins enable the engineering of a range of tissues or organs such as bone, cartilage, muscle, marrow stroma and tendon.^{58–60} However, once cells are isolated from their natural habitat, they also lose their natural cues, potentially resulting in changes in

their phenotype and differentiating abilities. Although some studies attempted direct transplantations of cell suspensions without a scaffold, it soon became apparent that a lack of control over the localization of transplanted cells required the use of a protective delivery vehicle.^{61,62} Several studies thereafter and more recently have demonstrated enhanced capacity of cell delivery, viability and engraftment using both synthetic and naturally derived biomaterial scaffolds.^{63,64} However, once seeded, stem cells continue to depend on external signals to differentiate down a desired pathway.

Numerous studies have investigated a plethora of growth factors which when delivered alongside stem cells 'persuade' those to switch on specific genes to enable controlled differentiation into mature tissue-specific cells.^{65–67} Despite being routinely used in basic and clinical research, the combination of growth factors and stem cells for TE purposes elicits a feeling of unease due to the theoretical potential of tumorigenesis;⁶⁸ the feature most sought after in stem cells – continuous self-renewal – simultaneously may give rise to tumours due to transformations of normal stem cells. Equally, similar signalling pathways may regulate self-renewal in stem cells and cancer cells. The boosting of said pathways by a cocktail of growth factors remains a prohibitive feature for the transition of many promising TE strategies into clinical reality. Therefore, in an effort to harness the undoubted potential of stem cells, science re-evaluated their focus and concentrated on the influence of physical factors such as scaffold surface topographies and mechanical properties on the differentiation potential of stem cells. Zhu *et al.* demonstrated controlled differentiation of mesenchymal stem cells into osteoblasts when cultured on grooved biological nanofibres (width of 6.6 nm) equipped with osteogenic peptides.⁶⁹ Similarly, Kumar *et al.* showed that bone marrow stem cells grown on nano-rough surfaces obtained by solvent etching induced osteogenic differentiation and proliferation, whereas unetched scaffolds did not.⁷⁰ Further, cellular morphology was significantly different when grown on rough compared to smooth surfaces, indicating a significant role in surface roughness not only in directing cellular differentiation but also in dictating morphology.

The next section will delve further into the physical properties of TE scaffolds and their influence on cell fate.

Physical characteristics of tissue engineering scaffolds direct cell fate

Stem cells, although equipped with extraordinary intrinsic potential, require an inductive external environment in order to fulfil their desired functions. Unlike most other cell types, stem cells may differentiate into several different lineages. However, like most other cell types they are governed, to a large extent, by a supportive ECM capable of providing just the right cues at just the right times. It is thus up to the scientist to attempt to recreate such a space by means of designing a suitable scaffold. This section will focus on the development of TE scaffolds and

their importance in terms of directing (stem) cell fate and differentiation. The myriad of different fabrication techniques will be critically appraised in the context of clinical translatability. A particular focus will be placed on the differences observed between micro- and nanotechnological approaches in scaffold synthesis and related to cellular compatibility and host tissue responses.

The aim to create biomimetic scaffolds (i.e. a dynamic physical surrounding which replicates the cell's natural environment as closely as possible) makes use of a diverse range of materials including naturally derived or synthetic materials. More and more, a shift to taking a combinatorial approach harnessing the advantages of biological and synthetic biomaterials while cancelling out their individual drawbacks is favoured. The following paragraphs will discuss the importance of the physical environment in terms of TE scaffold design, pros and cons of commonly used biomaterials and the feasibility to create an exact replica of the human ECM.

Micro- versus nanotechnologies in scaffold fabrication

Despite a relatively advanced knowledge of the necessary physical environment required for cell growth, engineering a structurally ideal scaffold remains challenging. Current TE strategies as well as clinically used regeneration templates mainly focus on macrostructured porous scaffolds which primarily aid in initial cell attachment as demonstrated both *in vitro* and *in vivo*.^{13,15,71,72} Although relatively successful at reconstructing or replacing simple structures, developments of more complex tissue and organs in terms of both physical size and functionality are currently limited.⁷³ This is mainly due to insufficient or delayed vascularization of scaffold constructs and subsequently inhibited oxygen and nutrient delivery and removal of waste products. A recent study by Bai *et al.* demonstrated the crucial importance of scaffold pore size and pore interconnectivity for the development of blood vessels.⁷⁴ Interestingly, their data suggested that a pore size of up to 400 µm and numerous pore interconnections resulted in the most vascularized scaffolds. The strong link between scaffold macroporosity and enhanced vascularization potential was further demonstrated by several other studies.^{75–78} However, other studies found that in the *in vivo* setting, pore size was of lesser relevance compared to, for example, the degrees of chemical crosslinks present in TE scaffolds.⁷⁹

Macroporous structures may be advantageous for blood vessel formation but have been demonstrated to be detrimental for the proliferative capacity of certain cell types. It is well known that different cells respond to different pore sizes such that bone cells, for example, prefer larger pore sizes whereas fibroblasts preferentially grow and proliferate on scaffolds with significantly smaller pore sizes.^{80–82} Such discrepancies are thought to be due to the individual ECM characteristics of specific tissue types and must be taken into account when designing TE scaffolds. A further challenge in the design of TE scaffolds is the optimization of

interactions occurring at the cell–material interface. As complex tissues and organs demonstrate highly defined and organized structures, the growing field of nanobiotechnology has enabled scientists to refine scaffold architectures to obtain a much finer control over cellular positioning, organization and cell–cell as well as cell–matrix interactions.¹⁸ Tailoring of precise features on the material's surfaces enables the development of nanotopographies which render such materials unique and capable of controlled interactions with their environment. In fact, today's goal of TE is to create not merely simple substrates for cellular adhesion, but 'smart' platforms that are actively involved in tissue development and regeneration.¹⁷ Figure 7.3 illustrates the range of different sizes and proportions found within the human body to highlight the importance of nano-sizes in terms of cell–matrix recognition.

Bottom-up versus top-down scaffold fabrication methods

The motivation to create TE scaffolds with nanotopographical features stems from the knowledge that natural tissues and ECM components are composed of nanostructured cues.^{83–85} Cells then respond to such cues, which influence the material's surface properties, including surface chemistry, charge, wettability, topography and surface energy.^{86–88} Nowadays, nanotechnological advances in materials science and biotechnology are more and more able to create spatially controlled surface-specific adjustments down to a micro- and nanoscale, which encourages anchorage-dependent cells to recognize 'foreign' surfaces as natural, resulting in biological interactions, cell growth and differentiation.⁸⁹ This ability of cells to direct their own position and orientation on the basis of surface topographies is called mechanosensing.⁹⁰ Converting these physical forces into intracellular biochemical responses is referred to as mechanotransduction.⁹¹ These interactions between biomaterials and cells are primarily influenced at the interface between these two entities and attempts at recreating such surfaces involves the use of micro- and nanotechnological fabrication methods.

Such fabrication methods can be subdivided into two areas according to two different concepts: bottom-up and top-down approaches (Figure 7.4). The former mainly involves material self-assemblies, whereas top-down TE approaches include methods such as photolithography, microcontact printing, microtransfer moulding, capillary force lithography, scanning probe lithography and electrospinning.⁹² Both approaches, although coming from opposing ends of the materials science spectrum, require the same intricate knowledge of materials surface properties and the potential to trigger a variety of molecular events and processes. In both cases, scientists must understand the principles of device miniaturization, kinetic processes and cell biology to excel. The aim of bottom-up scaffold fabrication is to create modular units which assemble into larger tissue constructs with predetermined micro- and nanoarchitectural features.⁹³ This may be achieved through self-assembled

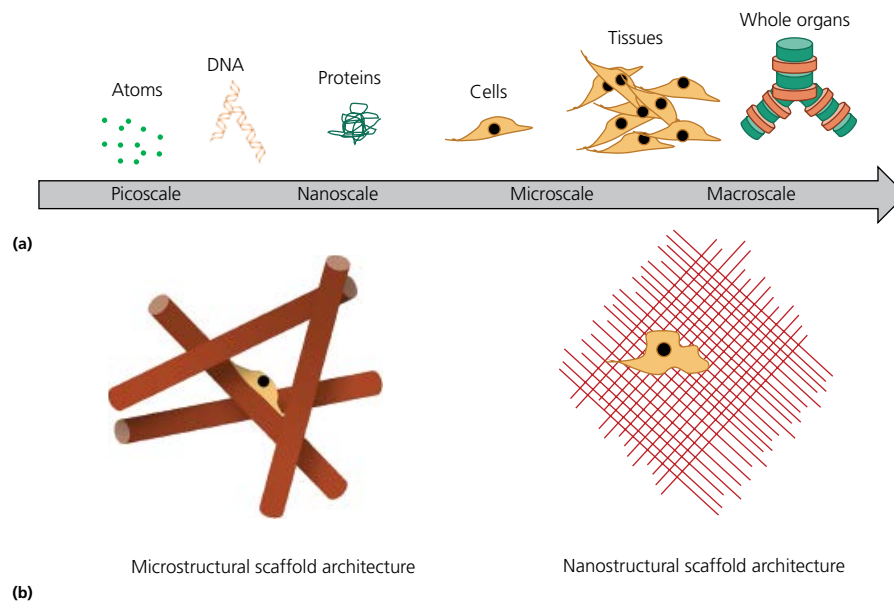


Figure 7.3 (a) A schematic representation of biological entities on a scale ranging from pico-sized atoms to macroscopic organs. Not to scale. Inspired by (9). (b) Cells behave differently on microstructures compared to nanostructures.

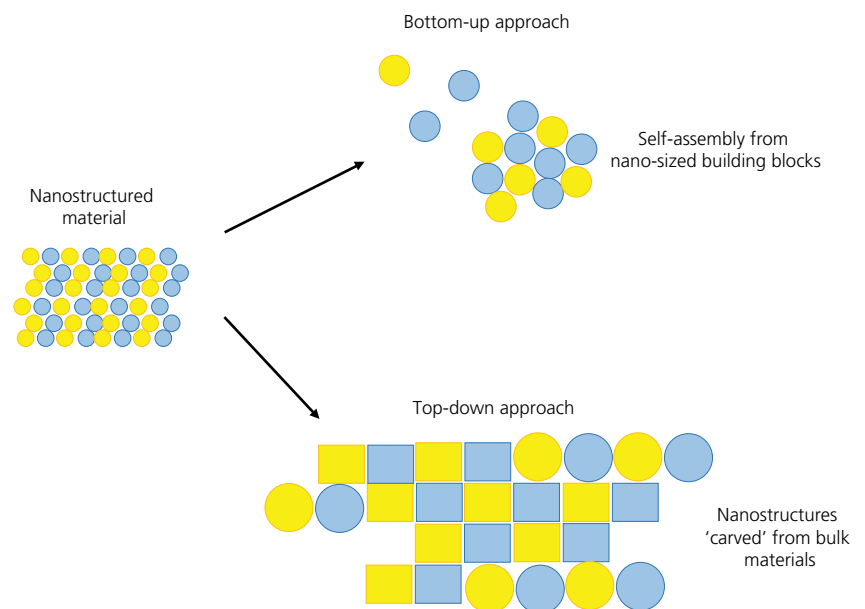


Figure 7.4 A schematic representation of the bottom-up and top-down concept.

aggregation, microfabrication of cell-laden hydrogels, cell sheet synthesis and direct printing of cells.

The underlying theory is based on the concept of tissue fluidity and fusion of cell aggregates as observed in embryonic development during which tissues may be considered as liquids.⁹⁴ Bottom-up approaches are generally based on the use of bio-ink containing cells, a bio-printer or ink dispenser and a temporary support for the deposited ink.^{95,96} Through controlled deposition of cell-containing ink, bottom-up fabrication methods can carefully engineer complex structures such as vascular trees and multilayered structures containing multiple, but spatially distinct cell

types. This has proved useful as recently documented by Koch *et al.*, who bioengineered a 3D skin construct composed of multicellular collagen layers deposited using a laser bio-printing system.⁹⁷ Cell viability and proliferative capacity was found to be adequate over a period of 10 days, with different cell types remaining distinct throughout the study period.

In addition to cell printing, bottom-up fabrication methods allow for the incorporation of cell-signalling factors within slowly degrading bio-ink and a more precise recreation of the natural extracellular environment. Bottom-up self-assembly approaches are not rarely inspired by nature itself; nanostructures may be

obtained by biologically inspired self-assembly as demonstrated by amphiphilic peptide self-assembly into hydrogels for TE strategies.⁹⁸ Bottom-up approaches rely on the incorporated cells' own production of ECM to create a supportive and non-immunogenic structure. However, insufficient ECM production in some cell types is thought to limit cellular migration and formation of cell–cell as well as cell–matrix junctions.⁹⁹ Furthermore, the harsh, relatively non-permissive conditions created by injuries and the lack of nutrients is thought to result in poor cell engraftment.^{100–103}

Top-down fabrication methods involving the use of biodegradable scaffolds as a longer term substrate for cellular adhesion, proliferation and new tissue formation may thus be required. Cells and cell-signalling factors may further be incorporated into such scaffolds and subsequently released in a temporo-spatially controlled fashion, either within a dynamic bioreactor system or *in situ* to accelerate functional regeneration.^{96,104,105} Scaffolds may consist of natural or synthetic biomaterials, or of a combination of both material classes to create a composite scaffold. The ideal scaffold for TE purposes should actively guide tissue formation, recuperate lost function and deliver cells and/or cell-signalling factors by mimicking, as close as possible, the natural bioenvironment. Despite ongoing research into scaffold fabrication methods, the ideal system remains elusive which, to a large part, may be due to the ambiguity of beneficial and detrimental scaffold characteristics, thus requiring more focused research.

The future of material fabrication for TE purposes has been suggested to lie in a combinatorial approach to harness the structural support provided by top-down fabricated scaffolds and versatility implemented by nanodimensional features within bottom-up assemblies. Such complementary, 'intelligent' scaffolds would then be able to actively regulate the development of ingrowing cells by providing external cues with or without supplementation with trophic signalling factors.

The next section will critically appraise the current status in TE of artificial organs and tissues in the context of a novel nanocomposite material, POSS-PCU, which draws its benefits from nano-sized surface structures as well as mechanical properties ideally suited for TE scaffolds.

Tissue engineering of artificial organs

The field of TE has come a long way, but demands for replacement organs and tissues still far outnumber currently available treatment strategies, rendering millions of individuals dependent on altruistic organ donations. In such circumstances, glimpses of hope involving clinical applications of engineered artificial organs go a long way in boosting scientists', clinicians' and patients' spirits. In this section, we will present and discuss the first-in-human applications of several different POSS-PCU-based TE organs and obtain a further insight into other promising tissues which are thought to reach the clinical setting fairly soon. We will bring together the concepts of stem cells and

organ scaffolds to demonstrate the ultimate goal, which is to create whole functional organs. At the end, the current situation in TE and regenerative medicine will be critically appraised within the realms of feasibility, current limitations and suggestions of how we may overcome these.

First-in-human applications of tissue engineering organs

In our laboratories, we have developed and patented a family of novel polymeric nanocomposites, POSS-PCU, for the development of synthetic organ scaffolds and the regeneration of viable tissues. The fusion of nanotechnology with regenerative medicine for the development of TE organ replacements guides us one step closer into a future independent from organ donation schemes – a feat recognized to solve a multitude of fundamental clinical as well as ethical issues. The capacity for successful replacement and regeneration of critically non-viable organs and tissues using our POSS-PCU nanocomposite scaffolds has been demonstrated in several recent high-profile first-in-human cases and exemplified by the transplantation of the world's first completely synthetic TE trachea which clearly demonstrated proof-of-concept for this new nanocomposite material.¹⁰⁶

TE trachea using the novel nanocomposite POSS-PCU seeded with autologous stem cells

Tracheal cancer, although one of the rarer neoplasms, is characterized by a high mortality rate and is conventionally managed with surgical resection and primary reconstruction.¹⁰⁷ However, the insidious onset and subsequently delayed diagnosis results in most primary malignant tracheal cancers presenting as inoperable diseases with 5-year survival rates of 5%.¹⁰⁸ A further obstacle to recovery includes the sheer lack of reconstructive options, rendering complete tumour resection unsafe in more than 40% of cases.¹⁰⁹ Attempts at transplanting TE tracheal substitutes into human patients using a decellularized human donor trachea as a base scaffold reseeded with bone marrow-derived mesenchymal stem cells and respiratory cells have provided first insights into the anatomical, physiological and biomechanical properties required for optimal TE tracheal replacement.¹¹⁰ It was found that apart from the prevailing donor organ shortage, mechanical instability and unpredictable functionality and immunogenicity rendered decellularized tracheal scaffolds ultimately unsuitable. Consequently, an alternative, tailor-made synthetic tracheal scaffold was fabricated using the nanocomposite material POSS-PCU (Figure 7.5) and transplanted into the tracheobronchial airway of a patient with recurrent primary tracheal cancer.¹⁰⁶ *Ex vivo* seeding with autologous mononuclear cells¹¹¹ and growth factor supplementation resulted in early reepithelialization of the transplanted TE construct.

The results of this first-in-human bioengineered artificial tracheal transplantation provides proof-of-concept for TE organ and tissue systems. Further research will have to focus on evaluating cell differentiation and ultrastructural organization on the transplanted construct.



(a)



(b)



(c)

Figure 7.5 (a) The world's first completely synthetic trachea transplanted into a human patient in 2011. Prior seeding with autologous bone marrow-derived stem cells enabled rapid integration into the host system and epithelialization with bronchial cells. (b, c) Realistic replicas of a nose and ear of two different patients made from POSS-PCU.

Lacrimal duct engineering using POSS-PCU nanocomposite

Lacrimal drainage obstructions causing excessive tearing, blurry vision and infections are common problems, which are usually tackled either by perforation of the obstruction, dilatation of the narrowed portion or bypassing a total obstruction. Although lower lacrimal duct obstruction can be treated with almost perfect success rates, canalicular (upper or presaccal) obstructions can only be insufficiently addressed with currently available therapies. Due to the extremely small diameter of the canaliculus (<1 mm), restenosis and stent failure are frequently observed. Bypass tubes, although temporarily effective, frequently encounter mucous blockage necessitating manual cleaning or replacement. Tube dislocation represents an additional complication.¹¹²

The ideal lacrimal duct conduit should inhibit scar formation to prevent later obstruction or dislocation. It should further be of anatomically suitable size (internal diameter ~1 mm), yet kink resistant but elastic and compressible to allow surrounding muscles to pump tears forward into the nasal cavity.¹¹³ The novel POSS-PCU nanocomposite material with its highly viscoelastic, biocompatible and non-immunogenic properties further exhibits a relatively 'non-stick' surface, ideally suited as a lacrimal duct drainage material (Figure 7.6).¹¹⁴ Several patients have so far benefited from this new artificial canaliculus.

Artificial vascular bypass graft using POSS-PCU nanocomposite

Cardiovascular diseases are responsible for over 20% of global mortality and represent the primary cause of death worldwide.¹¹⁵ Once pharmacological interventions prove unsuccessful, transplantation of vascular grafts remain the only realistic option. Autologous blood vessels are the ideal candidate in terms of long-term outcome. However, only about a third of patients have viable, autologous tissues available for harvest.¹¹⁶ Currently used prosthetic grafts such as expanded polytetrafluoroethylene (ePTFE) or polyethylene terephthalate (Dacron)^{116,117} are relatively successful as larger diameter grafts but tend to collapse or occlude once their internal diameter drops below 6 mm due to their inherent thrombogenic material surface¹¹⁸ and their relatively non-compliant mechanical properties. Besides, the use of prosthetic grafts in the paediatric population gives rise to a whole plethora of further complications related to the child's somatic growth, which necessitates frequent reoperations¹¹⁹ resulting in considerable morbidity.¹²⁰ These disadvantages led to the development of TE vascular grafts that attempt to facilitate the regeneration of lost or damaged blood vessels while remaining patent and antithrombogenic even at relatively smaller diameters.

In 2001, Shin'oka initiated the search for the optimal TE vascular graft by fabricating a biodegradable TE vascular graft based on polycaprolactone/polylactic acid copolymer reinforced with woven polyglycolic acid and seeded with autologous cells obtained from one of the patient's peripheral veins.¹²¹ Subsequent attempts to clinically translate TE vascular grafts based on either synthetic, decellularized or autologous scaffolds achieved relative success.^{122–128} However, the engineered conduits used in all of the above instances had internal diameters of more than 6 mm. It appears that below this value, graft patency becomes critical, necessitating a different approach to TE small diameter vascular grafts. The nanocomposite material POSS-PCU has demonstrated excellent patency in even smaller diameter grafts (see section above on lacrimal duct conduit) and has recently been transplanted as an ilio-femoral bypass graft in a first-in-human procedure (Figure 7.7).

The ability of POSS-PCU grafts to resist thrombo-occlusion, calcification and degradation has previously been extensively studied and confirmed.^{129–131} Based on the hypotheses that

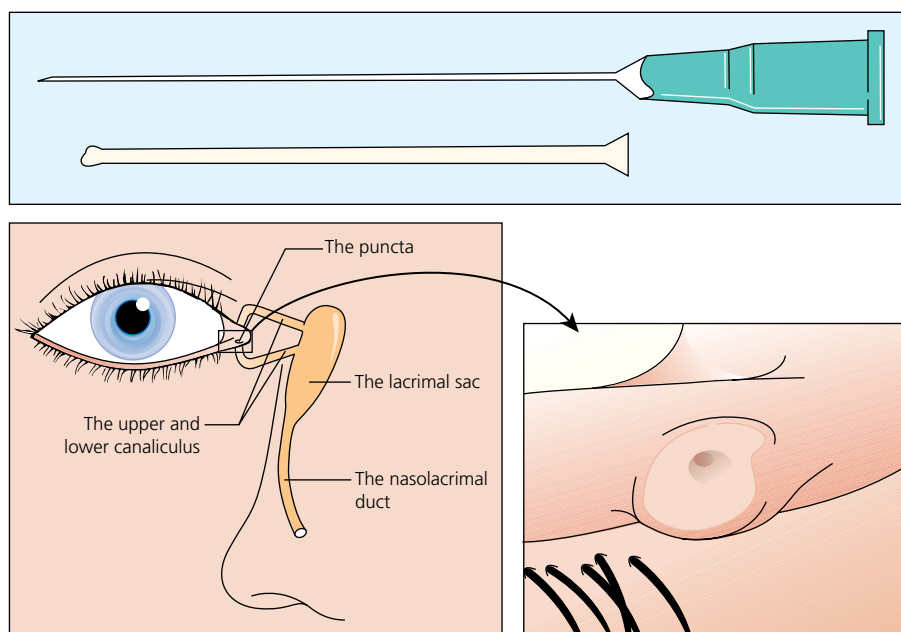


Figure 7.6 An artificial lacrimal duct conduit made of POSS-PCU. The internal diameter is <1 mm but kink-resistant and non-occlusive. In clinical use.

material hydrophobicity favours platelet and protein repulsion and yet amphiphilicity allows for endothelial cell attachment, it was postulated that the integration of the hydrophobic POSS nano-cage into a more hydrophilic PCU backbone will create a material with both anticoagulant as well as cyto-attractant properties. In addition to antithrombogenicity, several other factors have been found to contribute to long-term vascular graft patency; a viscoelastic profile similar to native arteries as well as the potential for luminal endothelialization is known to create favourable conditions preventing intimal hyperplasia and luminal thrombus formation, respectively, two factors intrinsically associated with early graft failure.

The graph in Figure 7.8 compares the viscoelastic profiles of native arteries with (a) conventionally used replacement conduits made of Dacron or PTFE, (b) veins and (c) vascular grafts made of POSS-PCU. POSS-PCU grafts exhibited an exceptionally suitable compliance profile very similar to that of native arteries, particularly in comparison to Dacron or PTFE grafts which did not demonstrate any significant degree of compliance. The inherent viscoelasticity of POSS-PCU vascular grafts stems from the alternating soft and hard segments of the PCU backbone with variations in respective percentages determining the degree of elasticity and hardness. This certainly affects long-term graft patency *in vivo*, where compliance mismatch between native artery and Dacron/PTFE grafts results in thrombus formation secondary to turbulent blood flow at the junction between native artery and graft as well as the development of intimal hyperplasia, eventually culminating in luminal narrowing and graft failure.

In several *ex vivo* and *in vivo* large animal studies, we have demonstrated the endothelialization potential of POSS-PCU vascular grafts and heart valves, a technique further enhanced

by modifications of the surface luminal aspect via peptide functionalities.^{132–134} Modifications of vascular graft interfacial surfaces are aimed at regulating (a) cell-surface interactions, (b) protein and platelet adsorption and (c) thrombin formation (and eventual blood coagulation) in order to optimize patency rates. In general, surface morphology and nanotopography directly influence a material's chemical characteristics and are, in turn, directly dependent on both intrinsic properties such as the material's molecular structure, composition and molecular weight¹³⁵ as well as extrinsic surface modifications including physical and chemical alterations and biofunctionalization.

Other tissues and organs based on POSS-PCU with substantial clinical potential

It is encouraging to see the successful transition of several TE POSS-PCU-based organs from the laboratory bench to the clinical bedside. Several more are currently under investigation, including heart valves, skin tissue and other cartilaginous organs such as nose and ear (Figures 7.5 and 7.9). Taken together, POSS-PCU appears highly advantageous, being immunogenically inert while providing nanostructural anchorage points for cellular attachment, proliferation and differentiation. The next section will provide a more in-depth insight into the features of nanocomposite materials to explain the underlying mechanisms of physical induction of cell fate.

Nanocomposites explained—the best of both worlds

Composite materials consist of two or more components of different classes. Nanocomposites consequently consist of two or more components with at least one material dimension below 100 nm. Although reinforcement of composites at the microscale

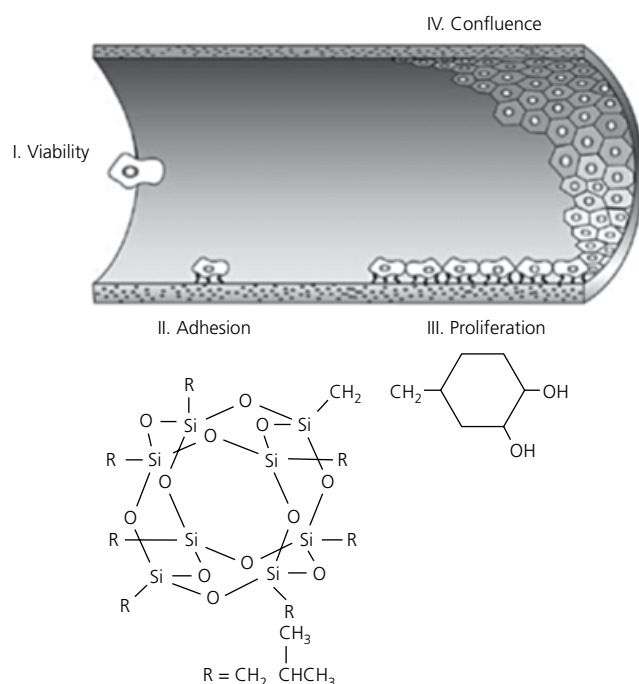


Figure 7.7 A POSS-PCU ilio-femoral bypass graft transplanted into a human patient. The POSS nanoparticles enhance the endothelialization potential of POSS-PCU grafts to optimize antithrombogenic properties.

(microcomposites) has already been shown to improve mechanical properties, mixing of the starting material with fillers at the nanoscale (nanocomposites) is postulated to have characteristics unanticipated by conventional laws of physics due to the quantum confinement effect¹³⁶ which maintains that the band gap width (which determines the energy needed to promote an electron from the valence to the conduction band and thus is proportional to the potential energy that can be emitted on the electron's return to the valence band) is inversely proportional to the particle's size. This quantum phenomenon underlies the principle that when a certain size (usually <10 nm) is reached, energy levels exist not in continuous states but in discrete quanta, thus supporting the theorem of size-dependence. This can be attributed to the nanoparticle's surface properties and interfacial

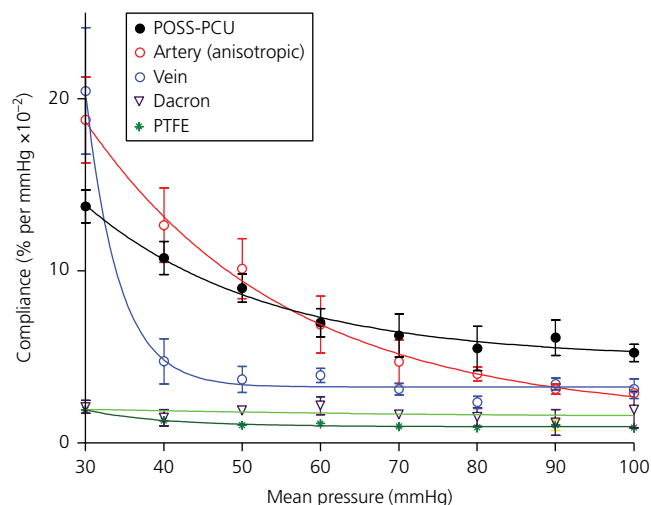


Figure 7.8 Comparison of compliance profiles of native arteries (red) with native veins (blue), commercially available vascular replacement grafts (Dacron, dark green; PTFE, light green) and POSS-PCU grafts (black). POSS-PCU grafts show viscoelastic properties similar to those of native arteries.

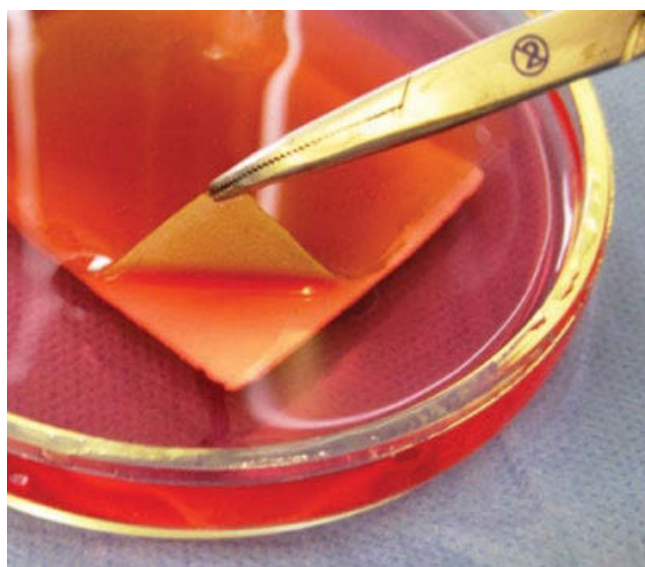
interactions, as with decreasing dimensions surface area-to-volume ratio increases exponentially, allowing for substantially more reactivity between particle surface molecules and polymeric matrix. Such minute dimensions permit tremendous interfacial contacts between the nanoparticle and the components of the polymer matrix, leading to the aforementioned synergistic improvements of the composite material.

Nanocomposite materials can be classified into biological,¹³⁷ synthetic¹³⁸ or hybrid¹³⁹ materials, with synthetic nanocomposites seemingly dominating the biomedical market. POSS nanoparticles are hexagonal nanocages of roughly 1.5 nm in diameter and can interact with the polymer matrix in all three dimensions.

Polyhedral oligomeric silsesquioxane

First introduced into the biomedical field as an alternative, more biocompatible breast implant material in the 1960s, POSS has risen considerably in global popularity ever since.^{140,141} This is because of its unique 3D cage structure consisting of Si-O and Si-C bonds which can serve as nano-sized building blocks in the manufacture of unique and precisely controlled hybrid materials.^{142,143} POSS is a highly versatile member of the silsesquioxane family and may be shaped into ladder structures, cage and partial cage structures. Nanocomposite materials incorporating POSS are particularly well suited for biomedical applications due to their biostability and degradative resistance, which is due to shorter bond lengths and strong intermolecular forces leading to a strong framework.¹⁴⁴

POSS is regarded the smallest possible silica particle, with an overall diameter of 1.5 nm including the R groups.¹⁴⁵ It has an extraordinary potential to succeed both in scientific, biomedical



(a)



(b)



(c)

Figure 7.9 (a) Bilayered skin regeneration scaffold based on the biodegradable version of POSS-PCU. (b, c) A heart valve using POSS-PCU leaflets, soon to be in clinical trials.

and commercial terms due to several synergizing factors: (a) POSS is inherently versatile, thus customizable, due to the ability to attach any chemical moieties to the organic vertex groups (R), (b) the manufacturing process with yields of up to 90% and more is straightforward, facilitating large-scale production, (c) the raw materials are inexpensive, thereby making commercialization economically beneficial with a multitude of possible applications ranging from packaging over inert material coatings to biomedical system enhancements,^{129,136,146} and (d) its rigid inorganic core renders the POSS molecule chemically and thermally stable. This 'self-protection' property of POSS is increasingly exploited for biomedical purposes to create an ideal and dually active biosynthetic interface combining both antithrombogenicity and amphiphilicity.^{147,148} Antithrombogenicity is a principally important feature of cardiovascular graft materials because thrombo-occlusive events with subsequent early graft failure represent the main limiting factors for successful vascular bypass procedures. Amphiphilicity (i.e. the state of being simultaneously hydrophilic and lipophilic) is postulated to be of great exploitable significance

within the field of regenerative medicine by rendering the material highly versatile and enhancing cellular attachment by providing a suitable interface.^{149,150}

Conclusion – Are we there yet?

Recent high profile in-human transplantations of our scaffold have underlined its superb biointegrational ability as well as given credence to the clinical translational potential of laboratory implementation. This chapter has summarized the parameters important for scaffold fabrication and tissue regeneration that seem to underlie the elucidation of the ideal combination of scaffold architecture, mechanical properties and cellular integration.

Decades of research into TE scaffolds have focused on recreating as much as possible various human body parts, frequently using biologically derived biomaterials. The development of the novel and completely synthetic POSS-PCU nanocomposite scaffold and its successful clinical application as a regeneration scaffold raises the question whether or not the scientific community has inadvertently built too restrictive walls in order to mimic as closely as possible the human body, including all its imperfections, which ultimately led to the initial requirement for TE strategies. Thus, instead of attempting to recreate imperfections, perhaps we need to shift our focus and the scientific community should start again with a tabula rasa to re-evaluate the importance of different components and the functions of our organs and tissues.

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PART II

Integument

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CHAPTER 8

Skin structure

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The skin is the largest organ in the body, covering around 2 m² in an average adult. It is imperative for barrier protection of the host and to prevent water loss. The skin also plays important roles in initiating immunological defence, thermoregulation and metabolism. The aesthetics of the skin and hair appendages are often overlooked as a function, but these provide the building blocks for social interaction.

This chapter will outline the structure of the skin and its functions. The development of the skin will be traced from its origin in the embryo to the maintenance of homeostasis in the mature adult. An understanding of the basic structure of skin gives clarity to the impact of debilitating skin diseases that affect the components of skin, while stem cell research provides hope that novel cures await.

There are three components to skin: the epidermis, dermis and hypodermis. The epidermis is stratified into four layers:

- the stratum basale;
- the stratum spinosum;
- the stratum granulosum; and
- the stratum corneum.

A fifth layer (stratum lucidum) acts to prevent shearing in thick-skinned areas, such as the hand, between the stratum granulosum and stratum corneum.

The stratum basale is adherent to a basement membrane – the junction between dermis and epidermis – and consists primarily of keratinocytes. Melanocytes (pigment cells), Merkel cells (light touch) and Langerhans cells (antigen-presenting cells) are also found at this level.

The keratinocytes go through the process of proliferation and then differentiation in the stratum spinosum and then granulosum. The terminally differentiated keratinocyte (now referred to as the corneocyte) is stripped of its nucleus to provide the tough protective layer of skin in the stratum corneum. These cells slough off and regenerate from the stratum basale to replenish the reservoir of corneocytes.

The dermis is composed of only two layers: the papillary dermis and reticular dermis. The papillary dermis lies deep to the basement membrane. As its name implies, it is undulating

with the rete ridges of the epidermis. The straight layer deep to this is the reticular dermis, which is primarily composed of elastin and collagen dermal matrix. The appendages to the skin traverse the two structural layers, with the base of the hair follicle, sebaceous glands, sweat glands, vascular supply and lymphatics lying within the dermis (Figures 8.1 and 8.2).

The blood supply to the skin and its appendages is divided into two horizontal plexuses. The upper lies at the level of the papillary dermis, whereas the lower lies at the dermal–hypodermal junction. The upper plexus arterioles divide into capillary loops which supply nutrients to individual dermal papilla (see Hair).¹ Flow between the two plexuses occurs through paired arteriole/venous communications. In addition to nutrient supply to the skin, thermoregulation is aided by the horizontal plexus system. Tactile and nociceptive nerve endings are found in the dermis and all layers of the epidermis² with Meissner corpuscles (light touch receptors) within the papillary dermis. Nerves not only feedback sensory information, but also have been shown to contribute to epidermal homeostasis.³ Skin that has lost nervous innervation is irregularly thin in comparison to the innervated skin.

Epidermal structure and differentiation

The morphology of the epidermis is well understood; however, the molecular process that controls cellular renewal and differentiation are the subject of ongoing research. The basal keratinocytes undergo a process of proliferation and then differentiation to form the four layers. Epidermal stratification relies on two methods: detachment from the basal membrane – known as delamination – and asymmetric cellular division, where the daughter progenitor cell is pushed suprabasally.⁶

The cuboid structure of the stratum basale morphs into the stratum spinosum with the addition of a spinous process to the cell. The transition to the stratum granulosum includes the formation of lamellar bodies (LB) in the cytosol of the keratinocyte. These LBs excrete lipid precursors into the intercellular

matrix, while dense cross-linked proteins engulf the cell membrane. The nucleus and organelles of the cell undergo apoptosis and the skeletonized body remains as the terminally differentiated keratinocyte takes its final form as a flattened polyhedron corneocyte. These two features form the basis for the formation of the barrier membrane.⁷

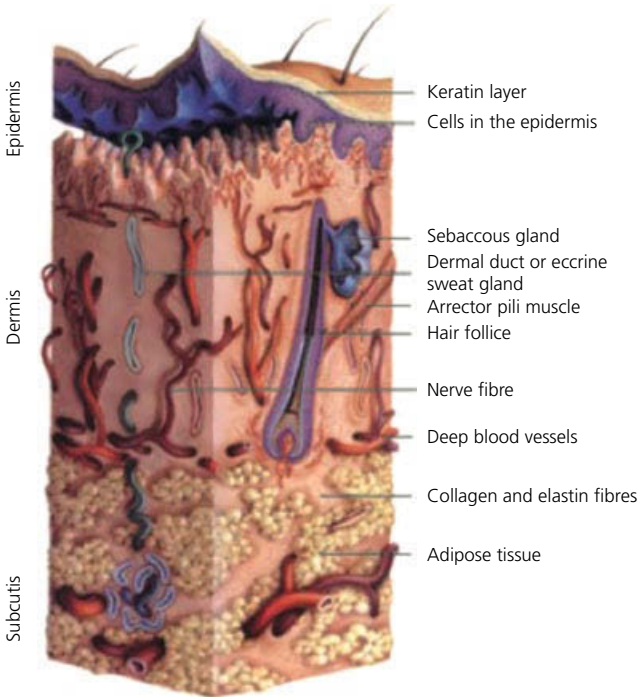


Figure 8.1 Skin structure and appendages of the dermis and epidermis. Source: Bhushan *et al.*, 2010.⁴ Reproduced with permission of Elsevier.

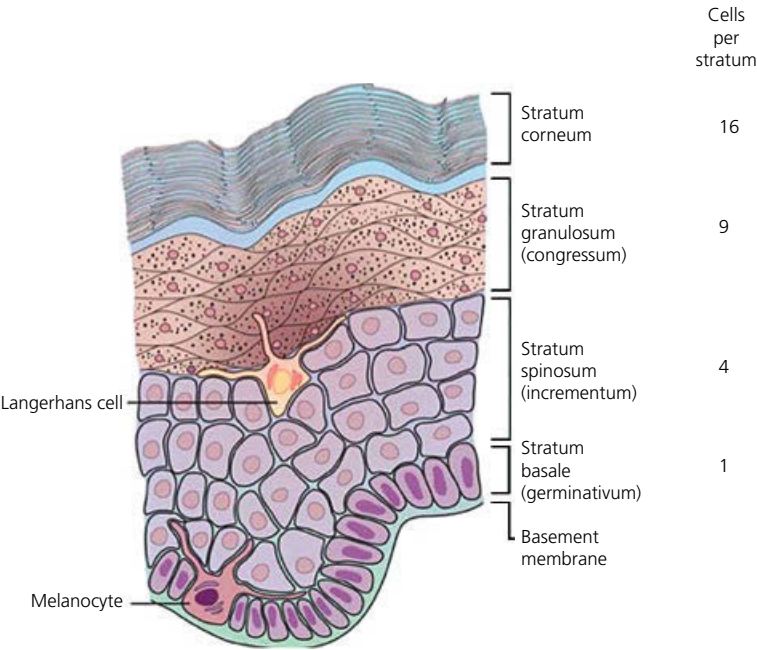


Figure 8.2 Epidermal layering of keratinocytes with the location of melanocytes and Langerhans cells. Source: Hoath and Leahy, 2006.⁵ Reproduced with permission of Elsevier.

The morphogenesis from basal keratinocyte to barrier-protecting corneocyte takes approximately 14 days. It is a further 14 days before the squame is sloughed from the outer layer. The presence of keratin-5/keratin-14 (K-5/K-14) followed by the replacement with keratin-1/keratin-10 (K-1/K10) marks early differentiation, while the formation of loricrin, fillagrin and involucrin are indicative markers of terminal differentiation.^{7,8}

Homeostasis of the skin relies on multiple regulatory pathways that share commonality with those that are also involved in embryogenesis of the epidermis. At the heart of the process are p63 and Notch signalling.

p63 is the key regulator in determination of epidermal lineage (see Embryology) as well as basal proliferation and differentiation. The dominant epidermal isoform is deltaNp63.⁹ This isoform has important roles in shaping the structural properties of basal keratinocytes, while also signalling the proliferation and then differentiation of the basal keratinocyte. Both integrins and desmosome expressed on the keratinocyte membrane are regulated by p63. They are necessary for both cell-cell and cell-basement membrane adhesions – the structural integrity of the skin is compromised if these unions cannot be formed.¹⁰ p63 is the initial signal for expression of K-14 in basal keratinocytes; without this input, the ectodermal cells would remain in their non-proliferative embryological keratin-8/keratin-18 state.¹¹

The Notch pathway is antagonistic to p63, promoting differentiation and suppressing p63's proliferative potential when highly expressed. This regulatory balance is the basis for homeostasis in adult skin, where the equilibrium exists between the requirement of proliferation and cellular differentiation of the layers.^{12, 13} The Notch pathway is necessary for terminal

differentiation of mature basal keratinocytes and for release of the cells from the basement membrane.⁶ Immature daughter cells of basal progenitor cells, however, will not undergo terminal differentiation in the presence of Notch until reaching maturity.¹⁴ p63 and Notch's role in proliferation and differentiation allow the skin to continually renew its function as a barrier to the external environment.

Barrier formation

The combination of stratification and differentiation forms the skin's barrier function. The final process of cornification results in a membrane that provides rigidity, prevents water loss and gives protection to the external environment. This is achieved through cross-linked proteins that surround the now keratinized core and an extracellular lipid mesh that seals the corneocytes. The resulting hydrophobic environment that surrounds the corneocyte is referred to as the corneocyte envelope. Desmosomal structures (termed corneodesmosomes) and the lamellar lipid network bind the corneocytes together to form the skin barrier.

The primary lipids of the corneocyte envelope are ceramides, fatty acids and cholesterol, which are formed in the Golgi apparatus in the granular layer. These lipids are stored in lamellar bodies before being released into the extracellular environment during cornification, where enzyme modification results in the lipid structure. Concurrently, the production of profilaggrin by the keratohyalin granules in the granular layer is the precursor to filaggrin. Filaggrin condenses the keratin in the cell to give the corneocyte its unique flattened polyhedral shape. The resulting filaggrin matrix constitutes 80–90% of the epidermal protein weight.¹⁵ Terminal protein markers of differentiation, loricrin and involucrin, have important structural roles in the corneocyte envelope. Involucrin is often described as the scaffold to which other proteins cross-link; it also provides covalent bonding with the external lipid network.¹⁵ Loricrin is cross-linked with small proline-rich proteins (SPRs) before incorporation beneath the plasma membrane. Transglutaminases synthesize these proteins into cross-linked peptides; the strength of the corneocyte envelope is reliant upon the isopeptide bonds formed by the transglutaminases.¹⁵

Basement membrane

The basement membrane is the acellular divide between the epidermis and dermis. The primary function of this membrane is to resist shear forces between the epidermis and dermis, while creating a physical bond between the two structures. In addition, the basement membrane plays important roles in establishment of keratinocyte migration in the epidermis and polarization for keratinocyte cell division.¹⁶ Structurally, the basement membrane is similar to the mature skin by the end of the first trimester¹⁷ and is reliant upon epithelial–mesenchymal interaction for its development.^{18,19}

The four components of the basement membrane are: the plasma membrane of the keratinocytes, the lamina lucida, lamina densa, and sublamina densa.²⁰ Integrins link hemidesmosomes on the plasma membrane of the basal keratinocytes to the basement membrane.¹⁹ The thin lamina lucida is composed of laminin-332 and laminin-511 (previous nomenclature was laminin-5 and laminin-10, respectively), while collagen IV along with nidogens and perlecan are predominant in the lamina densa that provide mechanical strength.¹⁶ Bridging of collagen IV with nidogens and perlecan join the lamina densa to the laminin scaffold of the lamina lucida.²¹ Finally, collagen VII in the sublamina densa extends to underlying stromal tissue through anchoring fibrils that insert into plaques in the reticular dermis. This is the strongest point of epidermal–dermal adhesion within the basement membrane, followed by the hemidesmosome attachments from keratinocytes.¹⁷ Fibroblasts and keratinocytes are responsible for the deposition of collagen IV and VII along with regulation of the other basement membrane components from these key cells.¹⁹

Dermis

The dermis lies deep to the epidermis and the basement membrane. It is composed of the undulating papillary dermis and the straight reticular dermis.

The papillary dermis interdigitizes with the rete ridges of the epithelium at the dermal–epidermal junction. It serves to increase signalling between the layers and provide nutrient supply.²²

The predominant reticular dermis houses the extracellular matrix of collagen, elastin, glycoaminoglycans and adipocytes. The elastin fibres are composed of an elastin core surrounded by microfibrils of primarily fibrillin.²³ These fibres in the reticular dermis are thick and arranged in parallel to the skin surface. This arrangement contrasts with the thin perpendicular elastic fibres (elastin fibres) of the reticular dermis. The elastin fibres give the skin its characteristic resilience to deformity.²³

At least half of all collagens are expressed in the skin, with the fibril-forming collagens I and III being the most abundant in the dermis, lying parallel to the skin surface (in the reticular dermis).²¹ The collagen fibrils provide a scaffold for other proteins to attach to, which confers tensile strength to the skin.²¹

Hyaluronic acid, a glycosaminoglycan, is responsible for water retention in the dermis, conferring the skin's plump fullness when saturated. Fibroblasts in the dermis are primarily responsible for the synthesis of the extracellular matrix. Below the dermis lies the hypodermis, with the reservoir of subcutaneous tissue that provides the stored energy for development in adipocytes.

Embryology

The embryonic development of the epidermis is from the ectoderm with signalling from the mesenchyme. The ectoderm is evident from embryological day 21, where it consists of a single proliferative layer. Bilayer formation with the periderm occurs from day 55 before the formation of an intermediate layer at day 70. Four-layer stratification is evident at day 90, including the stratum corneum – indicative of late terminal differentiation (Figure 8.3).²⁴ The maturation of the stratum corneum is necessary for the assault on the skin from the extra uterine environment. With the formation of the stratum corneum, the periderm is sloughed, allowing the skin to form its mature barrier protection. The immediate function of skin in the neonate is to ensure water retention to prevent potentially lethal dehydration. By the end of the first month post partum, the transepidermal water loss (measured in g/m² per h) is similar to that in an adult. In addition, the acidity of the skin increases by the end of the first week post partum, which enhances the mechanical and immunological defence mechanism of the skin.²⁵

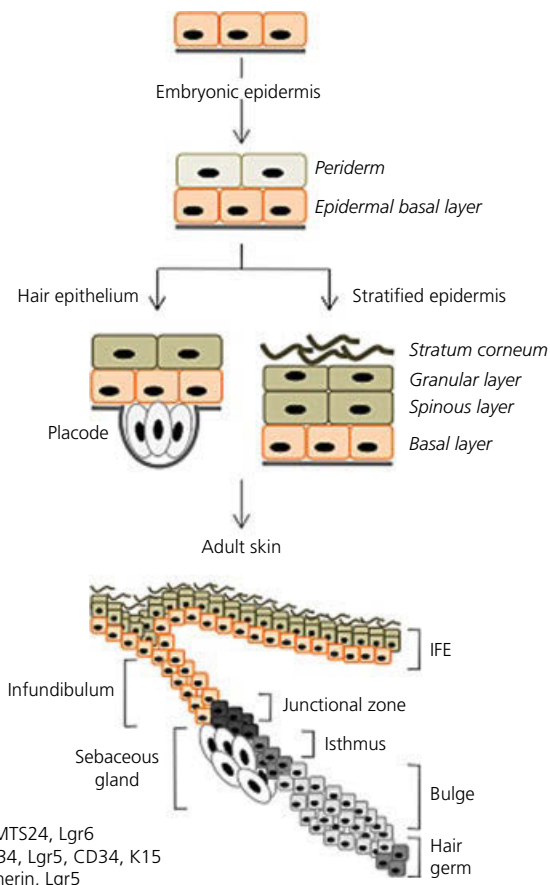


Figure 8.3 Ectodermal lineage selection promotes stratification of the newly formed epidermis. The protective surface of the periderm allows the immature epithelium to form. Hair follicle formation originates from the hair placode adjacent to the interfollicular epithelium (IFE). Some of the many stem cells markers of this region are indicated in the left corner. Source: Benitah and Frye, 2012.³³ Reproduced with permission of Elsevier.

Skin appendages

Hair

Hair serves multiple purposes, including thermoregulation, sebum and sweat dispersion as well as important social functions. Human hair is composed of pigmented terminal hairs – primarily found on the scalp – and the universally found vellus hairs.²⁶

The structure of the pilosebaceous hair follicle from deep to superficial is the dermal papilla, followed by the basement matrix that provides the growth front for the hair follicle. The regenerative capacity of the hair follicle is stored in the bulge zone, which is activated when the follicle grows. The sebaceous gland opens distally to the bulge with the erector pili muscle attaching at this level. The isthmus is the bridge between the hair bulge and the transition zone before the hollow infundibulum continues out into the interfollicular epithelium (IFE) (Figure 8.3).

The hair shaft is formed from dividing cells of the bulge, which migrate to the base of the shaft – now termed matrix cells – before stratifying into the cells of the hair follicle. The hair follicle is divided into three zones: the outer root sheath (ORS) that maintains its connection with the basement membrane; the inner root sheath (IRS); and the hair shaft, all of which are composed of terminally differentiating keratinocytes formed from the matrix cells.²⁷

Pigmentation of the hair shaft comes from melanin (eumelanin for black and pheomelanin provides red colouration). Melanin is released from hair follicle pigmentation units near the dermal papilla. The principal cell of the pigmentation unit is the melanocyte, which is in close proximity to the melanin-receiving keratinocytes.²⁶

Hair follicles form from signalling between the developing mesenchyme and epidermis. The initial hair placode forms in the IFE and projects into the dermis. Signalling from the placode forms a dermal ‘condensate’ of fibroblast cells known as the dermal papilla.^{26–29} The downward-growing placode (soon to be hair bulb) and the dermal papilla constitute the growing hair follicle. The initial signal for hair follicle formation comes from the Wnt/beta-catenin pathway, which is evident from knockout of beta-catenin which universally affects hair follicle formation in the murine model.^{26,27} The regular spacing hexagonal spacing of new hair follicles in the epidermis is evidence that signalling from the hair follicle has an inhibitory effect on where the next follicle is placed.²⁷ This ensures maximum resource distribution between developing hair follicles.

The necessity of the hair follicle to shed and renew requires the ability of the matrix to undergo cell cycling. Matrix cells proliferate through the anagen phase, resulting in the growth of the hair shaft (Figure 8.4). During catagen, the apoptosis of these cells result in shrinkage of the dermal papilla until it is level with the regenerative cells of the bulge.²⁶ Telogen is the resting phase of the cell cycle, where the matrix cells lie dormant until signalled to once again undergo proliferation. With age, the length of time for the cells to enter anagen increases, thereby decreasing cycling frequency.²⁶

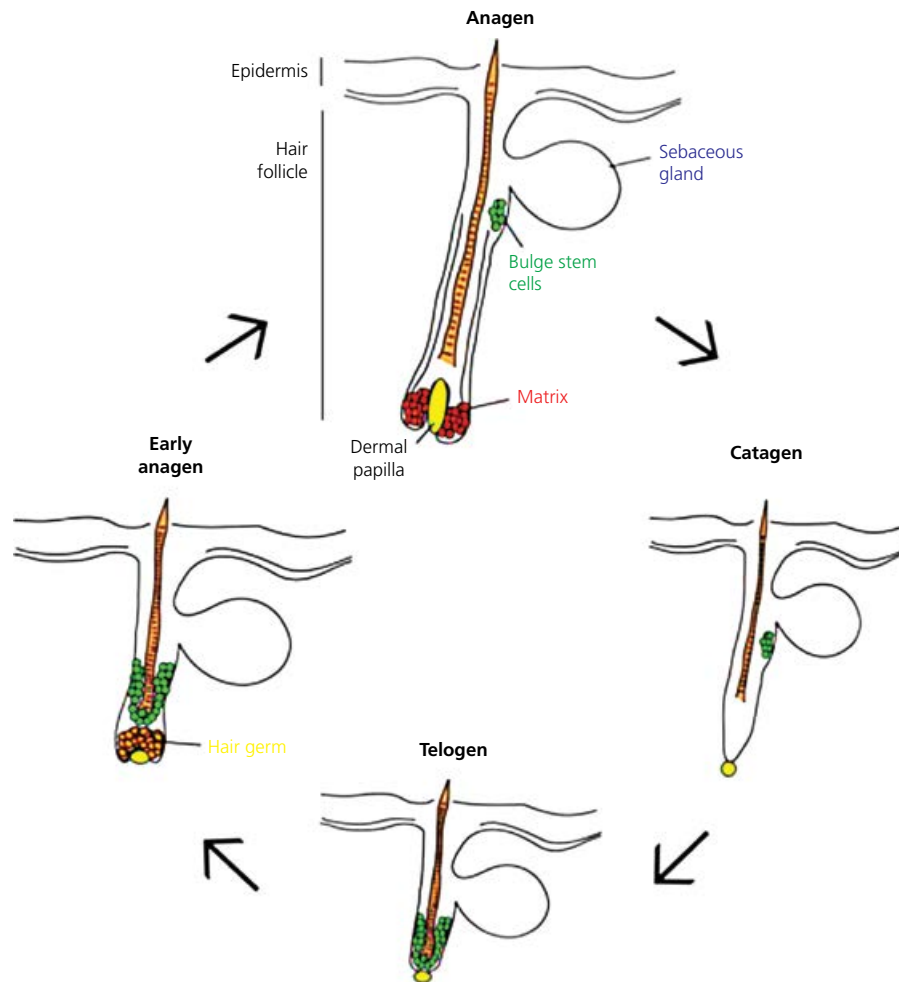


Figure 8.4 Hair follicle growth originates in early anagen whereby the bulge cells are stimulated to multiply. These cells migrate to the matrix initiating the growth phase of the hair follicle in late anagen. Catagen signals the apoptosis of the matrix and the shedding of the hair shaft. During telogen, the hair follicle is refractory to growth until the dermal papillae returns the level of the bulge cells. Source: Goldstein and Horsley, 2012.³⁰ Reproduced with permission of Springer.

Sebaceous glands

Sebaceous glands empty sebum – a dense lipid-filled mixture – into a secretory duct in the junctional zone of the hair follicle. The waxy sebum prevents water loss from the IFE and assists in thermoregulation.³¹ The sebaceous gland is a sac-like structure lined with keratinocytes that specialize to sebocytes. The sebocytes differentiate concentrically until the core undergoes cell death, resulting in a central core of necrosis. The lysis of the necrotic cells results in the release of lipids into the secretory duct usually associated with the hair follicle. The sebaceous gland is formed in the human embryo at the 13th to 14th week of gestation; however, it is not until puberty that the sebaceous gland enlarges in response to sex hormone stimulation.

Stem cells

Stem cells are the focus of recent investigation into the regenerative capacity to maintain homeostasis of the IFE, hair follicle and sebaceous gland. Stem cells are defined by their ability to

undergo lineage commitment. The cyclical process of epidermal renewal in the IFE, hair follicle and sebaceous gland suggests that a single cell could be responsible for lineage renewal. Isotope tracing has identified such cells in the bulge of the forming hair follicle as well as a separate multipotent cell in the upper isthmus. The cells of the bulge undergo their cycle slowly and appear to have the ability to differentiate into cells of the ORS, IRS and hair shaft. They express for the transcription factor Sox9, which promotes the expression of the CD34 cell receptor.³² The multipotent cells of the upper isthmus expressing MTS24 surface marker were traced to IFE, sebaceous gland as well as hair follicle.^{30,32} In addition, Lrig1-positive cells of the transition zone can also be traced to all three epidermal lineages and provide a pool of regenerative cells for the IFE^{30,33,34} (see Figure 8.3).

Whether these stem cell groups function independently, or form from the same multipotent progenitor, remains the focus of research. It is unclear if any of these lineage cells, once terminally differentiated, are able to regress to their original progenitor cell. However, it has been found that the pathways responsible for

morphogenesis have similar roles in homeostasis.^{31,32} Further research in this area will help elucidate the role of stem cells and their potential for wound healing, regeneration of skin appendages and the role of stem cells in cancer.

Fitzpatrick's skin classification

Skin type is commonly classified from type I to type VI based upon Fitzpatrick's skin classification system. American dermatologist TB Fitzpatrick designed the original classification system based on the response of skin to the minimum erythema dose (MED) of UVB. He based skin types I–III on his 1972 study of response to a time-dependent exposure of midday sun in Australians with Caucasian skin type. Type I skin was characterized as skin that burns easily with no appreciable tan at one week post exposure; type II skin burned but formed a light tan at one week; and type III skin had little burn and moderate tan at one week post exposure.³⁵ Subsequent formulation of types IV–VI included pigmented skin. Type IV skin did not burn and tanned at one week, whereas types V and VI are characterized by brown and black pigmented skin, respectively.³⁶

Determination of the Fitzpatrick skin classification is by a series of nine questions scored out of 4 points. These questions are broken into two sections: genetic disposition and sun exposure.³⁷ One criticism of the classification system relates to the subjectivity in issuing the questionnaire. However, several studies have shown that there is a strong correlation between cancer risk and skin type for both non-melanocytic and melanocytic cancer.^{37,38} In addition, skin type is an important determinant in evaluating treatment doses for laser therapy and topical treatments.

Ageing skin

The ageing process of the body is visibly manifested in the skin. Aged skin is characteristically thinner and dry with visible wrinkling, reduced elasticity and abnormal pigmentation.³⁹ The causes can be classified into changes at the cellular or extracellular level. Cellular damage involves mutations to the DNA that have the potential to cause abnormal cell functioning and signalling. Extracellular damage can manifest in protein degradation or reduced synthesis of proteins, importantly collagens I and III in the dermis as well as glycosaminoglycans and proteoglycans.^{39,40} The flattening of the dermal–epidermal junction results in the loss of the papillary vasculature and reduced ability to deliver nutrition to the skin.⁴¹

Epidermal stem cells associated with homeostasis of the skin barrier are unique in the body: the frequency in which they divide and the number of stem cells are maintained through adulthood. These stem cells are not instigators of the ageing process.⁴² The widely accepted cause of ageing is a result of accumulation of reactive oxygen species (ROS).³⁹ The ROS cause damage to the

cell's DNA as well as the mitochondria, which can result in a shift to glycolytic anaerobic metabolism.⁴³ This shift increases the accumulation of oxidative free radicals that can result in the degradation of collagen through the indirect stimulatory effect of ROS on matrix metalloproteinase (MMP) production.³⁹

There are many treatments available to prevent skin ageing. Topical application of coenzyme Q10 is shown to stabilize the mitochondria and help prevent the oxidative effects of the glycolytic pathway.⁴³ To improve the skin texture and density, topical application and nutritional intake of antioxidants have proven efficacy.⁴⁴ Preventative measures for premature ageing of the skin involve the avoidance of UV exposure and smoking. UV has a similar destructive pathway on skin as ageing with the promotion of MMP production, resulting in the breakdown of collagen.³⁹ The skin of smokers is dry and dull with associated wrinkling of the mouth from the pursed lip used for smoke inhalation. Smoking increases the production of collagenase while reducing its blood supply, resulting in the characteristic smokers' skin.³⁹

Clinical diseases

Disease of the skin manifests at all structural layers of the skin. The following review of skin diseases gives examples of how the molecular structure of the skin can be compromised.

Connective tissue diseases

Ehlers–Danlos syndrome is a heterogeneous group of diseases that affect the collagenous network of the dermis. The clinical presentation is of weak, hyperelastic and easily bruised skin. Ehlers–Danlos syndrome results from deficits in the production or quality of collagen fibrils I, III and IV. These deficits are caused by mutations in the genes that code for the collagens or their associated structural proteins or production pathways.²¹

In contrast, the appearance of skin in the rare disorder cutis laxa is loose tissue that gives an appearance of premature ageing. Cutis laxa is caused by deficits in the production of elastic fibres, which result from a heterogeneous group of mutations that affect either the composition of elastin or the microfibril, which disrupt the elastic fibre composition. In autosomal recessive cutis laxa, the disorder not only affects dermal tissues, but also causes cardiovascular deficiencies and pulmonary emphysema.⁴⁵

Marfan's syndrome is known for its multiple and severe cardiac manifestations caused by a mutation of the gene for fibrillin-1 that results in both morphological changes to the microfibril and an increase in transforming growth factor beta (TGF- β) signalling. TGF- β stimulates the increased production of collagen and the resultant skin is thin with increased laxity.^{21,45}

Disease of the basement membrane and epidermis

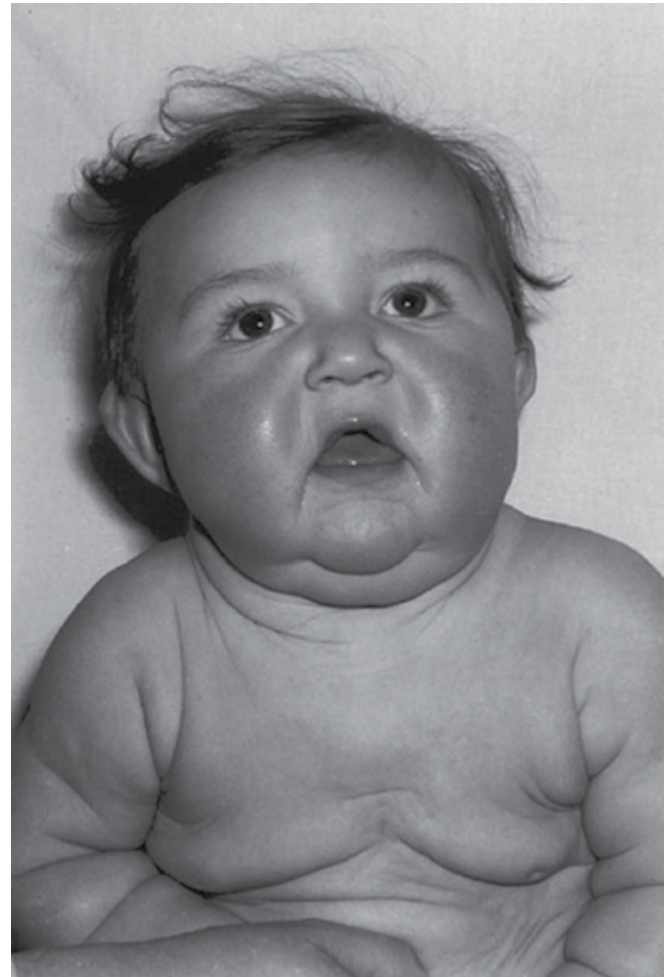
Epidermolysis bullosa is categorized into inherited and autoimmune disease. The disease is characterized by bullous blisters that occur either in the epidermis or the basement membrane.



(a)



(b)



(c)

Figure 8.5 (a) Blistering associated with DEB. (b) Resultant chronic scarring from DEB. Source: Van Agtmael and Bruckner-Tuderman, 2010.⁵² Parts (a) and (b) reproduced with permission of Springer. (c) Cutis laxa. Source: Tassabehji *et al.*, 1998.⁴⁷ Part (c) reproduced with permission of Oxford University Press.

Epidermolysis bullosa is caused by weakening of the dermal–epidermal attachment of keratinocytes at the basement membrane. There are three types of inherited epidermolysis bullosa: epidermolysis bullosa simplex (EBS), junctional epidermolysis bullosa (JEB) and dystrophic epidermolysis bullosa (DEB).⁴⁶

EBS forms blisters at the junctional layer due to mutations in the gene coding for keratinocyte proteins K-5 and K-14, which are essential for cellular connectivity and structure.

JEB is a result of mutations in collagen XVII or laminin-332, which link keratinocytes to the basement membrane in the lamina lucida. The most severe form is the Herlitz variant where the absence of laminin-332 results in ulcerative erosions. Without epithelial regenerative capacity, uncontrolled fluid loss is lethal within the first year of life.

DEB is inherited as the severe autosomal recessive form or the mild dominant form, affecting collagen VII formation and the adhesion of the anchoring fibrils into the sublamina densa. The resultant chronic wounds are healed by extensive fibrosis formation (Figure 8.5).

Two autoimmune diseases have the same clinical appearance as epidermolysis bullosa: bullous pemphigoid and epidermolysis bullosa acquisita. In bullous pemphigoid IgG autoantibodies target collagen XVII, which is a transmembrane protein responsible for keratinocyte adhesion to the basement membrane. The IgG autoantibodies in epidermolysis bullosa acquisita attach to the binding sites of collagen VII, preventing adhesion to laminin-332 and thereby stopping adhesion of the anchoring fibril to the basement membrane.⁴⁶

The previously untreatable condition of epidermolysis bullosa is the focus of multiple treatment pathways from protein replacement to gene modification and stem cell transplantation,⁴⁸ which give hope for treatment options for these debilitating and potentially lethal diseases.

Current research in recessive DEB has achieved synthesis of collagen VII via the introduction of fibroblasts in the preclinical murine model, with improved strength at 100 days post implantation.⁴⁹ Another potential treatment method involves the injection of collagen VII directly into the wound sites: at four

weeks post injection in mice there is improved integrity of the dermal-epidermal junction.⁵⁰ Finally, mesenchymal stem cells derived from bone marrow were used in human trials to stimulate the production of collagen VII.⁵¹ The progress of this and other treatment options for the spectrum of debilitating skin diseases will provide new hope for those suffering from the multiple heterogeneous disorders of the skin.

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CHAPTER 9

Melanoma

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Epidemiology

Over the past four decades, melanoma has shown a consistent increase in incidence in White populations, becoming one of the most frequent cancers in light skin populations. The incidence rate of this disease varies widely in relation to race, occurring much less commonly in Black, Asian, or Hispanic populations. Although White and non-White populations have similar risks of developing plantar melanoma, non-White populations have a greater risk of developing non-cutaneous melanomas (e.g. mucosal). Melanoma is now regarded as the fifth most common cancer in men and the sixth most common cancer in women in the United States. The highest recorded incidence of melanoma worldwide is in Australia and New Zealand, with an incidence of 27.3–55.8/100 000 for males and 23.4–41.1/100 000 for females. In the USA, the incidence rate is 19.4/100 000 for men and 14.4/100 000 for women. In European countries, the incidence rates are lower, being highest in Switzerland and Scandinavian countries. Moreover, they show a north to south gradient, with the highest rates in northern countries. This is probably related to both the increased protection against the sun rays of the pigmented skin of southern Europeans and the different pattern of sun exposure (chronic in southern Europeans, intermittent in northern Europeans).¹

In many countries, early detection of primarily thin melanomas and improved survival rates have been observed, especially in young females. However, in most countries the incidence of thick melanomas remains constant or continues to increase, especially in the older age group. Melanoma occurs approximately 1.5 times more often in men than women, and prognosis is slightly better for women, considering other prognostic factors. The anatomic distribution of melanoma is sex-dependent, occurring more often on the trunk in males and on the arms and legs in women.

Melanoma can occur at any age but is rare before the age of puberty. The median age at the time of diagnosis ranges between 45 and 55 years, with an increase in incidence after the age of 25 until 50 years.

Risk factors

Current considerations in the epidemiology of melanoma showed that melanoma results from an unidentified interaction between mutations in various genes and constitutional and/or inherited factors combined with environmental factors, mainly solar ultraviolet radiations.^{2,3} Exposure to UV is the most important environmental factor predisposing to melanoma in susceptible populations. It is reported that people incurring severe burns in childhood appear to be at higher risk for the development of melanoma in later age. Iatrogenic exposures, such as immunosuppressive agents and non-steroidal anti-inflammatory drugs, have been studied as additional environmental factors. Well-known host risk factors, such as melanocytic naevi on the skin, skin type, family history and genetic susceptibility, have been validated as risk factors in large-scale association studies. Patients with more than 100 naevi or giant naevi (>20 cm) have an increased risk of melanoma. In addition, genome-wide association studies have revealed genetic loci that underlie the genetic susceptibility of melanoma, some of which are related to known risk factors. Mutations or epigenetic silencing of the cyclin-dependent kinase (CDK) gene are linked to 20–40% of familial melanoma. Other genes involved in familial melanoma carcinogenesis include *CDK4*, xeroderma pigmentosum genes and melanocortin 1 receptor gene (*MC1R*). Activating mutations in the *BRAF* gene have been discovered in over two-thirds of melanomas. Melanomas on skin without chronic sun-induced damage had frequent mutations in *BRAF*; in contrast, acral and mucosal melanomas as well as skin melanomas with chronic sun damage are frequently characterized by *Kit* mutations. These mutations are responsible for cancer cell behaviour through mechanisms that are still undefined.

Clinical diagnosis

Melanoma usually appears as an irregularly pigmented skin lesion with an irregular border and a tendency to grow or change over time. Melanomas arise mostly de novo, just occasionally

within a congenital or acquired naevus. In the past, they were often diagnosed in advanced stages as large, ulcerated or vegetating lesions. The need to detect melanomas at an early stage of disease led to the development of the ABCDE criteria, an acronym for asymmetry, border irregularity, colour alterations, diameter >6 mm and evolution. Not only are these simple criteria intended to allow both patients and non-dermatologist healthcare professionals to differentiate common melanocytic lesions from suspicious pigmented lesions, they are also a useful tool for dermatologist in guiding the decision to perform a biopsy. The sensitivity of clinical diagnosis by physicians with experience in differential diagnosis of suspected cutaneous lesions is about 70%. Dermoscopy, also known as epiluminescence microscopy, improves the diagnostic accuracy of melanocytic lesions. Dermoscopy, when adopted by experts, increases the sensitivity of clinical diagnosis up to 89%, reducing the number of negative biopsies. In high-risk patients (e.g. with atypical mole syndrome), a follow-up examination with digital dermoscopy and total-body microphotography is also helpful in detecting early changes in lesions or in discovering new atypical lesions. In patients with a previous melanoma, the risk of harbouring a second melanoma is significant and implies scheduled examinations of the skin at regular intervals for detecting second tumours or skin metastasis.

Biopsy

In the presence of a skin lesion with a clinical suspicion of melanoma, the patient is referred for an excisional biopsy. The biopsy of a suspected lesion is a simple procedure, practicable under local anaesthesia, on an outpatient basis. The procedure consists in complete removal of the lesion with about 1–2 mm of surrounding healthy skin up to the subcutaneous fat. During the procedure, care should be taken to avoid causing trauma or injury to the specimen, which could compromise a correct histopathological analysis. The choice of incision lines of the biopsy must take into account the further excision of the skin involved, preferring longitudinal incisions in the biopsy of the limbs while trying to minimize as much as possible the need of skin grafts. Excisional biopsy allows an accurate histopathological diagnosis, providing all the histological parameters necessary to formulate a prognosis and to plan the best therapeutic strategy. However, excision of the whole lesion may not be indicated in some cases: for example, in the presence of a large lesion, or in the case of proximity of the lesion to difficult areas (subungual) or important structures (eye, nose and ear). In these circumstances, punch or incisional biopsy should be considered. In the incisional or so-called 'punch' biopsy, only a full-thickness portion or a pill of the most suspected portion of the lesion is removed. Superficial biopsies, 'shave biopsy', and any type of laser or diathermocoagulation should be avoided, as they do not allow an accurate pathological staging and can lead to a significant delay in diagnosis of any potentially malignant lesions.

Histology

The histological diagnosis of cutaneous melanoma is not always easy and it would be desirable for an expert pathologist in the diagnosis of pigmented lesions of the skin to perform the analysis. In doubtful cases, morphology may indicate the use of immunohistochemical surveys (such as HMB-45, Ki-67, p16) and fluorescence *in situ* hybridization (FISH) techniques, the reading of which requires experienced operators. Histologically, invasive cutaneous melanomas are divided into four major subtypes. These are based on growth pattern and location, and are categorized as follows:

- superficial spreading,
- nodular,
- lentigo maligna, or
- acral lentiginous.

A fifth subtype is also defined: desmoplastic melanoma. In general, the histologic subtype of melanoma is not a major factor in determining prognosis, but some histologic subtypes are more likely to be detected at an advanced stage, thus indirectly affecting prognosis. Melanoma originates from the proliferation of melanocytes in the basal layers of the epidermis, which tend to expand radially and later invade deep into the dermis.

Superficial spreading melanoma is the most common (70%), can occur in any location and is characterized by a radial growth associated in the later stages to a pattern of vertical proliferation. Nodular melanomas, occurring in about 15%, are characterized by an exception to this pattern, with an exclusive vertical growth not accompanied by radial growth. Because of its invasiveness, it is usually diagnosed at a more advanced stage of invasion. Lentigo maligna accounts for about 10% of melanomas and is characterized by onset in old age, in chronically sun-exposed skin sites with a history of slow growth. These lesions are characterized by a long phase of intraepithelial growth and for this reason are considered a lesion with a better prognosis, though always remaining linked to the degree of invasion. Acral lentiginous melanoma is the less common type (5%); it is classified mainly for its site of origin (subnail bed and the palmar and plantar surfaces) rather than for histological features, which are similar to melanoma of the mucous membranes. Diagnosis is often delayed, especially for subungual forms often confused with posttraumatic haematoma. Among the rarer variants, desmoplastic melanoma has non-typical melanocytic cells and is characterized by a significant deposition of collagen, which hinders differential diagnosis with other fibrohistiocytic tumours or schwannomas.

The vertical growth phase is the tumourigenic phase in which the melanoma acquires the ability to metastasize. Morphologically, it is characterized by the presence of an expansive nodule larger than the intraepidermal component and/or by the presence of mitotic figures in the invasive component. In 1969, Clark formulated a histological classification with a prognostic value for melanoma based on the level of invasion in the anatomical layers of the skin, highlighting for the first time the prognostic significance of the degree of invasion (Table 9.1). However, the

Clark system has proven to be a source of significant variability among pathologists, especially in stages III and IV, thereby losing its prognostic relevance in more recent years.

In 1970, Breslow described a more objective method of classification, based on the measurement in millimetres of the vertical thickness of the tumour.⁴ The Breslow thickness is measured from the granular layer or, if the lesion is ulcerated, from the bottom of ulceration, up to the point of maximum infiltration. This classification system has been shown to be more reproducible among pathologists, showing an excellent correlation with mortality. Prognosis tends to become progressively worse in logarithmic function, up to 8 mm where it

reaches a plateau, beyond which never reaching 100% mortality. Among histological features assessed for their prognostic value, the presence of ulceration and/or mitosis are considered the most important, correlating in a totally independent way to the prognosis. Ulceration is defined as the absence of intact epithelium overlying the melanoma. Patients with ulcerated melanomas have a worse prognosis even among those with regional nodal metastasis. Ulceration represents a phenotypic marker of aggressive tumour biology and predicts a greater propensity for invasion and metastasis.

In the same way, the mitotic index, although more recently validated, has been recognized as an important histological parameter in melanoma, especially in melanomas with Breslow less than 1 mm. The mitotic index is expressed as the number of mitoses per mm² in the invasive component of melanoma, rated from the areas with increased mitotic activity ('hot spots') after extending the count to an adjacent area of 1 mm². Several other prognostic factors have been recognized, such as tumour-infiltrating lymphocytes, regression, lymphatic invasion and microsatellitosis, but they showed minor effects on prognosis.

Table 9.1 Melanoma invasion expressed as Clark level

Level I	Confined to the epidermis
Level II	Invade but not fill or expand the papillary (superficial) dermis
Level III	Fill and expand the papillary dermis with extension to the papillary–reticular dermal interface
Level IV	Infiltrate into the reticular dermis
Level V	Infiltrate into the subcutaneous fat

Table 9.2 TNM staging categories for cutaneous melanoma

T category	Thickness (mm)	Ulceration status/mitoses
Tis	NA	NA
T1	≤1.00	a: Without ulceration and mitosis <1/mm ² b: With ulceration or mitosis ≥1/mm ²
T2	1.01–2.00	a: Without ulceration b: With ulceration
T3	2.01–4.00	a: Without ulceration b: With ulceration
T4	>4.00	a: Without ulceration b: With ulceration
N category	Number of metastatic nodes	Nodal metastatic mass
N0	0	NA
N1	1	a: Micrometastasis ^a b: Macrometastasis
N2	2–3	a: Micrometastasis ^a b: Macrometastasis c: In-transit metastasis or satellites without metastatic nodes
N3	4+ metastatic nodes, or matted nodes, or in transit metastases or satellites with metastatic nodes	
M category	Site	Serum LDH
M0	No distant metastasis	NA
M1a	Distant skin, subcutaneous or nodal metastases	Normal
M1b	Lung metastases	Normal
M1c	All other visceral metastases	Normal
	Any distant metastasis	Elevated

Source: *AJCC Cancer Staging Handbook* (7th edn), 2010. Reproduced with permission of Springer.

NA, not applicable; LDH, lactate dehydrogenase.

^aMicrometastases are diagnosed after sentinel lymph node biopsy. Macrometastases are defined as clinically detectable nodal metastases confirmed pathologically.

Staging

The most widely used staging system for melanoma is based on the analysis of the American Joint Committee on Cancer (AJCC) melanoma staging database (Tables 9.2 and 9.3).⁵ This staging system has recently been updated with important modifications with respect to the previous edition. Major changes include mitotic rate that replaces level of invasion (Clark) as primary criterion for defining T1b melanomas. Moreover, patients with microscopic nodal disease, regardless of extent of tumour burden (also including metastasis detected by immunochemistry), are classified as stage III. For distant metastasis, the most determinant factors for prognosis continue to be the site (non-visceral versus lung versus visceral) and an elevated serum lactate dehydrogenase (LDH) level. Outside the histological microstaging, no consensus has been reached on the value of additional staging examination for detecting metastatic melanoma and on their applications after a primary diagnosis or during follow-up.⁶

Today, 50–70% of melanomas are diagnosed in an early stage (stage IA), with a very low risk of recurrence and distant spread. Therefore, it is important to identify which staging investigations are indicated in the different classes of risk. A meta-anal-

ysis performed comparing the usefulness of ultrasound, CT scan, PET and PET-CT in the staging of patients with melanoma concluded that ultrasound is superior to the other methods in detecting metastasis to the lymph nodes, while PET-CT is superior in detecting distant metastases.⁷ It is difficult to draw clear guidelines for examinations that should be used for the staging of patients with melanoma. A general indication is to modulate the examinations in relationship with the risk and/or the presence of metastases. No examination staging should be performed in patients with melanoma *in situ*. For asymptomatic patients with T1 melanomas, clinical examination alone seems adequate for staging and follow-up. Careful skin and lymph node examination excludes other concomitant lesions and is considered sufficient for skin and lymph node metastasis. However, even in this lower risk group, ultrasonography of the drainage basin (or basins) of the primary melanoma is advocated by some for its simplicity, high efficiency, low cost and good tolerability, especially when the examination is performed by an experienced operator. In case of suspicious lymph node on ultrasound investigation, fine needle biopsy, combined with cytology, allows diagnostic confirmation in the vast majority of cases. Very controversial is the role of chest X-ray and abdominal ultrasound in the presence of intermediate risk (T2–T4a), while a certain consensus seems to emerge on the indication for total body CT scan or PET-CT in melanomas at high risk for distant metastasis (T4, ulcerated, mitotic index >1) and/or in presence of lymph node metastases. Finally, among blood tests, an indication for dosage of LDH in patients with distant metastases (or high risk) is widely shared, as LDH levels are able to stratify risk of death in these patients.

Table 9.3 Anatomic stage for cutaneous melanoma

Clinical staging ^a				Pathologic staging			
	T	N	M		T	N	M
0	Tis	NO	M0	0	Tis	NO	M0
IA	T1a	NO	M0	IA	T1a	NO	M0
IB	T1b	NO	M0	IB	T1b	NO	M0
	T2a	NO	M0		T2a	NO	M0
IIA	T2b	NO	M0	IIA	T2b	NO	M0
	T3a	NO	M0		T3a	NO	M0
IIB	T3b	NO	M0	IIB	T3b	NO	M0
	T4a	NO	M0		T4a	NO	M0
IIC	T4b	NO	M0	IIC	T4b	NO	M0
III	Any T	N>NO	M0	IIIA	T1–4a	N1a	M0
					T1–4a	N2a	M0
					T1–4b	N1a	M0
				IIIB	T1–4b	N2a	M0
					T1–4a	N1b	M0
					T1–4a	N2b	M0
					T1–4a	N2c	M0
					T1–4b	N1b	M0
					T1–4b	N2b	M0
					T1–4b	N2c	M0
				IIIC	Any T	N3	M0
					Any T	Any N	M1
					Any T	Any N	M1
IV	Any T	Any N	M1	IV	Any T	Any N	M1

^aClinical staging includes microstaging of the primary melanoma and clinical/radiologic evaluation for metastases. By convention, it should be used after complete excision of the primary melanoma with clinical assessment for regional and distant metastases.

Pathologic staging includes microstaging of the primary melanoma and pathologic information about the regional lymph nodes after partial (i.e. sentinel node biopsy) or complete lymphadenectomy. Pathologic stage 0 or stage IA patients are the exception; they do not require pathologic evaluation of their lymph nodes.

Wide excision

Wide excision represents the standard treatment for primary melanoma and has the primary goal of achieving durable local control of disease, minimizing the risk of local recurrence and curing patients with clinically localized melanoma. Wide excision involves removal of a surrounding margin of normal skin, *en bloc* with subcutaneous tissue down to the muscular fascia, which should be excised only if clearly infiltrated. It is a simple surgical procedure, performed in most cases under local anaesthesia, on an outpatient basis and less frequently under general anaesthesia, especially when complex reconstruction techniques are required or a concomitant sentinel lymph node biopsy or lymphadenectomy is planned.

The most important oncological principle is to preoperatively define the healthy margin that must be obtained for adequate surgical resection. The free excision margin should be measured radially from the edge of the lesion or the biopsy scar and should be adjusted according to the thickness of the primary tumour at histological examination. The width of excision margins has been investigated in several randomized controlled trials. Some meta-analysis show no differences in overall survival between

Table 9.4 Recommended excision margin according to melanoma thickness

Thickness	Excision margins
<i>In situ</i> melanoma	5 mm
Breslow ≤ 2 mm	10 mm
Breslow > 2 mm	20 mm

large excisions (3–5 cm) and narrow excisions (1–2 cm), whereas they seem to highlight significant differences in disease-free survival.⁸ On the basis of currently available data, the appropriate excision margins should be tailored on the thickness of primary melanoma as reported in Table 9.4.

Although melanoma *in situ* is considered a non-infiltrating lesion, in some cases a local recurrence can occur; in rare cases, it occurs as an infiltrative lesion with metastatic potential. A margin of 0.5 cm ensures excellent local control. In a particular form of melanoma *in situ*, lentigo maligna type, the lesion appears more extensive microscopically than upon clinical examination. For this reason, a 1 cm margin is advisable in the presence of lentigo maligna, due to the possibility of finding residual foci in the final pathologic assessment when conventional excision is adopted. Despite a close observation of these guidelines, some melanomas *in situ* require multiple excisions to achieve a safe margin, and occasionally split-skin grafts or flaps are necessary for wound closure.

The surgical specimen, including skin and subcutaneous tissue, should always be sent for final histological analysis to detect residual tumour or satellites nodules. For this analysis, it is advisable to send an orientated specimen, which facilitates re-excision if margins are involved or are very close to the tumour. Primary closure can be obtained in the vast majority of cases, sometime after mild mobilization of skin edge from the underlying fascia. Skin grafts or tissue flaps may be necessary for melanomas located in less mobilizable anatomical areas, such as the distal extremities and the scalp. In specific areas (nose, ears, eyelids), extensive demolition by removing portions of the cartilage may be necessary if clearly infiltrated. In these areas, if surgery involves disabling outcomes from an aesthetic and functional point of view, the width of excision can be modulated based on the risk of recurrence. The reconstructive technique should be properly planned and all the cosmetic and functional factors should be discussed with the patient. In these situations, some advocate Mohs micrographic surgery (MMS). MMS is a technique that involves sequential margin excision of skin around the tumour with immediate intraoperative histological margin assessment. MMS provides more a limited margin of excision and is performed, with favourable outcome, mostly in squamous cell and basal cell carcinoma. For melanoma, the presence of potential satellite microscopic disease discourages the use of MMS in infiltrating melanomas. A lower recurrence rate after use of MMS has been reported for melanoma *in situ* but, at this level of evidence, the technique should be performed in selected cases, preferably in the context of clinical trials.

For melanoma of the nail bed and the distal phalanx, recommendations for treatment are not evidence based. In general, for subungual or distal phalanx melanomas ≤ 2 mm thick, generally a 1 cm gross margin from the tumour can be obtained with amputation at the level of the distal phalanx. For more infiltrating melanomas, amputation is indicated at the level of the metatarsal/metacarpal phalanx. The function of the finger involved (first/second finger) can suggest more limited resection and all the pros and cons of radical surgery must be discussed in detail with the patient.

Sentinel lymph node biopsy

The rationale for sentinel lymph node biopsy (SLNB) is based on the hypothesis that the lymphatic spread of metastatic cells from the primary melanoma to the regional lymph nodes is an orderly and definable process. Accordingly, the first lymph node(s) that receives lymphatic drainage from the affected area is considered the sentinel lymph node (SLN), because it is the most likely to contain metastatic disease in that specific lymphatic basin. It follows that successful identification, surgical removal, and careful histologic examination of all SLNs in a patient should provide a more accurate regional nodal staging. A significant percentage (15–20%) of patients with a diagnosis of primary melanoma harbour undetectable metastasis (either clinically or with ultrasonography) in the regional lymph nodes. Micrometastases, if left untreated, will become clinically evident with time, thus motivating the need to discover this occult disease at an early stage. The impact on survival of SLNB in melanomas remains unknown, however. The Multicenter Selective Lymphadenectomy (MSLT-I) trial randomized 1347 patients with melanomas 1.2–3.5 mm thick to undergo wide excision alone or wide excision with SLNB. The results of this study showed that the 5-year melanoma-specific survival rates were not significantly different for the two arms (87.1% versus 86.6%); however, a small but significant 5-year disease-free survival rate was detected in the SLNB group.⁹

The incidence of SLN metastases directly correlates with tumour thickness, ulceration and a variety of other known primary tumour factors (lymphatic invasion, mitotic rate, Clark level, anatomic site and patient age). Substantial agreement exists for considering SLNB the standard of care in patients with cutaneous melanoma > 1 mm in thickness.¹⁰ For thin melanomas (< 0.99 mm), the risk of lymph node metastasis is estimated to be approximately 5.1% only. Among this group, however, a risk of positive SLN up to 20% has been reported in melanomas with a thickness of 0.76–0.99 mm, associated with ulceration and/or mitotic index $> 1/\text{mm}^2$. In these patients, SLNB is therefore highly recommended. For melanoma ≤ 0.75 mm thick or less in presence of ulceration and/or mitotic index $> 1/\text{mm}^2$ and other established risk factors, the probability of detecting a positive SLN are less but not irrelevant and the indication for a SLNB should be discussed with the patient.

SLNB is a surgical procedure that involves preoperative lymphoscintigraphy, obtained through the injection of a radio-pharmaceutical (human albumin nanocolloid labelled with technetium ^{99m}Tc). The injection is intradermal, around the scar of the removed melanoma or close to the tumour if still present, and followed by early and late scintigraphic scans in the likely locations of lymphatic drainage. Once the location of the sentinel node(s) has been identified, cutaneous projection area of each single node is marked on the skin. SLNB is performed in the operating room, usually under local anaesthesia associated with sedation or under general anaesthesia according to the site of lymphatic basin. Prior to sterile preparation of the patient, the primary site is further injected intradermally with 0.5–1 mL of a vital dye (Patent Blue), to increase the sensitivity of the method and to facilitate the finding of the lymph node. The use of the vital dye for SLNB should be avoided if the primary tumour is located on the face because of the risk of permanent tattooing, and in the case of a pregnant patient because of the low but not rare risk of reported allergic reactions (about 1% of cases). If SLN is identified in the parotid or in a deep location, SLNB should be extensively discussed with the patient before surgery, because in these areas SLNB may require more extensive surgery and the patient is at a higher risk for potential complications.

The SLNB technique is very simple. Through a small skin incision, which takes into consideration the incision necessary for a subsequent radical lymphadenectomy, the district is entered and under the guidance of a radioisotope probe, tracing the blue lymphatic channels, the SLN(s) is identified and removed. Care should be taken to not disrupt or cauterize the lymph node capsule. Each SLN removed is checked *ex vivo* for radioactivity and the nodal basin is rescanned. In case of potential background or shine radiation from the primary melanoma sites, concomitant wide excision can be performed beforehand to decrease radioactivity disturbance. All lymph nodes that are coloured or that measure >10% of the radioactivity of the *ex vivo* count in the more radio-emitting sentinel should be considered sentinel nodes. Drains are infrequently required and most patients are discharged on the same day. The incidence of complications is low, mainly related to wound dehiscence/infection or lymphatic collection (seroma). The success of the procedure, which is related to a well-known learning curve, also depends on the location of melanoma and on the lymph node basin involved (95% in the axillary and inguinal regions and 85% in head and neck).

Histological examination of the SLN should be performed on thick sections cut from paraffin-embedded tissue. Intraoperative pathology evaluation is not recommended, because in frozen sections identification of single or small clusters of melanoma cells is more difficult and a potential loss of diagnostic tissue can occur during specimen sectioning. The histopathological analysis of the SLN needs multiple sections stained with haematoxylin–eosin for immunohistochemistry. Some standardized protocols define the number of histological sections, the interval between these and the type of immunohistochemical markers to use for histological examination of the SLN in melanoma, such

as those proposed by the European Organisation for Research and Treatment of Cancer (EORTC) Melanoma Group. The percentages of positive sentinel nodes are affected by the type of protocol used, with a range between 18 and 33%. Because of prognostic relevance, the histologic report should specify the number of SLNs analysed, the number of positive lymph nodes, as well as the characteristics of the lymph node metastasis, reporting the maximum diameter, site (subcapsular, parenchymal or mixed), parenchymal invasion and extracapsular extension. The presence of naevus cell clusters should be noted. In fact, the SLN is considered positive in presence of isolated tumour cells only, even if these are displayed with immunohistochemical techniques only.

Histological status of the SLN is the most powerful independent predictor of survival in clinically node-negative melanoma patients. A negative SLNB is associated with a 5-year melanoma-specific survival rate of approximately 92%. Recurrence and death may occur in this group of patients due to a false negative SLNB or a pure haematogenous pattern of metastases. A false negative SLNB occurs in up to 10% of patients and is defined as the rate of development of clinical nodal disease in the same nodal basin. The reasons for a false negative event after SLNB probably have a multifactorial origins, related to an incorrect identification of the SLN during the procedure, to the presence of in-transit microscopic disease and, finally, to the presence of a very small burden of disease undetected at histological examination.

Lymphadenectomy

Lymph nodes are the most frequent site of metastases of melanoma. The lymphatic basin (or basins) at risk depend on the location of the primary; for melanomas of the head and neck, the laterocervical lymph nodes; for those of the trunk positioned above the Sappey line (extending from the navel to the iliac crest to the second vertebra lumbar), the axillary lymph nodes; and for those of the trunk below this line and the lower limb, the inguinal lymph nodes. Even the epitrochlear and popliteal lymph nodes, although rarely, may be the site of locoregional metastases. The main purpose of lymphadenectomy is to provide a locoregional control of disease and it is indicated for patients with histo- and/or cytologically proven lymph node metastasis.

Lymph node involvement is diagnosed in different ways, most frequently after SLNB, but a quota of patients may present clinical evident metastases at diagnosis or during follow-up. In case of positive SLNB, 15–20% of patients subjected to lymphadenectomy present additional lymph node metastases in the dissected basin, confirming the value of surgery for regional control of disease. However, the role of completion lymphadenectomy after SLNB remains controversial.⁹ In an MSLT-I subgroup analysis of patients with nodal metastasis, a significant difference in 5-year overall survival was detected in patients who underwent completion lymphadenectomy versus those

who developed clinically evident nodal disease requiring delayed lymph node dissection. Moreover, less positive lymph nodes were present in the SLNB arm who underwent completion lymphadenectomy compared with those analysed after delayed lymphadenectomy in the observation group (1.4 positive nodes versus 3.3 positive nodes, $p < 0.001$).

Despite these results, the impact of radical lymphadenectomy after SLNB on prognosis still remains unknown, and the enrolment of patients with positive sentinel lymph node biopsy in the two ongoing prospective studies (MSLT-2 and MINITUB), investigating the role of completion radical lymphadenectomy after the SLNB positivity, is highly recommended. While waiting for the results of these studies, radical lymphadenectomy remains the standard of care in patients with lymph node metastases.

The general principle in performing lymphadenectomy for melanoma is the *en bloc* excision of lymph nodes with surrounding fat tissue. Every attempt should be made to minimize morbidity through preservation of the motor nerves and muscle. The relative frequency of complications after lymphadenectomy has led some groups to consider more limited interventions, particularly in patients with positive SLNs in locations at risk of more significant complications. Axillary radical lymphadenectomy should include complete dissection of level I, II and III lymph nodes, removing all fibro-fatty tissue around the axillary vein. A section of the pectoralis minor muscle is suggested by some for better access to level II–III lymph nodes or in presence of bulky level II and III nodes. During dissection, long thoracic and thoracodorsal nerves should be identified and sectioned only if directly involved by the tumour.

In the case of metastasis to the inguinal lymph nodes, the standard approach involves the removal of the inguinal, external iliac and obturator lymph nodes.¹¹ In the classic description of inguinal dissection, a longitudinal skin incision is employed, extending 2–3 cm supero-medial to the superior anterior iliac spine up to the apex of the femoral triangle. The skin flaps are created medially up to the pubic tubercle, the anterior margin of the gracilis and abductor muscles and laterally up to the superior anterior iliac spine and Sartorius muscle. The dissection continues deeply, through the incision of the fascia lata, over the underlining muscles and femoral vessels. The saphenous vein is sectioned at the apex of the femoral triangle and at the level of the saphenofemoral junction. Deep dissection (iliac and obturator) involves sectioning of the oblique muscles and the inguinal ligament and through an extra-peritoneal approach, access to the pelvic area of the iliac and obturator lymph nodes is achieved. Even if the use of inguinal lymphadenectomy is performed by some surgeons in selected cases (negative pelvic CT scan, negative lymph node of Cloquet, number of positive inguinal lymph nodes are ≤ 2 , absence of a lymphatic drainage deep the pelvis view lymphoscintigraphy), evidence is too low to recommend this approach and a proper designed trial on this topic is desirable.

For cervical lymph node metastasis, clear indications on the thoroughness of dissection are lacking and all the recommenda-

tions are supported by a low level of evidence and follow opinions of experts in this field. In case of clinically evident cervical lymph node metastasis, surgery is aimed at the removal of all five levels of lymph nodes (submandibular, jugular and supra-clavicular). In case of metastasis discovered after sentinel lymph node biopsy, selective lymphadenectomy, involving the removal of lymph nodes at different levels, depending on the site of the primary tumour, the site of the sentinel node and the lymphatic drainage highlighted at lymphoscintigraphy, is suggested. Removal of the superficial part of the parotid is recommended only if clinically involved, because of the high risk of nerve damage observed.

All lymphadenectomies are characterized by significant morbidity, which is variable depending on the site of dissection. Inguinal–iliac–obturator lymphadenectomy is the intervention burdened by the greatest number of complications by far (wound infection/dehiscence, lymphocele, lymphoedema and nerve damage) with an incidence that can reach up to 80%.¹² Aimed at reducing postoperative morbidity after inguinal lymphadenectomy, several variations have been proposed which consider skin-sparing techniques, sartorius muscle transposition and saphenous vein preservation. The benefits of these technical variants still remain uncertain, as no randomized trials have reported a clear reduction in postoperative morbidity. An interesting advancement to minimize surgical morbidity after inguinal dissection is videoendoscopic inguinal lymphadenectomy¹³ (Figure 9.1). After blunt dissection under the fascia of Camper, the working space is obtained using high-pressure CO₂ levels and three trocars are placed at a variable distance from the apex of the femoral triangle. The postoperative morbidity appears lower with respect to the open procedure, mainly for wound/skin-related complications, and the number of lymph nodes harvested seems comparable to that of conventional groin dissection. Nevertheless, before this technique is considered suitable for clinical practice, comparable oncological outcomes, lower postoperative morbidity and better quality of life should be assessed in an appropriately designed randomized controlled trial.

In patients who undergo lymphadenectomy, the number of positive lymph nodes plays an important prognostic value. If only one lymph node is metastatic, the 10-year survival rate is about 50%, dropping to 30% if 2–3 positive lymph nodes are found.¹⁴ The 'N'-ratio (i.e. the ratio between the number of positive lymph nodes and total lymph nodes removed) seems to have a completely independent prognostic impact. Patients with a low N-ratio (less than 0.1 or including between 0.11 and 0.25) have a significantly better prognosis than patients with a high N-ratio (>0.25).¹⁵

For the prognostic importance of the number of lymph nodes and the N-ratio, the extension and quality of surgery becomes a primary factor in a patient's prognosis. Despite this evidence, shared parameters about quality control of lymph node dissections for melanoma are still lacking. The recommendations of the National Comprehensive Cancer Network (NCCN) require

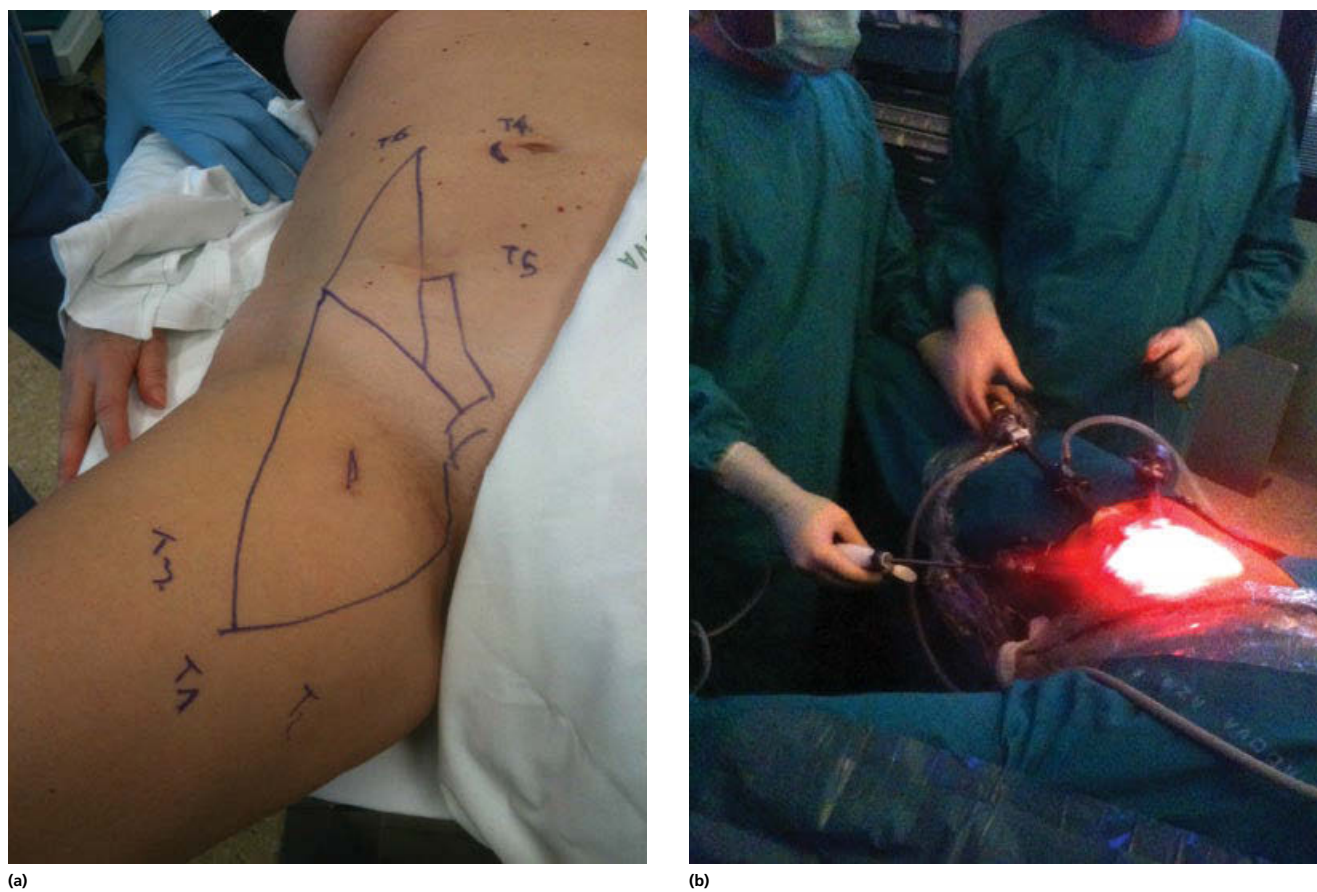


Figure 9.1 Videoscopic groin dissection for groin metastases. (a) Trocar positions; (b) Intraoperative view.

only a clear description of the anatomical limits of the dissection. The number of lymph nodes (with different limit values) and the *N*-ratio could be a simple and objective parameters of quality, but no consensus on their use in clinical practice has been found.¹⁶

Adjuvant therapy

Patients with lymph node metastasis are at high risk of recurrence and/or systemic metastases. For this reason, adjuvant (i.e. postoperative) therapy has been proposed to reduce the risk of death and recurrence in patients with high-risk melanomas. The only compound that has shown some positive therapeutic effects is interferon alpha.¹⁷ Several randomized studies, confirmed by a meta-analysis, showed a 13% reduction in the 5-year recurrence rate and an 11% reduction on the risk of death in patients treated with interferon. Although further studies are required to select patients who are most likely to benefit from this treatment, data on interferon treatment represent today the best referral values in investigating new therapeutic agents for adjuvant treatment. Adjuvant chemotherapy and immunotherapy with immunostimulatory agents (bacille Calmette Guérin/BCG), cytokines (interferon gamma, interleukin 2) and melanoma-specific vaccines have not demonstrated any therapeutic advantage. Among

vaccines, the Mage 3A vaccination seems the most promising but the results of the trial concluded in 2011 are still awaited. The exciting survival benefits seen in patients with metastatic melanoma treated with immune-targeted and target drugs (Ipilimumab and BRAF inhibitors) have suggested its use in the adjuvant setting but their potential benefit is still under investigation.

The use of adjuvant radiotherapy after lymphadenectomy for melanoma remains controversial and no significant improvement of the overall survival has been observed. For patients at high risk for regional relapse (multiple affected lymph nodes or extracapsular spread), adjuvant radiotherapy after lymph node dissection seems to improve local control and should be considered in this group of patients.¹⁸

Locoregional treatment

'In-transit metastases' or 'satellites' are defined as the locoregional spread in the lymphatic system between the site of the primary lesion and the main regional lymphatic basin. More than 20% of all recurrences for melanoma are in the form of in-transit metastases or satellites. The 5-year survival rate of these patients stands at 25–30%, with a better prognosis than other metastatic sites.



Figure 9.2 Hyperthermic isolated limb perfusion (HILP) for bulky metastatic melanoma of the leg. (a) Before and (b) response after treatment.

In the presence of cutaneous and subcutaneous metastases, the therapeutic choices depend on the number, size and location of the lesions. For a single or very few lesions, especially if the primary melanoma is associated with favourable prognostic factors, a simple radical excision represents the first-line treatment, possibly associated with SLNB, since the occurrence of occult metastases in regional lymph nodes is significant. Excluding these more favourable cases, the vast majority of patients present with multifocal and progressive lesions or bulky disease. In these cases, locoregional treatment should be proposed in absence of systemic metastases. For in-transit lesions located in the limbs, hyperthermic isolated limb perfusion (HILP) represents the gold standard of treatment¹⁹ (Figure 9.2). The rationale of HILP is the administration of a high dose of one or more chemotherapeutic drugs to the limb while avoiding systemic toxicity. During HILP, the femoral/axillary vessels are cannulated and the perfusion circuit is obtained through a specifically designed perfusion machine, which maintains an adequate blood flow under moderately hyperthermic conditions (39–41°C). Melphalan is the drug of choice, with a complete response in around 50% of cases and a 5-year survival rate of 28.5%. In cases of no response, relapse or in the presence of a high tumour burden, the association of melphalan with tumour necrosis factor alpha (TNF- α) provides a significant improvement in the complete response rate without an increase in toxicity. In patients with recurrence after HILP unfit for surgery, isolated limb infusion (ILI) can be used.²⁰ With respect to HILP, isolated limb perfusion is performed in absence of hyperthermia under hypoxic conditions (without oxygenation

of the perfusate) and is considered less invasive since the catheters are placed percutaneously. The combination of melphalan and actinomycin-D achieves a complete response rate in 38% and a partial response rate in 46% of cases, with a 5-year survival rate similar to that for HILP.

Recent evidence has suggested electrochemotherapy as a new therapeutic modality for patients with in-transit metastases from melanoma (Figure 9.3). Electrochemotherapy is based on the physical and biological principle of electroporation (i.e. the ability of an external electric field applied on biological tissues to increase, by using a short and intense pulse, the permeability of cell membranes to various molecules). The use of electrochemotherapy in the presence of cutaneous and subcutaneous metastases has proved effective and the technique has recently been standardized. The European Standard Operating Procedures of Electrochemiotherapy (ESOPE) classified the use of electrochemotherapy according to the type of tumour, the drug used (bleomycin or cisplatin), the route of administration (venous or intratumoural) and the type of electrode used.²¹ The main advantage is that electrochemotherapy is a minimally invasive procedure well accepted by patients. It is performed in a day hospital setting under local anaesthesia and/or sedation, with limited side effects and very few contraindications.

Electrochemotherapy has the considerable advantage, with respect to HILP and ILI, that it is easily repeatable in different sessions and can be applied to any region of the body. The complete response rate after a single session ranges from 9% to 100%, while the objective response varies from 62% to 100%, results that tend to increase after multiple sessions. Main prognostic factors identified as related to the tumour response are the nodule size and number.

In conclusion, in the presence of in-transit metastases, HILP remains the treatment of choice, as it ensures the highest rate of complete response, which is a factor that is able to influence both locoregional control and survival. ILI is a viable alternative, especially in frail patients with multiple comorbidities, for sites of disease distal to the middle third of the thigh. Electrochemotherapy is a promising technique that can be used in case of failure of HILP and isolated limb perfusion, as complementary treatment in combination with other locoregional therapies, or as an alternative where these are contraindicated or cannot be accomplished (metastases in the trunk, neck and head).

Distant metastases

The most common sites of metastases from cutaneous melanoma are extraregional lymph nodes, the lung, the brain, the skin and the liver, less frequently, bone, adrenal glands and gastrointestinal tract. In the presence of distant metastases, surgical treatment may have a dual purpose – palliative or curative. In the first case, surgery is mainly directed to treat a symptom that



Figure 9.3 Electrochemotherapy treatment for superficial metastases. (a) Hexagonal-array needle electrode for tumour electroporation; (b) cutaneous metastatic melanoma, baseline; (c) follow-up at 44 weeks; (d) Follow-up at 60 weeks.

adversely affects the quality of life of a patient. For example, in selected patients with metastases to the gastrointestinal tract, surgery may offer good palliation for bleeding or intestinal occlusion. On the other hand, radical resection should be considered as first-line therapy whenever complete removal of all disease can be accomplished, since surgery with curative intent ensures 5-year survival of up to 30%, a result not obtainable with other systemic therapies.²² For this reason, radical surgery should be considered in selected patients after multidisciplinary discussion, taking into consideration, as prognostic factors, the metastatic site (skin versus lung versus other sites), the number of metastases, the number of organs involved, the disease-free interval, the doubling time of the lesions, the LDH levels and the performance status of the patient.

However, for the majority of patients with stage IV disease, radical surgery remains contraindicated for the presence of

unresectable, progressive or disseminated disease.²² These patients should be considered for systemic therapy or palliative care. Until a few years ago, patients with metastatic melanoma had few treatment options. They had been treated with dacarbazine or other palliative chemotherapies, with poor results (median survival 6.2 months and only 25.5% of 1-year survival). Over the last decade, significant progress in understanding the molecular pathway of tumourigenesis and metastasis of melanomas, together with the definition of the regulatory role of the immune system in these mechanisms, led to the discovery of new drugs that have shown an encouraging clinical activity against metastatic melanoma. Among these, ipilimumab is a monoclonal antibody that blocks the cytotoxic T-lymphocyte-associated antigen (CTLA-4), a molecule that has an inhibitory role in T cell activation. Treatment with ipilimumab promotes antitumour immunity against

melanoma, showing significant improvement in survival for patients with metastatic disease, with a reported median survival of 10 months.²³ Another recent finding, closely linked to molecular biology of melanoma cells, was the discovery of the role of protein kinase pathway in tumour progression, which is regulated by the protein BRAF. Mutations in BRAF protein (mainly at codon 600) are found in half of patients with melanoma, resulting in constitutive activation of the kinase pathway. Almost 90% of BRAF mutations are in the codon V600E, which is specifically inhibited by the monoclonal antibody vemurafenib. In the clinical setting, vemurafenib has been shown to improve tumour response rate and overall survival.²⁴ Despite the continuous improvement in target therapy for metastatic melanoma and new promising drugs under study, the response rate for ipilimumab is low and the durability of clinical response of vemurafenib remains poor. For these reasons, further trials evaluating combination therapies are essential to improve the results of treatment of stage IV melanoma in the near future.

Non-cutaneous melanomas

After cutaneous melanoma, ocular melanoma represents the second most common type and the most common primary intraocular malignant tumour in adults. The vast majority of ocular melanomas originate from uvea, whereas conjunctival melanomas are far less frequent. Incidence of uveal melanoma has remained stable over the last three decades. Patients with uveal melanoma are often asymptomatic and the tumour is diagnosed upon clinical examination with great accuracy. Fine needle biopsy is usually not suggested, as seeding of the needle tract has been reported. The choice of treatment takes into account many factors; among them, the size of the tumour is the most important. Historically, enucleation has been considered the definitive treatment of uveal melanoma, now restricted for large or symptomatic lesions. The alternative treatment most commonly adopted is radiotherapy. Among the radiotherapeutic strategies, the most common treatment is plaque brachytherapy, in which a small plaque with radioactive seeds is attached to the outside surface of the eye, overlying the tumour.²⁵ The plaque is left in place for a few days and then removed. After plaque radiotherapy, the survival results are the same as that after enucleation. Despite an optimal local control, half of patients develop metastases within 15 years after treatment of the primary tumour. The most common site of metastasis for uveal melanoma is the liver, which is also the first site of metastasis for 80–90% of ocular melanoma patients. Other common sites of metastasis include the lung, bones and the skin. The survival time after diagnosis of liver metastases depends on the extent of systemic spread. Recent findings in cytogenetics and genetics have allowed a better prognostic stratification and the determination of tumours with higher metastatic potential.²⁶ However, an effective systemic therapy is still lacking.

Primary mucosal melanomas arise from proliferation of melanocytes located in mucosal membranes of the gastrointestinal, respiratory and urogenital tract.²⁷ More than half of mucosal melanomas originate from the mucosa of the nasal and oral cavity and the accessory sinuses. Less frequently, the sites of origin of mucosal melanomas are ano-rectum, vulva and vagina. Mucosal melanomas can arise in almost any part of mucosal membranes, with a reported 1% of cases of occult disease. The lack of early and specific symptoms contributes to late diagnosis, and consequently to adverse prognosis. Because of their low incidence, no established protocols for treatment are available, and surgery represents the mainstay of therapy. More recently, more conservative surgery has been advocated, since a demolitive approach does not seem to show an advantage for survival. Adjuvant radiotherapy can provide better locoregional control in some locations, but did not show improvement in survival. Compared with cutaneous and ocular melanoma, mucosal melanomas have the lowest percent of 5-year survival. There is no effective systemic therapy for these aggressive tumours. Recent advances in molecular biology underlying mucosal melanomas have identified KIT as a potential marker for targeted therapy in around 25% of patients.²⁸ Clinical trials with KIT inhibitors are ongoing.

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CHAPTER 10

Non-melanoma skin cancers

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We live in an age of increased awareness of the linkages between genetics, skin type, ultraviolet light exposure and the development of skin cancer. Among the general public, melanoma receives the bulk of attention in this area as a tumour (of melanocytes) that demonstrates early metastasis relative to its tumour burden. However, melanoma represents less than 2–5% of all cutaneous malignancies. The other 95–98% are the non-melanoma skin cancers and, as a group, these are the most common form of cancer in humans. The extensive incidence of the non-melanoma skin cancers (NMSC) within humans is often underrepresented in cancer rankings. NMSC is usually excluded from these rankings by mention in the fine print of cancer fact sheets. Recent estimates demonstrate that more cases of NMSC have occurred in the past 30 years than all other forms of cancer combined.¹

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are the most common forms and account for the vast majority of NMSCs. Despite growing public awareness campaigns on the harmful effects of ultraviolet (UV) exposure, the incidence of NMSC has been rising by 3–8% per year since 1960, with as much as 300% increase in the past two decades in countries such as the United States.² This trend is predicted to continue as populations age worldwide. Despite this increase, the death rate from NMSC is decreasing due to increased awareness, earlier diagnosis and improved treatment options. A thorough understanding of the NMSCs and current management options is essential to continue this important survival trend.

Basal cell carcinoma

Basal cell carcinoma is a malignant neoplasm of keratinocytes that is exceptionally common (>80% of skin cancers) and rarely metastatic. In the United States it affects 1 million people each year. BCCs may be either sporadic or related to heritable mutation(s). Most BCCs are sporadic, with environmental exposures being the major aetiological factors, with a latency period of anywhere between 20 and 50 years from exposure to

the development of BCCs. For this reason, most sporadic BCCs occur in older males (>50 years) with fair complexions (Fitzpatrick skin types 1 and 2) as this group has the greatest lifetime exposures to environmental influences.

Environmental influences

Intensive, intermittent UV exposure (particularly UVB rays) resulting in sunburn and blistering (especially during childhood/adolescence) is a major risk factor for BCC. Fair skin types (Fitzpatrick skin types 1 and 2) and individuals in hot sunny climates are at greater risk of incidental intermittent sunburn. In Australia, which experiences the greatest UV solar irradiation per area of any continent, these factors combine to produce the highest incidences of NMSCs and melanoma in the world. Additional environmental factors include tanning bed use, chronic inflammation (burns, scars), ionizing radiation, arsenic and hydrocarbon exposure. The association between human papillomavirus and BCC is not yet clear, however immunosuppression poses greater risk of BCC development, particularly among organ transplant recipients using photosensitizing antifungal prophylaxis such as voriconazole.

Genetic basis

Multiple types of genes have been implicated in the development of BCC, including tumour suppressor genes, DNA repair genes and genes involved in skin and skin appendage pigmentation. Approximately 90% of sporadic BCCs have activating mutations in the hedgehog signalling pathway, resulting in uncontrolled proliferation of epidermal basal cells. This pathway was identified by studying patients with basal cell naevus syndrome (BCNS or Gorlin syndrome), an autosomal dominant disease with constitutive mutations of the hedgehog pathway. The specific gene involved in the majority of cases is the tumour suppressor gene *PATCHED* (chromosome 9q), mutations of which lead to an increased risk of developing multiple BCCs from a young age, as well as a cluster of keratocystic odontogenic tumours (KCOTs; 75% of patients), palmar and plantar pits (Figure 10.1), multiple skeletal abnormalities (e.g. bifid



Figure 10.1 Palmar pits in Gorlin syndrome. The patient's mother and brother were also both affected, with greater penetrance of phenotypic changes, but all three have palmar pitting.

ribs, frontal bossing), and calcification of the falx cerebri. Approximately 10% of BCNS patients do not develop BCCs, indicating the involvement of multiple genes. The tumour suppressor gene P53 and melanocortin-1 receptor gene are also important for the development of BCC, but much less specific to this tumour. Those with pigment disorders and defects in DNA-repair mechanisms, such as albinism and xeroderma pigmentosum, respectively, are also more susceptible to NMSC development.

Clinical presentation and classification

BCCs usually present on sun-exposed areas, such as the head and neck, although they can occur at more photo-protected sites, particularly in the immunosuppressed. Due to its prevalence, many different classification schemes have been developed for BCC. A clinicopathologic classification is the most common in clinical use, but suffers from significant overlap between specific types (e.g. between desmoplastic/morpheaform and infiltrating) and multiple names used for the same or similar subtypes. Commonly described subtypes include: superficial, nodular, micronodular, morpheaform/desmoplastic, infiltrating, pigmented and mixed. A simpler, management-oriented approach is to classify BCCs as either discrete or diffuse, referring directly to how easy it is to judge their margins. This approach groups poorly circumscribed lesions (e.g. micronodular, morpheaform, infiltrative) on the basis that wider margins of excision are necessary to achieve clearance when compared with discrete lesions (such as superficial and nodular types). The anatomic location of BCCs may also favour the development of a particular subtype.

Superficial basal cell carcinoma

Superficial BCCs (Figure 10.2a,b) typically appear on the trunk as single or multiple well-defined erythematous finely scaled patches or plaques with pearly edges. They can be confused with

actinic keratosis, superficial SCC or eczema. Any solitary patch of 'eczema' not responding to treatment should raise suspicion for superficial BCC. Numerous superficial BCCs have a well-known association with a history of arsenic exposure.

Nodular basal cell carcinoma

Nodular BCC (Figure 10.2c,d), the most common subtype (50–85% of tumours) typically presents as a pearly papule or nodule with raised, rolled edges and telangiectasia. These features are particularly obvious when the lesion is put under tension (to demonstrate pearly) and then released (to show refilling of telangiectasia). Ulceration may sometimes be present, hence the term 'rodent ulcer' and if left untreated local invasion can occur.

Micronodular basal cell carcinoma

Micronodular BCC (Figure 10.3a) appears as numerous very small tumour nests upon histopathology sectioning. The nodules do not appear to be contiguous with each other on individual sections, but sequential sections usually demonstrate that the individual nests are part of a complex network of intercommunicating tumour processes. The apparent lack of continuity between individual nests on single sections makes it difficult to comment on the adequacy of tumour clearance without a wide, uninvolved radial margin. This contributes to its higher recurrence rate when compared with superficial and nodular BCCs. It is considered to be of intermediate aggressiveness, with the other diffuse forms of BCC (e.g. morpheaform) demonstrating a worse prognosis again. Clinically, it can present as flat or elevated tumours that are most commonly found on the back.

Morpheaform or desmoplastic basal cell carcinoma

Morpheaform or desmoplastic BCCs (Figure 10.3b) appear as depressed, scar-like, ill-defined, indurated plaques often with focal penetration of tissues. Direct perineural invasion can occur, producing paraesthesia, hyperaesthesia or paralysis in the affected area, but due to the absence of haematogenous spread with these tumours, skip lesions are not found. These subtypes are associated with a worse prognosis and have an increased risk of recurrence following treatment.

Infiltrative basal cell carcinoma

Infiltrative BCC (Figure 10.3c) is similar to morpheaform/desmoplastic because clinically they are diffuse, can look like a skin reaction or scar and histologically, they demonstrate nests and cords of tumour invading the surrounding tissues. Unlike morpheaform BCCs, there is minimal to absent desmoplastic stromal response (the surrounding tissues can look relatively bland).

Pigmented basal cell carcinoma

Pigmented BCCs (Figure 10.3d,e) present as pigmented variants of the other subtypes, including flat patches, plaques, papules or nodules and are often difficult to differentiate from seborrhoeic

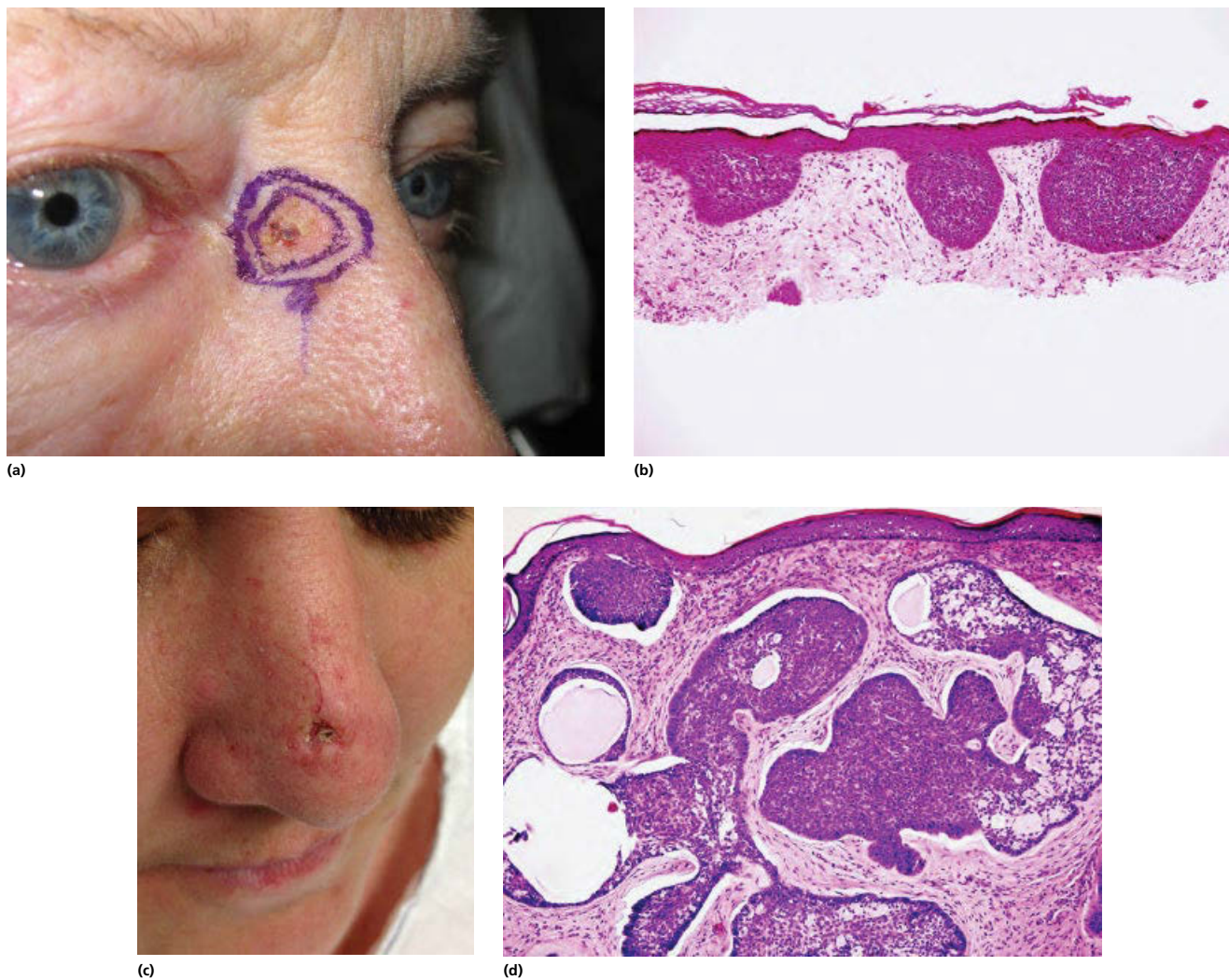


Figure 10.2 (a) Superficial BCC near the right inner canthus. (b) Representative histopathology specimen for superficial BCC – small islets of basaloid tumour cells with peripheral palisading. Nests are well circumscribed and have effaced the dermo-epidermal junction without invading the papillary dermis. (c) An ulcerated nodular BCC on the nose, demonstrating classic pearly and telangiectasia around an area of central ulceration. (d) Representative histopathology specimen for nodular BCC demonstrating islands of basaloid cells with peripheral palisading and peri-nest clefting from the surrounding specialized stroma (an artefact of processing). Note the relative basophilia of the tumour compared to the surrounding stroma.

keratosis and malignant melanoma. Pigmented lesions presenting in the normal anatomical distribution of BCC are more likely to be BCC than melanoma, but both should always be entertained in the differential diagnosis of pigmented lesions.

Mixed basal cell carcinoma

It is common to see patterns of superficial BCC mixed with micronodular or other variants. The margin of excision should be determined based on the required margin for each part and its proximity to the radial and deep margins.

Basisquamous cell carcinoma

The basisquamous cell carcinoma is an uncommon and unusual tumour that shares features of both BCC and SCC. Sometimes they are alternatively reported as a basaloid tumour with squamous features. They are to be distinguished from BCC by

their potential for malignancy and therefore clinically, should be managed as though they are an SCC.

Diagnosis

Dermoscopy

Dermoscopy aids with evaluation of pigmented cutaneous lesions and can be used to help differentiate pigmented BCC from melanoma. Typical findings of BCC on dermoscopy include the absence of a pigment network and at least one of the following: ulceration, multiple blue-grey globules, leaf-like areas, arborizing vessels, large blue-grey ovoid nests and spoke wheel areas.

Biopsy

Definitive diagnosis is made on histology following a punch, shave or excisional biopsy of the lesion. Histology of superficial BCC (Figure 10.2b) usually reveals a basophilic tumour (blue)

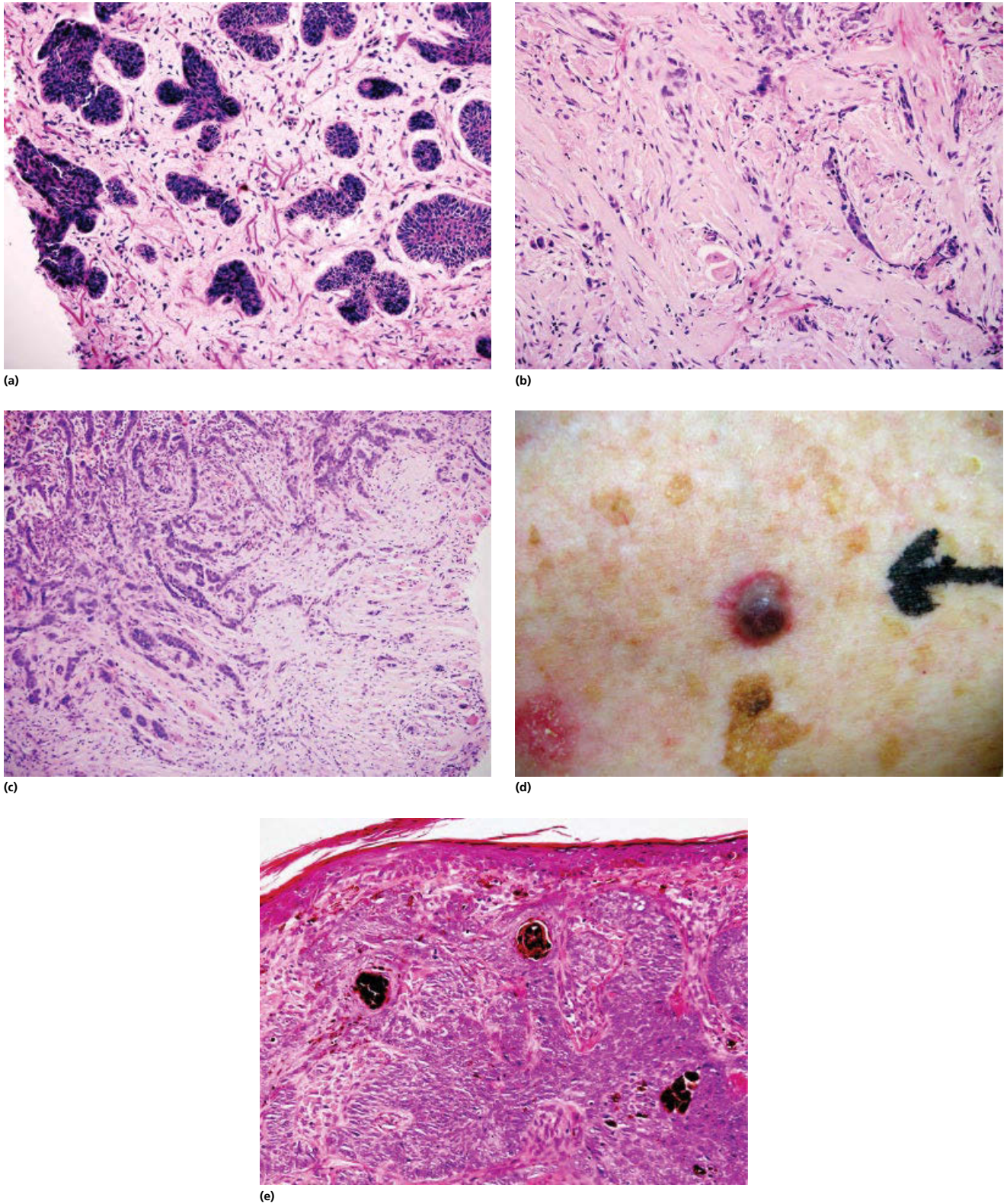


Figure 10.3 Representative histopathology specimens. (a) Micronodular BCC – uniformly small nests of neoplastic basal cells with rounded peripheral contours extending widely throughout the dermis with surrounding specialized stroma. (b) Morpheaform BCC – strands of basaloid tumour cells extending deeply into the dermis embedded with a dense fibrous stroma. (c) Infiltrative BCC – long, thin strands of basaloid tumour cells penetrate deeply among the collagen fascicles. (d) A 4 mm pigmented BCC in the central field could easily be diagnosed as a melanoma. An incidental superficial BCC is seen in the lower left corner. (e) Pigmented BCC – numerous melanocytes are present among the basaloid tumour nests and there are occasional melanophages within the stroma.

on standard haematoxylin and eosin (H&E) staining. Buds of basaloid cells extend from the epidermal basal layer into the reticular dermis. Nodular BCCs (Figure 10.2d) have nests of large basaloid cells with peripheral palisading in the papillary or reticular dermis. They may demonstrate peri-nest clefting consistent with retraction from surrounding stroma but this is usually a processing artefact. Micronodular BCC (Figure 10.3a) is composed of multiple microscopic nodules smaller than 15 μm , making it difficult to interpret tumour clearance when there are close margins. Tumours become infiltrative (Figure 10.3c) when basaloid islands lose their epidermal connections and invade the underlying dermis, often as sheets or finger-like projections of tumour cells without much stromal reaction. In contrast, morpheaform BCC (Figure 10.3b) consists of strands of tumour cells extending deeply into the dermis, embedded within a dense fibrous stroma. Pigmented BCCs (Figure 10.3e) have similar histology to the nodular subtype with numerous melanocytes among the tumour nests and melanophages in the stroma.

Staging for basal cell carcinoma

BCC and SCC share a common staging system. This is presented later in this chapter (see Squamous cell carcinoma).

Course and prognosis

BCCs grow slowly (doubling time 6–12 months) and the majority present at stage 0 or 1. Individuals presenting with one BCC are at increased risk of having more lesions at the time of diagnosis and a 3-year cumulative risk for a second primary of 44% (10-fold increase over the general population).³ They can be locally invasive, involving subcutaneous tissue, muscle and bone (Figures 10.4 and 10.5). Micronodular, morpheaform or infiltrative BCCs are more likely to recur after treatment. Long-term follow-up and annual skin examinations are warranted in those with a history of BCC, as they are also at an increased risk of developing other skin cancers. BCCs exhibit stromal dependence where tumour cells cannot engraft elsewhere without being transplanted along with adjacent stromal cells. Correspondingly, the incidence of metastasis is reported to be less than 0.55% and is associated with larger primary tumour size, greater depth of invasion, previous irradiation and central facial position rather than tumour subtype. When metastasis occurs, lymph nodes and lungs are the most commonly involved. Since 1981, 238 cases of metastatic BCC have been reported, most of which occurred in middle-aged men with an average tumour size of 7.5 cm and a median time of 9 years between primary tumour and first sign of metastasis.⁴ Once metastasis does occur, the median survival is 10 months.

Management

Complete surgical excision with pathology confirmation remains the gold standard in the management of BCC. Their abundance in clinical practice, slow doubling time, stromal dependence and significant rates of spontaneous regression (most studies demonstrate at least some degree of regression in



Figure 10.4 Local invasiveness of BCC of the left temple. Marked central tumour necrosis, chronic discharge, ipsilateral brow ptosis and loss of forehead wrinkling consistent with locally advanced BCC destroying the frontal branch of the facial nerve and underlying temporal bone. The tumour had reached the anterior cranial fossa.

around 20% of lesions⁵) have prompted the development of numerous treatment options, each with their relative merits. However, their potential to display a mixed clinicopathologic picture, variability in aggressiveness based on type, site and patient factors and the fact that recurrent BCCs tend to demonstrate poorer prognosis, should prompt management based on the most effective modality in the individual to achieve complete removal or destruction of the tumour. As a result, treatment varies depending on anatomic location, BCC subtype, tumour size, depth of invasion and patient comorbidities (Table 10.1).

Non-operative management

Topical therapies

Topical therapies provide a minimally invasive approach to manage BCC in specific situations. The success of the therapy is determined by the ability of the topical agent to reach the entire tumour, the tumour response and the compliance of the patient.

Cryotherapy uses liquid nitrogen to destroy superficial BCCs. It requires a minimum of two freeze–thaw cycles achieving temperatures of -50°C to destroy the tumour with treatment of a margin of normal tissue to ensure destruction of subclinical

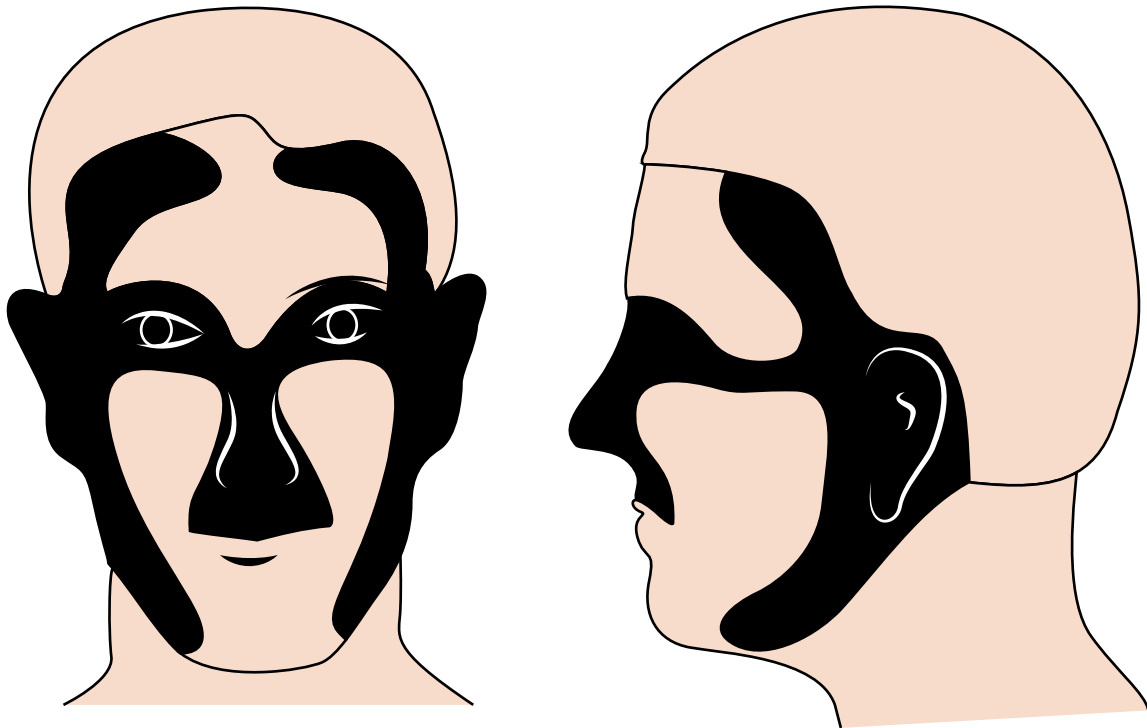


Figure 10.5 High-risk zone for basal cell carcinoma.

Table 10.1 Treatment options for basal cell carcinoma

Treatment modality	Best indication
Topical	
Cryotherapy	Small superficial BCC
Imiquimod ^a	Biopsy confirmed non-facial superficial BCCs <2 cm
5-Fluorouracil ^a	Non-facial superficial BCCs <2 cm
Oral	
Vismodegib	Locally advanced and metastatic BCC; neoadjuvant use prior to surgery to reduce surgical defect size of all subtypes
Photodynamic therapy	Multiple superficial or thin nodular BCCs
Radiotherapy	Primary BCCs in those unwilling or unable to undergo surgery; adjuvant therapy post surgery for remaining positive margins
Procedural	
Standard excision	Workhorse – surgical management of BCC, useful for all tumour types in all areas but clearest benefits in non-aggressive primary BCCs in non-high-risk locations
MMS ('Gold standard')	Selective application – primary facial morpheaform or infiltrative BCC; recurrent BCC; any BCC in a high-risk location
Electrodesiccation and curettage	Non-aggressive primary BCCs <3 cm in low-risk locations; unable or unwilling to tolerate surgery
Pulsed dye laser ^b	Superficial and nodular BCCs <1.5 cm

^a Can be tried for the off-label use of small nodular BCC.

^b Long-term follow-up outcomes still awaited.

extension. This method lacks histologic tumour confirmation but cure rates are reported to be over 95%. Care must be taken when treating BCCs near the eye. Adverse outcomes include scarring, hypo- or hyperpigmentation and the possibility of obscuring tumour recurrence by scar tissue. Some have reported greater patient satisfaction with surgical excision compared to cryotherapy due to better cosmesis.

Imiquimod 5% cream is an immune response modifier that increases the T-helper response by inducing interferon- α , tumour

necrosis factor- α and cytokines to destroy dysplastic keratinocytes by acting on toll-like receptor 7 (TLR7). Although it is only US Food and Drug Administration (FDA)-approved for biopsy confirmed non-facial superficial BCCs <2 cm in size, some have shown efficacy in the treatment of nodular BCCs. However, care should be taken with off-label treatment due to reports of recurrences with increased subclinical extension due to selective clearing of superficial tumour. Furthermore, there is significant genetic variation in toll-like receptors between individuals, with

some individuals being unable to respond to imiquimod therapy as a result. Adverse reactions include local skin irritation (the severity may or may not correlate with histological clearance) and hence it should not be applied near the eye.

5-Fluorouracil has also been used for superficial and nodular BCCs. A systematic review found clearance rates of up to 90% for superficial BCCs. However, adverse reactions of erythema, pruritus and pain limit its use. Imiquimod and 5-fluorouracil treatment should be restricted to small tumours in low-risk locations in patients who will not or cannot undergo treatment with more-established therapies of known long-term clearance rates.⁶

Oral therapy

The FDA-approved oral hedgehog pathway inhibitor vismodegib has shown efficacy in the treatment of locally advanced and metastatic BCCs,⁷ in reducing the size of existing BCCs and development of new BCCs in those with BCNS.⁸ A recent pilot study also demonstrated its potential to reduce tumour size prior to surgery for BCCs of all histologic subtypes. However, histologic changes to the tumour with this drug may cause difficulty in determining clear margins during Mohs surgery. In addition, the optimal duration of neoadjuvant treatment has not yet been determined. Adverse effects are the main limiting factor to continued use of vismodegib, including muscle cramps, hair loss, dysgeusia, weight loss and fatigue. With drug discontinuation the adverse events resolve but tumour regrowth occurs within months. Prolonged use may result in secondary acquired resistance.

Photodynamic therapy

Photodynamic therapy (PDT) involves the topical application of a photosensitizing agent on the desired treatment area, with subsequent activation of the drug by visible light to create reactive oxygen species to destroy neoplastic cells. Topical δ -aminolevulinic acid or methylaminolevulinate produce the photosensitizer protoporphyrin IX semi-selectively within tumour cells. In 2005, an international consensus on the use of PDT for NMSC recommended it as first-line treatment for superficial BCC. It is less effective in nodular BCC subtypes due to the thickness of the tumour limiting absorption of the topical medication into the dermis.⁹ Although studies have demonstrated superior cosmetic outcomes of PDT compared to surgical excision, some tumours do not respond to treatment and it is associated with higher recurrence rates than surgery with long-term cure rates for superficial BCC of approximately 75%. Adverse reactions include erythema and pain. The inconvenience of multiple hospital visits makes it unsuitable for some patients. PDT should generally be reserved for those with tumours not amenable to other established treatment methods or for those with multiple BCCs, such as BCNS patients.

Radiotherapy

Radiation therapy can be used for primary BCCs in those unwilling or unable to undergo surgery or as an adjuvant therapy post surgery for remaining positive margins. Control

rates of over 90% have been reported, although there may be potential for aggressive recurrences post treatment. Disadvantages include lack of histologic verification of tumour removal, prolonged treatment course and poor cosmetic outcomes. Radiotherapy is contraindicated in patients with BCNS or xeroderma pigmentosum (XP), as ionizing radiation can induce development of new BCCs within the radiation field.

Operative management

Current controversies in surgical management of BCC

Significant debate remains as to what is the most appropriate definitive treatment modality for BCC, especially whether standard surgical excision or Mohs micrographic surgery (MMS) is the most appropriate definitive surgical treatment. The two modalities use different processing techniques to determine margins, which can make direct comparison difficult. Compounding this in the literature is the variable use of clinical versus pathological margins, lack of direct comparison between these modalities in clinical trials and a paucity of appropriate outcome measures and suitable long-term follow-up. One example is the use of incomplete excision to suggest MMS provides better outcomes. Incomplete excision is an uncommon outcome following standard surgical excision using an appropriate margin. In specific instances, further resection may be stopped and the defect dressed or skin grafted for later reconstruction (delayed reconstruction after pathology evaluation; DRAPE¹⁰) if the formal histopathology result is clear. This minimizes the loss of important anatomical structures and has been referred to by some as 'slow Mohs'. If incomplete, further resection is undertaken, but the initial resection is considered as incomplete. MMS provides the benefit of being able to undertake multiple cycles of resection and microscopic evaluation on the same day, but the number of cycles is not generally included in statistics for comparison. Although DRAPE may be inconvenient for the patient, this approach provides excellent, cost-effective clearance while preserving important structures to aid the post-operative outcome. Therefore studies comparing modalities should be interpreted carefully as they often lack comparable definitions, intentions, terms or techniques that permit appropriate direct comparison. We will present each of these techniques and discuss their relevant merits and drawbacks in an effort to provide a balanced assessment.

Standard surgical excision

Surgical excision for BCC produces cure rates in excess of 90%. A meta-analysis (89 studies and 16 000 lesions) identified that a 3 mm clinical margin for non-morpheaform BCC resulted in a cure rate of over 95% for lesions under 2 cm in diameter. Incomplete excision in this study was associated with a 27% recurrence rate. Although this is somewhat high, incompletely excised lesions often do not show any additional tumour on re-excision and such re-excision usually results in cure in this rarely metastatic tumour. Standard surgical excision is one of the most widely used and reliable approaches to achieve definitive tumour

excision with confirmatory histopathology in a relatively cost-effective manner. However, incomplete excision rates for aggressive subtype primary BCCs or recurrent BCCs have been shown to be higher with this method compared to MMS, with over 10% incomplete excision rates in some reported studies. Although this may reflect the clinical practice of staged excision/DRAPE, proponents of Mohs correctly identify the capacity of Mohs to produce equivalent or higher cure rates in a single visit. A wider radial margin (5 mm) is typically used for diffuse lesions up to 2 cm in diameter for this reason. This wider margin may increase the defect size relative to MMS with potential significance for aesthetically or functionally sensitive areas such as in the central face. Reconstruction of these areas is commonly undertaken as aesthetic units for improved postoperative appearance, with MMS demonstrating benefit if the excision can be contained within a specific subunit. Standard surgical excision remains the workhorse for definitive BCC excision and is one of the most cost-effective means to manage the millions of BCCs that occur worldwide every year.

Mohs micrographic surgery

MMS is considered by many as the gold standard in BCC removal by providing equal or higher rates of tumour clearance when compared with other modalities. MMS involves histological analysis of tissue margins and therefore provides tumour clearance with maximal tissue conservation in areas of high risk for recurrence (periorbital, periauricular and central face), resulting in smaller surgical defects compared to standard surgical excision (Figure 10.5). Five-year recurrence rates for primary and recurrent BCCs treated by MMS are approximately 1–2% and 4–7%, respectively. Therefore it is especially useful for primary facial BCCs with aggressive clinicopathologic subtypes (morpheaform or infiltrative), recurrent facial BCCs and any BCC in a high-risk location. The use of MMS to treat the majority of BCCs is probably inappropriate however. The process can be lengthy for the patient and the Mohs practitioner as a number of cycles of excision, processing and interpretation may be necessary to achieve clearance. From a health economics perspective, MMS is significantly more expensive than standard excision, and for non-recurrent disease it has not been shown to produce better 5-year outcomes.¹¹

For these practical reasons, MMS is applied selectively in centres that offer it as a treatment modality. It is likely that formalized criteria for its use will be required into the future as populations age, rates of NMSC increase and health budgets become tighter.

Electrodessication and curettage

Electrodessication and curettage is an efficacious and cost-effective treatment modality, however, it is operator-dependent. Its use should be limited to small (<3 cm) non-morpheaform primary BCCs in low–moderate risk locations. Larger (>2 cm) and more aggressive tumours are associated with lower clearance (84%), when compared with the excellent clearance rate (98%) achieved in tumours <1 cm.

Pulsed dye laser

Pulsed dye laser (PDL) 595 nm has shown some efficacy for superficial and nodular BCCs. It targets the vascular network of these tumours, leading to size reduction and complete regression in some cases. Clearance rates up to 90% with four treatments have been reported for small superficial and nodular BCCs (<1.5 cm). Cure rates were lower with larger tumours. The use of larger spot sizes and double stacked pulses may allow single treatments to clear BCCs¹² but only a limited number of studies have been performed and the long-term effectiveness is unclear. Adverse events include hypopigmentation and scarring.

Squamous cell carcinoma

SCC is the second most common type of skin cancer, with an estimated annual incidence of 250 000 cases in the United States. It is the second most common cancer in the White population.

SCC is a malignant neoplasm of suprabasal epidermal keratinocytes with full-thickness epidermal dysplasia. They may arise *de novo* or from precursor lesions (actinic keratosis; AK), which have partial-thickness epidermal dysplasia. AKs are not considered to be a premalignant lesion, but rather an important sign of UV-induced photo-damage that signals an increased risk of NMSC. Their estimated progression rate from AK to SCC is 0.025–16% per year. Bowen's disease is the eponymous name for SCC *in situ* (SCCIS) and once the dysplastic keratinocytes have invaded the basement membrane, it becomes an invasive SCC. Although less common than BCCs, SCCs carry a risk of metastasis and death. Despite the rising incidence of SCC, there is a 20% reduction in mortality due to increased public awareness and more aggressive treatment of highly invasive tumours.

Aetiology

Increasing age and chronic UV exposure, including tanning bed use, are the most significant risk factors for SCC development, with a sharp increase in incidence over the age of 40. Approximately 90% of SCCs and over 75% of AKs have inactivation of the tumour suppressor gene p53 secondary to UV damage. p53 usually induces apoptosis of UV-damaged cells, thus preventing the development of dysplastic keratinocytes. In patients who received long-term therapy with psoralen and ultraviolet A (PUVA) for psoriasis, there is a 30-fold increased risk of NMSC, particularly SCCs. Additional risk factors include male gender, low Fitzpatrick skin type, albinism, XP, epidermolysis bullosa, human papillomavirus (HPV) infection, smoking, previous ionizing radiotherapy treatment, chronic inflammation and irritation and chemical carcinogens, such as arsenic and aromatic hydrocarbons. Immunosuppressed individuals, particularly organ transplant patients or those with leukaemia and lymphoma, have a significantly increased risk of SCC, mainly in sun-exposed sites with tumours typically developing 3–7 years after the onset of chronic immunosuppression. A third of individuals with epidermodysplasia verruciformis

develop SCCs due to chronic HPV infection. When SCC develops in an area of chronic inflammation and scarring, it is termed a Marjolin's ulcer (Figure 10.6f). This diagnosis should be considered in any non-healing wound as it carries a 50% risk of metastasis.

Mutational profiling of cutaneous SCC has been evolving over the past decade in an effort to understand SCC biology and identify molecular targets for novel therapeutics. Mutations involving *RAS* are one of the more common types, including *HRAS* and *KRAS*, but others include *TP53*, *CDKN2A*, *PIK3CA*, *MYC*, *FGFR3* and *VHL*. Of note, the use of RAF inhibitors in melanoma patients has resulted in the development of cutaneous SCC with a strong association with *RAS* mutations and epidermal growth factor receptor (*EGFR*) mutations are present in over half of all aggressive SCC.¹³

Clinical presentation

SCCs typically arise in sun-exposed sites (e.g. head, neck, ears, dorsal hands, lips and legs). In those with higher Fitzpatrick skin types, they may arise on both sun-exposed and protected sites. SCCs usually present as a changing solitary lesion developing from a pre-existing AK or Bowen's disease. AKs (Figure 10.6a) appear as multiple ill-defined scaly erythematous macules, papules or plaques, ranging in size from 2 mm to >2 cm, whereas Bowen's disease (Figure 10.6a) is usually a well-defined solitary patch/plaque. Both of these precursor lesions can be confused with eczema or psoriasis, however they are typically asymptomatic and do not respond to topical steroids. The development of tenderness, increased size or scale and erosions may represent progression to SCC. In the setting of immunosuppression, multiple SCCs may develop in an eruptive fashion.

The most common appearance of an SCC is a firm, solitary, skin-colored or erythematous hyperkeratotic papule or plaque (Figures 10.6c,f). It may also present as a pigmented lesion, chronic ulcer, smooth nodule, cutaneous horn or even a verrucous lesion. As the tumour progresses it invades and can become fixed to the underlying structures. Examination of regional lymph nodes is essential to exclude lymph node metastasis presenting as a non-tender enlarged mass.

Genital and perianal SCC may develop in the setting of lichen sclerosus et atrophicus in both men and women. Similarly, chronic evolving warty lesions in the perineal area and red velvety plaques on the penis are alternative presentations of SCC.

Keratoacanthomas (KA) (Figure 10.6b) are rapidly growing nodules with a central keratotic crater that can increase to several centimetres across within a few weeks followed by gradual spontaneous resolution. They typically arise in the sun-exposed extremities of elderly individuals. Due to the potential for local destruction and aggressive behavior they are regarded as a clinical subtype of SCC but do not metastasize and have a self-limiting course. Complete surgical excision to permit microscopic visualization of the junction between normal skin and the edge of the lesion is necessary to rule out well to moderately differentiated SCC and therefore surgical excision is usually

warranted, particularly if the lesion has been present for some time at presentation.

Diagnosis

As with BCC, definitive diagnosis of SCC is made on histology following a punch, shave or excisional biopsy, but clinical presentation with a hyperkeratotic or ulcerated lesion in actinic skin is often highly suggestive of SCC over BCC. The biopsy should be of sufficient depth to differentiate between *in situ* and invasive disease. For keratoacanthoma an excisional biopsy is recommended as the diagnosis depends on the overall architecture of the tumour. The presence of solar elastosis and marginal keratinocyte atypia suggests that the SCC is derived from a prior actinic lesion, whereas scar tissue may represent recurrent disease or SCC development within a prior scar. Immunohistochemical panels incorporating the cytokeratins and CD44 are often used to establish the diagnosis when lesions are poorly differentiated.

Histology usually reveals a predominantly eosinophilic (pink) tumour on standard H&E sections, with a combination of normal and atypical squamous cells arranged as single cells, nests of cells or a single mass. Full-thickness involvement of the epidermis indicates *in situ* disease (Figure 10.7a), whereas dermal involvement represents invasive SCC (Figure 10.7b). Subcutaneous involvement is unusual. Well-differentiated tumours retain squamous features such as foci of keratinization in concentric rings of squamous cells, known as 'keratin pearls'. Keratin production is reduced with loss of differentiation. The risk of recurrence and metastasis is 28.6% and 32.8%, respectively with poorly differentiated tumours, compared to 13.6% and 9.2%, respectively with well-differentiated SCC.¹⁴ When metastasis occurs it is usually to regional lymph nodes, and usually occurs 1–3 years following diagnosis and treatment. Recurrence of the primary lesion often precedes the development of metastasis. Features on histopathology associated with poorer prognosis include tumour arising in prior scar, tumour size >2 cm, depth of invasion >2 mm, desmoplastic reaction surrounding infiltrating keratinocyte islands and perineural or intravascular involvement. High-risk sites for local recurrence and metastasis are the ear and lower lip, respectively (Table 10.2).¹⁵

Dermoscopic findings of SCC include white circles, keratin, blood spots and white structureless zones. These findings may also be seen in keratoacanthoma.¹⁶

Classification

Cutaneous SCC can be broadly divided into *in situ* and invasive disease. SCCIS or Bowen's disease has not crossed the basement membrane and therefore does not have metastatic potential. Invasive cutaneous SCCs are classified by the degree of differentiation they show. Well-differentiated SCCs often demonstrate classic hallmarks of squamous differentiation, including scaly hyperkeratosis and/or the presence of keratin horns. KA is considered to be a form of well to moderately differentiated SCC that typically occurs in the elderly. Poorly differentiated lesions



(a)



(b)



(c)



(d)



(e)



(f)

Figure 10.6 (a) Actinic changes in the face of an elderly female. A crusty actinic keratosis (AK) is present over her right zygomatic arch, Bowen's disease (SCCIS) is present over her right zygomatic arch. (b) A keratoacanthoma (KA) developed quickly on the dorsum of the hand of this elderly woman. A central keratin plug is evident with surrounding elevated erythematous skin reaction. (c) Well-differentiated SCC on the helix of the ear. Ear SCCs are at high risk for metastasis. (d) Moderately differentiated SCC of the leg with central ulceration and surrounding crust. (e) Moderately to poorly differentiated SCC of the scalp with ulceration and recurrent bleeding. (f) A Marjolin's ulcer developing at the site of chronically unstable scar from extensive burns and skin grafting over 20 years before.

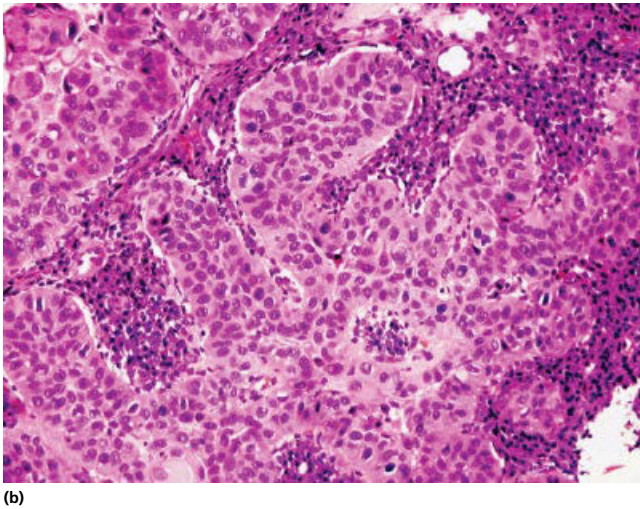
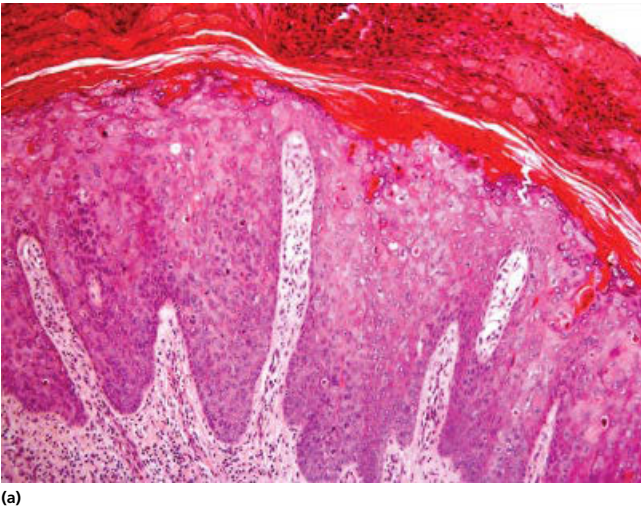


Figure 10.7 Representative histopathology specimens. (a) Bowen's disease (*in situ* SCC/SCCIS) demonstrating atypical cells arranged in nests or single cells involving the full thickness of the epidermis but not invading the dermis. (b) Moderately differentiated SCC with nests of atypical squamous cells infiltrating the dermis.

Table 10.2 High-risk factors for squamous cell carcinoma recurrence and metastasis

Risk factors	
Clinical	Tumour size >2 cm diameter Location: ear/lower lip/template/anogenital Arising in prior scar Immunosuppression
Histological	Poorly differentiated Depth >2 mm or Clark level ≥4 Desmoplastic reaction surrounding infiltrating keratinocytes Perineural or intravascular invasion

usually do not demonstrate much in the way of squamous differentiation and may be difficult to distinguish from other forms of cutaneous malignancy without immunohistochemistry. The other features used in SCC classification include size, location, lymphovascular or perineural spread and specific subtypes of SCC. Lesions under 2 cm in diameter arising with AK or associated with HPV, that are well-differentiated and do not demonstrate lymphovascular or perineural spread, usually have the best prognosis among invasive forms of SCC. Immune suppression, anatomic location on the lower lip, the ear, temple or anogenital region or the development of invasive disease from an SCCIS all carry a worse prognosis.

Cutaneous SCC of the lower lip arises inferior to the wet–dry border of the vermillion and therefore is managed as a cutaneous SCC. SCC arising in chronic wounds and/or scars (Marjolin's ulcers; Figure 10.6f) also pose a higher metastatic risk.

There are a number of rarer subtypes of SCC with varying prognosis. Spindle cell SCC (if not associated with irradiation) is of lower risk. Adenoid (acantholytic) SCC poses a more intermediate risk and adenosquamous carcinoma is a

higher risk tumour. Other subtypes can have varying degrees of risk, including SCC arising in adnexal cysts, clear-cell SCC, squamoid eccrine ductal carcinoma, follicular SCC and papillary SCC.¹⁷

Staging for basal cell carcinoma and squamous cell carcinoma

The American Joint Committee of Cancer (AJCC; Table 10.3) use the TNM classification system to stage both BCC and SCC. This system combines grades within each of the T, N and M components to produce a combined stage. Staging is divided by anatomic site and then into clinical (c) and pathology (p) stages. The eyelid, vulva and penis have separate classification systems to the rest of the skin due to their thinner skin and proximity to the brain, urogenital system/pelvis and large vascular structures, respectively. We will discuss the staging for the general skin.

Clinical stage (c) is the presumed staging based upon physical examination, medical imaging and biopsy results from the primary tumour and/or fine needle aspiration (FNA) cytology (with or without ultrasound guidance) of clinically involved nodes. Pathology staging (p) is the staging based upon the 'excisional' biopsy, including nodal basins if suspected of being involved. CT scanning with contrast is the most widely used imaging modality for advanced cases of NMSC. It gives good detail about the sites and size of involved nodal groups, provides definitive evidence of bony invasion and gross perineural involvement by demonstrating widening of foramina that transmit nerves. Positron emission tomography (PET) scanning can be combined with CT to increase sensitivity and specificity, but it is only effective for PET-avid tumours. The value of PET imaging in BCC is limited. Approximately half of BCCs are PET-avid (in small series in head and neck BCC) with the nodular type most likely to show fluorodeoxyglucose (FDG) uptake. PET can be very useful in SCC and occasionally

Table 10.3 The American Joint Committee on Cancer (AJCC) TNM system for basal and squamous cell carcinomas of skin (excluding eyelid, vulva and penis)

Component grade	Tumour (T0–4)	Lymph nodes (LN) (N0–3)	Metastasis (M0–1)
0	No evidence of primary tumour	No spread to LN	No spread to distant organs
is	Carcinoma <i>in situ</i> (confined to the epidermis)		
1	≤2 cm in longest diameter and has ≤1 high-risk feature ^a	Spread to 1 ipsilateral LN and is ≤3 cm in diameter	Spread to distant organs
2	>2 cm in longest diameter OR Any size with ≥2 high-risk features ^a	Spread to (a) 1 ipsilateral LN and is >3 cm but ≤6 cm in diameter or (b) >1 ipsilateral LN, ≤6 cm in diameter or (c) bilateral or contralateral LN ≤6 cm in diameter	
3	Any tumour size + LN OR Tumour extension to facial bones	Spread to any LN >6 cm in diameter	
4	Tumour extension to other bones or into base of the skull		

^aHigh-risk features include: depth >2 mm, invasion into reticular dermis or subcutis, perineural invasion, tumour on ear or non-hair-bearing lip, poorly differentiated or undifferentiated on histology.

demonstrates more soft tissue involvement than what was detected by PET alone. MRI is the imaging modality of choice to identify the extent of soft tissue involvement, including perineural involvement with thickening of nerves. This is especially important in tumour types such as SCC that demonstrate skip lesions (haematogenous spread) along nerves. Mutational profiling for p16, otherwise known as cyclin-dependent kinase inhibitor (a tumour suppressor gene) is principally used in oropharyngeal, perianal SCC as it is related to human papillomavirus status in these cases and has not found a role in cutaneous SCC currently.

The key points in the AJCC staging system are that stage and T level correlate (i.e. T4 lesions are stage 4) unless an N or M level upgrades the stage. Stage 1 and 2 are for T1 and T2 lesions (respectively) with no nodal involvement of distant metastases. Stage 3 and above indicates a T3 lesion or any nodal involvement. Stage 4 is for advanced T stage (T4), advanced nodal disease (N2 or above) or any distant metastases (M1).

Overall stage

Information from the stage categories is grouped together to provide the overall tumour stage.

- Stage 0: T0/is, N0, M0
- Stage I: T1, N0, M0
- Stage II: T2, N0, M0
- Stage III: T3, N0, M0 or T1 to T3, N1, M0
- Stage IV: T1 to T3, N2, M0 OR Any T, N3, M0 OR T4, any N, M0 OR Any T, any N, M1

Management

Staging permits an appropriate assessment of the severity of tumour invasion and whether a multidisciplinary team approach is necessary. Advanced skin cancer can be complex and present significant on-going treatment, management and surveillance problems that require a team approach to care. Luckily, the vast

majority of cutaneous SCCs are low grade at the time of diagnosis. In general, those with *in situ* disease or circumstances that prevent definitive excision may be treated with topical therapies, whereas more invasive disease requires surgical management. Treatment of SCC depends largely on assessment of risk of tumour recurrence or metastasis (Table 10.4). Non-surgical treatments can also be used as a neoadjuvant to reduce tumour size prior to excision. Close follow-up is necessary for all patients, as those who develop recurrence typically do so within 2 years of initial treatment.

Medical

Topical therapy

Cryotherapy is a quick and cost-effective method of treating SCCIS with a single 30 s freeze, achieving a clearance rate of 100% and a recurrence rate of 0.8% over a six-months to 5-year follow-up period in one large study. Overall, freeze times, number of cycles, clearance (50–100%) and recurrence (0.8–36.1%) rates are variable.¹⁸ Adverse events include blistering, scarring and hypopigmentation. This should not be used to treat invasive SCC.

Topical imiquimod 5% cream used for SCCIS (5/week for 16 weeks) resulted in a 73% clearance rate, with no recurrence after nine months of follow-up. 5-Fluorouracil once or twice daily demonstrated clearance in 69% and 93% of tumours after 4 weeks and 8 weeks, respectively, with a recurrence rate of 21% at 2 years.¹⁸ Adverse events include erythema, pruritus and pain. These agents cannot be used to treat invasive disease.

Topical diclofenac 3% gel has been approved for the treatment of AK but only a few studies have demonstrated clearance of SCCIS lesions. Diclofenac inhibits cyclooxygenase-2, which is upregulated in NMSC lesions, therefore it inhibits angiogenesis and induces apoptosis. Further research is required to determine its long-term efficacy and optimal dosing regimen.

Table 10.4 Treatment options for squamous cell carcinoma

Treatment modality	Ideal indication
Topical	
Cryotherapy, imiquimod, 5-fluorouracil, diclofenac ^b	SCCIS
Oral	
Capecitabine, cetuximab, erlotinib, gefitinib, interferon	Reduce incidence of SCC in organ transplant patients. Locally advanced and metastatic BCC. Neoadjuvant use prior to surgery to reduce surgical defect size of all subtypes SCCIS
Photodynamic therapy	Primary SCCs in those unwilling or unable to undergo surgery; adjuvant therapy post surgery for high-risk SCC; palliation; SCC of external auditory canal
Radiotherapy	
Procedural	
Standard excision	Suitable for majority of cutaneous SCC, clearance may not be as good as MMS in high-risk SCC in head and neck
MMS	High-risk SCCs in the head and neck ^a
Electrodessication and curettage	Low-risk, small (<1 cm), well-differentiated primary SCCs. SCCIS

^aHigh-risk: tumour diameter >1 cm, invasive (>6 mm deep) or aggressive histology, perineural, bone or muscle invasion, poorly defined clinical margins, prior site radiation or scarring, recurrent large SCC or immunosuppressed host.

^bLong-term follow-up outcomes still awaited. SCCIS, squamous cell carcinoma *in situ*.

Oral therapy

Capecitabine is an oral prodrug converted in the body to 5-fluorouracil (5-FU) and has demonstrated efficacy at low dose in the reducing the incidence of SCC in patients with solid organ transplants. Adverse events include fatigue, nausea, gout, hand-and-foot syndrome and poor renal function. Its optimal dosing regimen and use in non-organ transplant patients are yet to be established.

Approximately half of advanced SCCs contain *EGFR* mutations, which may lead to a more aggressive phenotype. Activation of *EGFR* stimulates keratinocyte proliferation and leads to resistance to apoptosis. Cetuximab, a monoclonal antibody against the *EGFR*, is FDA-approved for the treatment of recurrent or metastatic SCC of the head and neck. Additional treatments include interferon, erlotinib and gefitinib, which have been employed with various results. These treatments may be indicated in high-risk SCCs, which may require formal staging with CT and MRI to detect perineural involvement and sentinel lymphadenectomy in cases where lymph node involvement is suspected.

Photodynamic therapy

PDT can be used for SCCIS, in patients with multiple lesions or in poor surgical candidates (either unable or unwilling to undergo surgery). Clearance rates for SCCIS range from 84% to 96%, and recurrence rates vary from 0.5% to 31%. PDT is not

recommended for invasive SCC due to higher recurrence rates (up to 84% for invasive tumours) and risk of metastasis.

Radiotherapy

External beam therapy can be used for primary SCC treatment in those who are poor surgical candidates, as an adjunct treatment modality to surgery or as palliative treatment for symptomatic relief. Radiotherapy is useful in SCC because it semi-selectively targets rapidly dividing cells. Moderately to poorly differentiated (G3 and G4) tumours are often rapidly dividing and are more likely to demonstrate lymphovascular and perineural invasion and therefore radiotherapy helps control of the field and distant sites. However, when patients present late with advanced stage G1 and G2 tumours it can be challenging to treat them adequately with radiotherapy alone. Similarly, patients who do not complete their course of radiotherapy, select for more radio-resistant clones within their tumours and thereby decrease the effectiveness of radiotherapy for further dosing should this be required into the future. The use of adjuvant radiotherapy in high-risk SCC reduces the risk of local recurrence and improves overall disease-free survival. Despite the risk of hearing loss, it is particularly useful for SCCs of the external auditory canal. Ionizing radiation should be avoided in those with BCNS and XP.

Procedural

Standard surgical excision

Standard excision is the most widely used treatment options for invasive SCC. A 5 mm clinical margin removes almost 95% of low-risk SCC lesions.¹⁹ A wider margin up to 1 cm is used for more advanced lesions (e.g. >2 cm diameter, poorly differentiated SCC). DRAPE¹⁰ is used when there is uncertainty about the margin so formal histopathology assessment is awaited (with the excision site being dressed or skin grafted) prior to formal reconstruction. In advanced cases, the additional margin can provide added safety due to the risk of missing non-contiguous/in-transit metastases or skip lesions indicative of haematogenous spread and often does not change the type of reconstruction used (as seen in Figure 10.8a–d).

Mohs micrographic surgery

Mohs surgery offers the potential to achieve complete tumour resection while preserving as much healthy tissue as possible. However, Mohs surgery is time and resource expensive, requires specialized equipment and training and, as such, is poorly suited to managing the vast majority of cutaneous SCCs in our ageing populations. Case series demonstrate a lower local recurrence rate than with other modalities, with one series showing a cure rate of 92% at 4-year follow-up. MMS is considered by many to be the treatment of choice for high-risk lesions in the head and neck (Figure 10.9) where there is an increased chance of recurrence (lip, ear, nasal tip, eyelid, nail bed and anogenital area) but no controlled, clinical trial has been undertaken to permit direct comparison with standard surgical excision to date. Additional

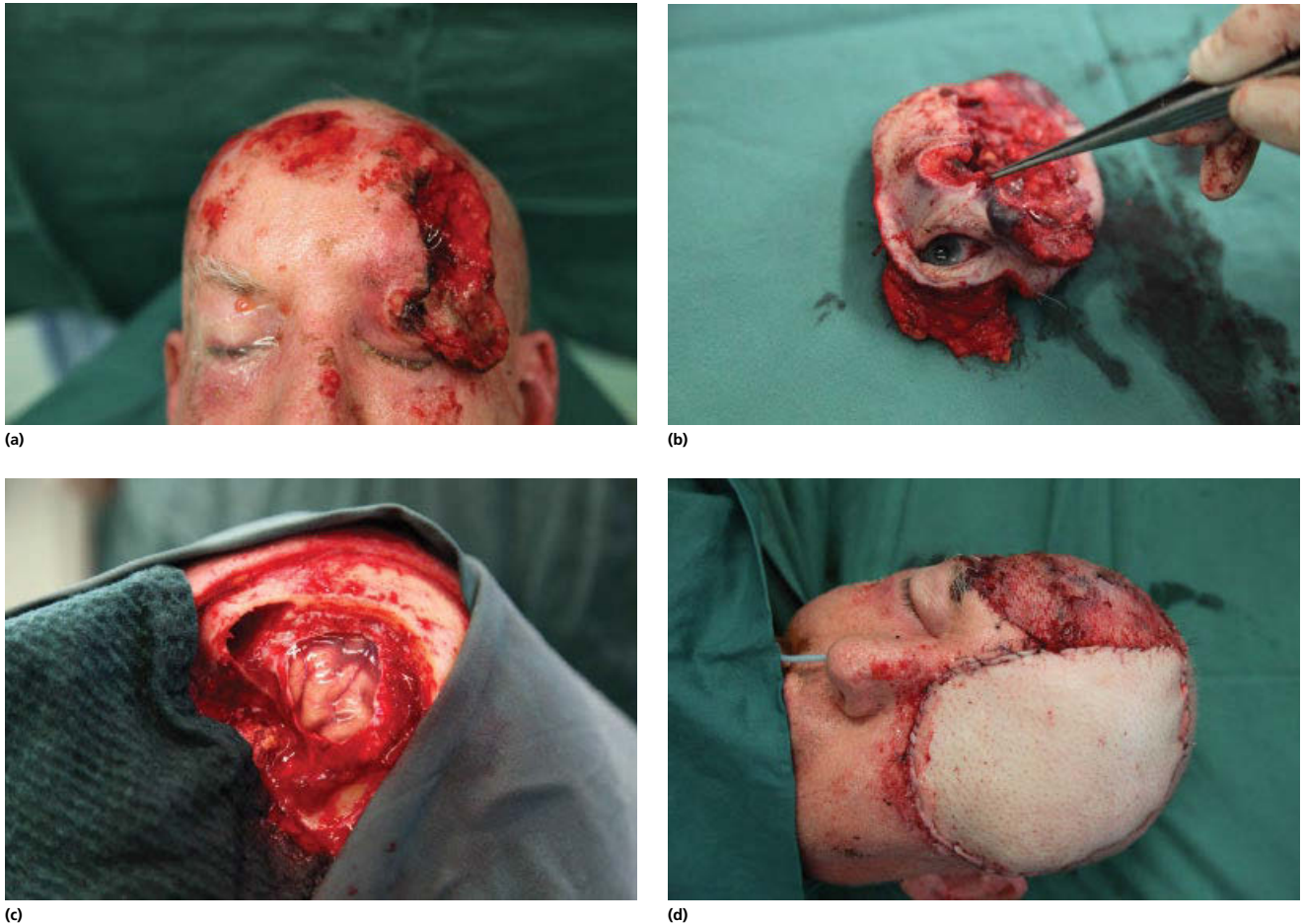


Figure 10.8 (a) An advanced fungating SCC infested with maggots and invading the frontal bone and the dura of the frontal lobe of the brain. There was no systemic disease evident on imaging or clinical examination so using a multidisciplinary approach, the tumour was resected with the globe en-bloc (b), the remaining defect can be seen with exposed brain (c) before reconstruction in layers for dura through to a vertical rectus abdominus myocutaneous flap for defect coverage (d). The patient had postoperative radiotherapy and continued to be followed up in a multidisciplinary setting (specialized nursing, ENT, neurosurgery, plastic and reconstructive surgery, radiation oncology and opinions from specialist head and neck medical oncologists and radiologists).

indications for MMS include tumour diameter >1 cm, invasive (>6 mm deep) or aggressive histology, perineural, bone or muscle invasion, poorly defined clinical margins, prior site radiation or scarring, recurrent large SCC or immunosuppressed host.

Electrodessication and curettage

Electrodessication and curettage has been demonstrated to be as efficacious as standard surgical excision in the treatment of low-risk, small (<1 cm), well-differentiated SCCs,²⁰ unless there is great concern over location risk and immune status. Recurrence rates vary with physician experience between 2 and 18.8% over 1–5 years follow-up.

Notes on reconstruction

Appropriate management of NMSC should follow sound oncology principles, including the establishment of the definitive diagnosis through biopsy, adequate staging, identification of

involved structures and complete tumour clearance where this is the goal. Sometimes it is not possible to cure the patient, but surgical or adjunctive therapies are used to remove tumour deposits that are symptomatic due to persistent bleeding or ulceration, sometimes referred to as a 'toilet' procedure to assist in daily cares. If a single treatment modality is most likely to be effective long term, then this modality is the treatment of choice in most cases, depending on a risk–benefit profile for the patient. It is essential to have a clear perspective of what the goal of any treatment is and be clear about this and the likely defect with the patient. It is not enough to just render the patient tumour-free. There should also be a plan for how to restore form and function. It is important to consider what should be done if the tumour is more advanced or has regressed upon presentation for treatment. In some cases, it is very obvious that a multidisciplinary approach is necessary (e.g. Figure 10.8). A patient-focused approach combined with further development of targeted therapies offers significant hope to further reduce the morbidity and mortality of NMSC in our rapidly ageing populations.

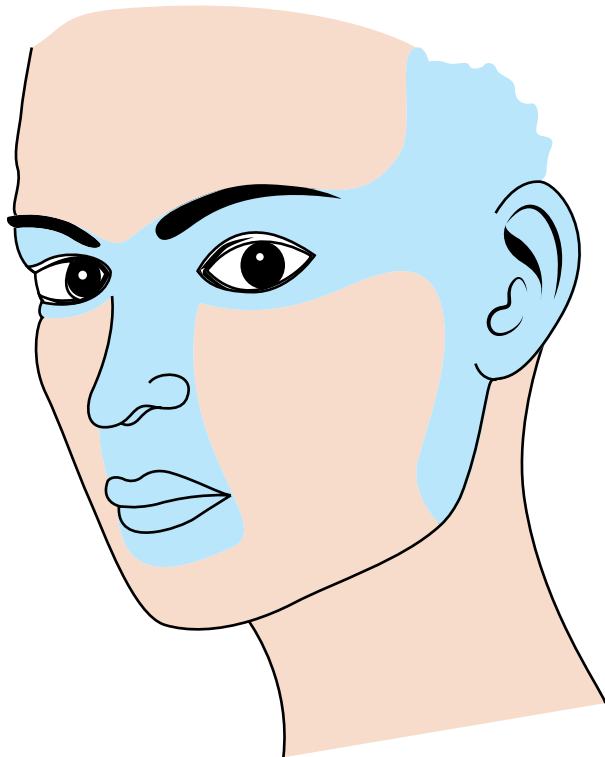


Figure 10.9 High-risk zone for SCC where Mohs micrographic surgery is most suitable.

Acknowledgements

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CHAPTER 11

Atypical skin lesions

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Introduction

The atypical skin lesions are a diverse group of tumours that are uncommon but clinically important. They include a spectrum from benign lesions through to those that demonstrate local invasion and/or distant metastatic potential. Since these lesions are only seen infrequently during clinical practice, it is important to have a solid grounding in the general principles of their management. We also cover some common surgically relevant lesions of the skin including common types of cysts. A clear understanding of cysts and their aetiology is important to direct their management. We have divided the atypical lesions into benign or malignant and then by their cell/tissue of origin to provide a framework for their study. Such a framework is not perfect due to our evolving understanding of the molecular and cellular bases of these tumours, but it provides a foundation for their study.

Benign lesions

Benign lesions are clinically important for several reasons. They can be confused with malignant lesions, be symptomatic due to their size, appearance, location, irritation and bleeding or may undergo malignant transformation. Knowledge of the uncommon benign lesions can also provide a means to understand their malignant counterparts. Such knowledge is invaluable to the surgeon when examining the correlation between the clinical presentation and the histopathologic diagnosis. Any discrepancy between these two should prompt the surgeon to seek clarification and/or a second opinion regarding the histopathologic diagnosis. Good communication between the surgeon and the histopathologist is essential for good care.

Cysts

True cysts are defined as epithelial-lined 'fluid-filled' spaces and are relatively common in clinical practice. There can be many names for various cysts, including a 'wen', and often terms are incorrectly applied in general usage.

Epidermoid cysts (keratin cysts)

These common cysts enlarge subcutaneously but maintain a connection to the overlying skin at their punctum. Lined by squamous epithelium, they produce soft keratin that fills the cyst with a waxy/cheesy material. Although often called sebaceous cysts, this is a misnomer as they do not contain sebum. True sebaceous cysts are uncommon, arising from the sebaceous glands. There are two types of epidermoid cyst. Epidermal inclusion cysts result from the inoculation of squamous epithelium into the dermis during trauma (e.g. body piercing). Infundibular cysts specifically arise at the infundibular portion of the hair follicle, but are otherwise clinically the same.

Management depends upon their state at presentation. Patients typically present with them either as non-tender lumps attached to the undersurface of the skin or when they become infected. Surgical excision in quiescence is ideal, but must include excision of their attachment to the overlying skin (the punctum). Significant compression can cause a release of keratin through the punctum, but this also increases the risk of infection. In infection, decompression of the cyst abscess, removal of the cyst material and curettage of the cyst lining allows resolution of the infection and often reduces the size of the lesion down to a small scar. Curettage is associated with a significant risk of recurrence because of its inability to ensure complete removal of the lining of the cyst. Once the scar has settled appropriately, the scar, remaining cyst and its punctum can be removed to stop recurrence. Uncommonly, they spontaneously resolve by becoming marsupialized and discharging.

Tricholemmal (pillar) cysts

Tricholemmal cysts are similar to epidermoid cysts except that they specifically arise from the outer root sheath of hair follicles. They typically arise in the scalp as this has the highest density of hair follicles. They are usually multiple and the tendency to form tricholemmal cysts runs in families. Surgical excision is the treatment of choice and when excised with the overlying hair follicle, recurrence is unlikely. On histology, they can demonstrate areas of nuclear atypia, mitotic figures and dyskeratosis

and therefore be confused with squamous cell carcinoma (SCC), but it is rare for them to undergo malignant transformation.

Dermoid cysts

Dermoid cysts are hamartomas, often containing multiple epithelial derivatives (skin, hair, etc.). They are found in numerous organs and regions of the body. In the skin they are often near the midline or in the head and neck along tissue fusion planes and therefore can have connections to deep structures such as underlying bone. Their superficial position and hamartomatous origin make them a lesion of childhood (present at birth, but enlarging post nately). They can be associated with other conditions (epibulbar dermoids and Goldenhar syndrome). Excision of the cyst with any communications to underlying structures is effective to prevent recurrence.

Benign lesions associated with epidermis

Acrochorda

Acrochorda (acrochordon = singular) or skin tags are fibroepithelial polyps that appear as raised, smooth or irregular-surfaced pieces of skin on a small stalk called the peduncle. Nearly half of the adult population has at least one near skin creases in the neck, axilla, groin, breasts and eyelids. Shear forces of skin on skin are thought to be their underlying cause and therefore they are common in obesity. Female gender, human papilloma virus 6 and 11 and familial tendency are all thought to predispose to them. They may be symptomatic due to irritation from clothing or jewellery or when they bleed because of tractional injury. Acrochorda are usually small (2–5 mm) but occasionally larger. They are composed of a fibrovascular core and adipose tissue covered by normal skin. Numerous treatment modalities are available from ligation to cryotherapy and simple surgical removal, but the patient must be aware of their risk of further lesions.

Warts

Warts are focal (verrucous) keratinized lesions caused by human papilloma virus (HPV). The virus replicates in the stratum granulosum, inducing underlying basal keratinocytes to proliferate, causing a hyperkeratotic response. They may appear raised, flat, filiform, cauliflower or ingrown (e.g. plantar warts) and can also occur under or around the nails. Over 150 different types of HPVs have been characterized but typically, only 1–2 strains cause each normal variant (verruca vulgaris HPV 2 and 4, cauliflower/Butcher's warts HPV 7, palmoplantar warts/myrmecia HPV 1, flat warts HPV 3 and 10).¹ These are distinct from the HPV strains associated with genital warts (HPV 6 and 8) and those producing an increased risk of intraepithelial neoplasia or invasive tumours affecting the vulva, penis, cervix or oral cavity (HPV 16 and 18).² Warts may be singular or multiple with a predilection for the hands due to contact transfer. Wart infections are self-limiting and resolve once the host mounts an immune response to the virus. Surgical excision produces some release of human papilloma virions, but topical therapies are a less invasive approach to induce an immune response.

Therapy is largely divided into management of the hyperkeratosis and induction of a suitable immune response. Hyperkeratosis management can be undertaken by multiple means, including use of salicylic acid (3–60%, daily or twice/day), occlusive dressings, cryotherapy and shaving by scalpel or sharp curette.

Topical therapies to promote an immune response include podophyllin (resin or toxin), formalin (3% solution), imiquimod (Aldara), retinoids such as tretinoin and 5-fluorouracil (5-FU). Podophyllin is available over the counter in most countries and can be applied without specific training. Other approaches that have been used include laser therapy, intralesional agents (bleomycin and interferon alpha) and topical allergens or caustic agents. Although usually successful, they have significant potential side effects not found with the mainstream therapies above. Patient education usually helps to avoid surgical intervention and scarring. Histologically, warts demonstrate koilocytes (vacuolated cells with viral infection) and hypergranulosis with columns of parakeratosis and compact orthokeratosis. Typically, the marked hyperkeratosis and papillomatosis are associated with elongated rete ridges sloping inwards to the centre of the lesion.

Malignant transformation is sometimes seen in long-standing immune suppression, but it is unclear to what degree HPV infection contributes because the patients already have increased risk of skin cancers. Epidermodysplasia verruciformis (Lewandowsky–Lutz dysplasia) is a rare autosomal recessive skin disorder that increases the susceptibility to HPV infections. These patients have a very high risk of skin cancer due to the increased risk of malignant transformation of their lesions.

Benign lesions associated with hair follicles

Pilomatrixoma

This is a benign appendageal tumour that typically occurs in the head and neck region and demonstrates differentiation towards hair (follicular) cells. Although considered a tumour of children, they can occur in adults. They are associated with a mutation in the *CTNNB1* gene and although familial cases have been observed, the mode of inheritance has not yet been established. In addition to sporadic cases, they may be associated with Gardner syndrome (familial colorectal polyposis), myotonic dystrophy (especially if multiple lesions) and sarcoidosis.

Pilomatrixomas present as firm nodules that are usually solitary and asymptomatic, but occasionally associated with local pain if infected or inflamed. Usually slow-growing, they can be confused with other skin lesions, including nodular basal cell carcinoma (BCC), despite their deep position in the lower dermis or underlying adipose tissue. Lesions often contain calcium deposits that are visible on medical imaging. Pilomatrix carcinoma is well documented but very rare.

Histology demonstrates irregular islands of epithelial cells surrounded by a connective tissue capsule that produces a diffuse lesion in most cases. The epithelial cells are divided into basophilic (germinal) cells around the periphery and shadow

cells (anucleated) centrally, in keeping with a hair-like process. Chronic lesions have fewer basophilic cells and calcium deposits can be visualized through the use of special stains (von Kossa). The diffuse nature of most lesions leads to a greater risk of local recurrence unless a wide local excision is performed (1 cm or greater). Mohs micrographic surgery has been trialed to minimize resection of uninvolved tissue, but its effectiveness remains unclear relative to standard surgical excision.

Trichoepithelioma

Trichoepithelioma is a tumour of the hair follicle and adnexae that is often confused with BCC (Figure 11.1). They typically arise as single or multiple as small (<1 cm), firm round lesions on the face before or after puberty that may display pearly. The lesions form a basic hair follicle but no hair shafts and may have a different surface colour depending on their site of origin. Solitary lesions are typically sporadic, but a familial (multiple) form of

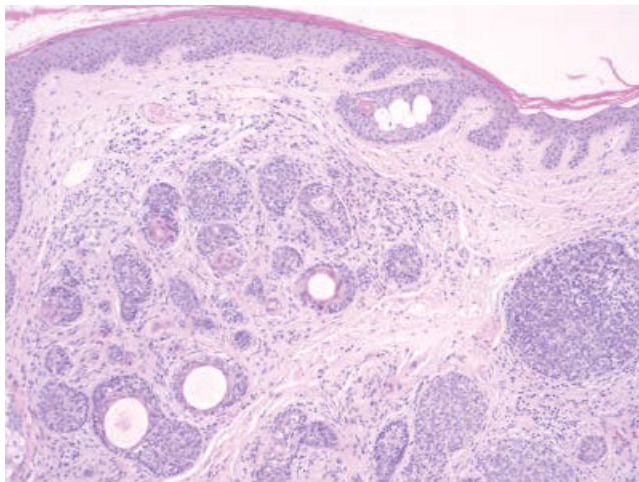
trichoepithelioma has been associated with mutations on chromosome 16q21 (Brooke–Speigler syndrome).³ Trichoepitheliomas are composed of nests of basophilic cells often arranged like a hair follicle but they do not display the surrounding myxoid stroma or perineal clefting found in BCC. A desmoplastic variant commonly forms an annular shape that can be confused with BCC and has a marked desmoplastic surrounding response on histology. Simple surgical excision of all forms without a significant margin is usually effective in preventing recurrence.

Trichofolliculoma

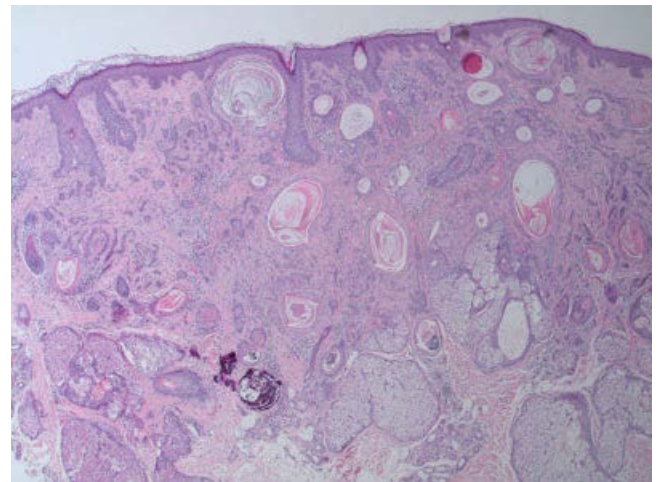
Trichofolliculomas arise directly from hair follicle tissue. They are uncommon small solitary nodules found around the face of adults. A central pore may express sebum and/or hair. They are thought to arise from hamartomatous rests of stem cells that show incomplete differentiation towards hair follicles, as supported by the prominence of Merkel cells within the follicular



(a)



(b)



(c)

Figure 11.1 (a) Multiple trichoepitheliomas within the V1 dermatome in a 6-year-old girl. No relatives had Brooke–Speigler syndrome and therefore this may have been the result of a sporadic mutation. The main cluster was treated by simple excision. (b) Histopathology demonstrates nests of basaloid cells (without clefting) throughout the dermis with keratin microcysts. There is recapitulation of primitive hair bulbs, but no epidermal connection. (c) Desmoplastic trichoepithelioma demonstrating a 'paisley-tie' pattern of strands and cords of basaloid cells, keratin microcysts, microcalcification and stromal fibrosis.

epithelium in these lesions.⁴ Their central follicle may have secondary or tertiary follicles and hair shafts may be present within the follicle lumen on histology, helping to distinguish them from trichoepitheliomas, along with a prominent surrounding stroma. There is a sebaceous variant where the central follicle has well-differentiated sebaceous lobules emptying into it. They are considered benign with only a single case of perineural invasion ever reported.⁵

Tricholemmoma (trichilemmoma)

Tricholemmoma is an uncommon benign tumour arising from the outer root sheath of hair follicles. It appears as a warty or smooth papule on the face and neck region and when multiple lesions are present, and it is diagnostic of Cowden syndrome (hamartomatous intestinal polyposis with multiple tricholemmoma lesions). Cowden syndrome demonstrates autosomal dominant germline inheritance associated with mutations of the tumour suppressor gene *PTEN* (phosphatase and tensin homolog) located on chromosome 10q23.3⁶ but other mutations are occasionally found (e.g. *BMPRIA*). *PTEN* is negative in sporadic tricholemmomas, but often positive in Cowden syndrome, presumably due to a loss-of-function mutation. On histology it appears as lobular downward growths of epidermis and is often surrounded by a hyaline band or thickened basement membrane. Clear cells or cells with prominent vacuoles are typically present (glycogen) and the lobules demonstrate peripheral palisading.

Benign lesions associated with sebaceous glands

Sebaceous hyperplasia

Sebaceous hyperplasia is a common condition characterized by single or multiple soft small papules on the face, neck and occasionally chest in middle-aged to elderly individuals. Most common on the forehead, nose and cheeks, it presents with raised yellowish areas around a central follicular ostium. It can also affect the areola, penis, vulva and oral mucosa.⁷ Rhinophyma is considered by many to be a severe variant of sebaceous hyperplasia, but has closer linkages with acne rosacea. The pathophysiology relates to the effect of androgens on sebaceous glands. Androgens drive sebocyte turnover and with advancing age, androgen levels drop and sebocyte turnover slows down, resulting in overcrowding by primitive sebocytes that do not contain much sebum. As a result, the glands enlarge with a large number of immature cells. There is no malignant transformation potential, but they may be associated with skin cancer in transplant patients.⁸ Sebaceous hyperplasia does not normally require any treatment, however patients may present due to aesthetic concerns or because the lesions may be confused with BCC. Treatment options are manifold, including cryotherapy, photodynamic therapy, chemical peels, laser therapy (CO₂ or pulsed-dye), shave excision or formal excision, but all of these are associated with varying degrees of scarring and/or pigmentation changes. Oral retinoids (e.g. isotretinoin) have been used with some success,

but sebaceous hyperplasia can recur upon cessation of therapy⁹ therefore they are only indicated for severe disfiguring sebaceous hyperplasia.

Benign sebaceous adenoma

A benign neoplasm with a well-differentiated sebaceous pattern that presents as a slowly enlarging smooth, sometimes yellow papule with a central depression on the face and scalp. It can readily mimic BCC and is distinguishable from sebaceous hyperplasia histologically by a higher turnover of sebocytes, good differentiation of these cells and because it is often mixed with other pilosebaceous components (e.g. hair). Benign sebaceous adenoma is the benign end of the spectrum of neoplasms involving sebaceous differentiation. Sebaceous neoplasms as a whole can show variable degrees of differentiation (from well to poorly differentiated) and a mixture of differentiation patterns based on the close proximity of the sebaceous gland with the pilosebaceous unit (hair, sebaceous and apocrine sweat glands). Lesions can be either solitary or multiple and, when multiple, may be associated with systemic conditions (e.g. Muir-Torre syndrome – internal malignancy with multiple sebaceous adenomas or sebaceous neoplasms).

Naevus sebaceous of Jadassohn

This is a well-circumscribed benign lesion presenting as a yellow to light brown velvety or papular plaque on the scalp and face of children or young adults (Figure 11.2). In addition aesthetic considerations, naevus sebaceous is important because it carries a risk of transformation to other tumours and can be associated with neurological phenomena as part of sebaceous naevus syndrome. Lesions may be present at birth, but typically get larger before or around puberty. Some lesions have a *PTCH* gene mutation, but most are considered to have a mosaic genetic abnormality. Naevus sebaceous is relatively unusual among the epidermal naevi because it often contains all skin components including epidermis, sebaceous and apocrine glands, connective tissue and primitive hair follicles. Therefore it is sometimes called an organoid naevus and can transform into numerous tumours both non-malignant (e.g. syringocystadenoma papilliferum, trichoblastoma or tricholemmoma) or more commonly malignant (10–30% lifetime risk of BCC or other malignant tumours). If a focal area of change develops within a sebaceous naevus, this should be biopsied to exclude malignant transformation, and patients and their families should be advised to monitor the lesions for such changes.

Rarely, patients with very large lesions can experience neurological disorders such as epilepsy. When these neurological disorders are more marked, including a combination of seizures, developmental delay, cranial nerve palsies and/or hemiparesis and structural abnormalities of the brain, the constellation is referred to as naevus sebaceous syndrome. Naevus sebaceous is well-circumscribed and therefore amenable to excision with a minimal margin and most practitioners would advise removal by teenage or early adult life.

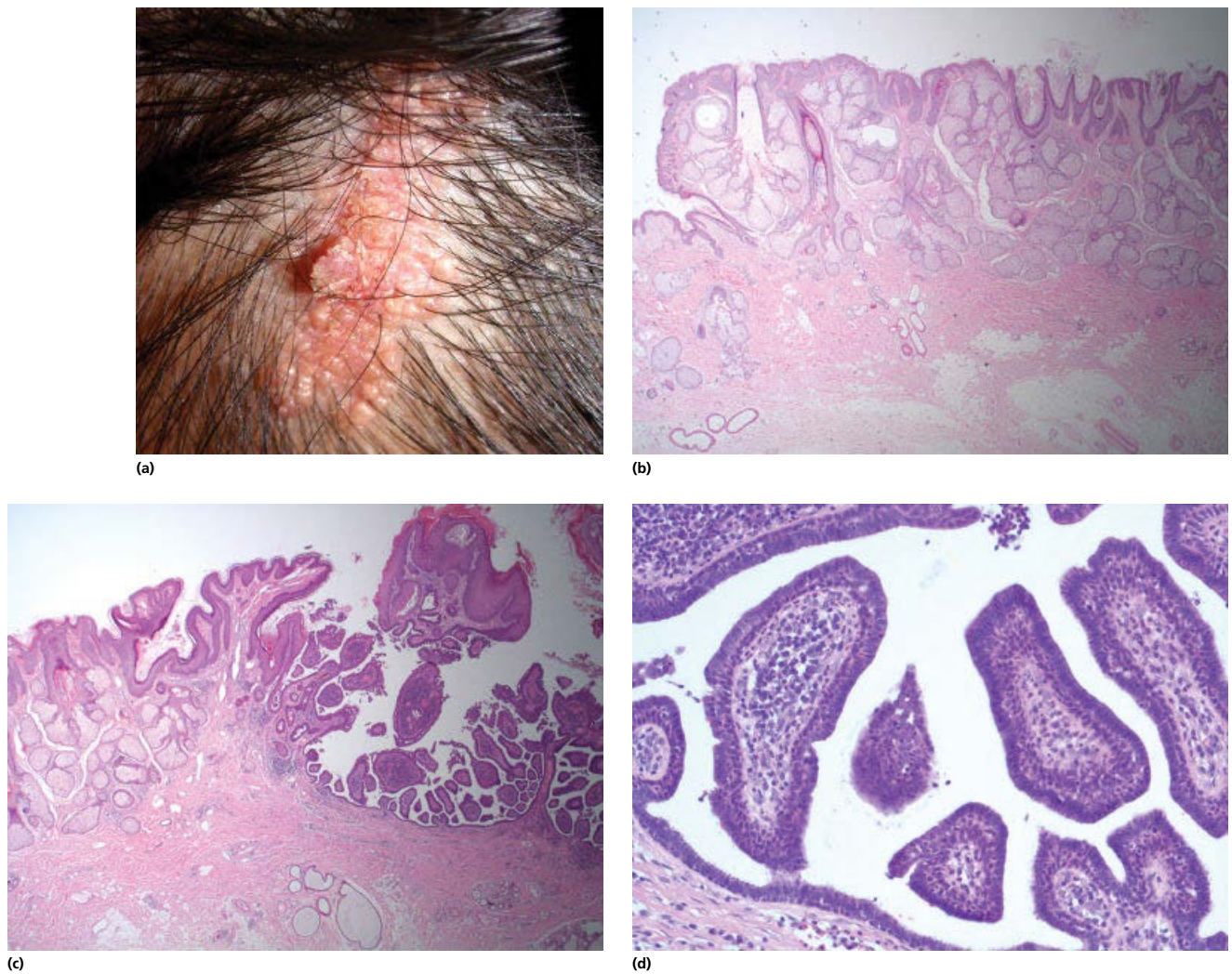


Figure 11.2 (a) Naevus sebaceus (of Jadassohn) of the scalp in an 11-year-old child, managed by simple excision. (b) Histopathology demonstrating epidermal naevus-like epidermal hyperplasia, increased sebaceous glands associated with alopecia and ectopic apocrine glands in deep dermis (bright pink staining). (c) The other end of the same lesion showing a junction with a syringocystadenoma papilliferum (right), (d) a benign apocrine neoplasm commonly associated with naevus sebaceus that has apocrine cells demonstrating 'decapitation' secretions.

Benign lesions associated with apocrine glands **Hidradenoma papilliferum**

This is a benign cutaneous tumour derived from apocrine tissue that most commonly presents in the vulval region of middle-aged women, but can appear at extravulval sites in both sexes. Lesions appear as round, domed nodules that may ulcerate. On histology, they are identical to intraductal papillomas of the breast and therefore are considered to be ectopic breast tissue. They have a complex papillary structure showing an inner layer of PAS-positive columnar/cuboidal epithelium, a basal layer of myoepithelial cells that are S-100 positive and a fine fibrovascular stroma.

Poroma

Poromas are benign adnexal neoplasms of epithelial cells that display ductal (usually distal) differentiation. Although often described as 'eccrine poromas', the majority are thought to be

apocrine in origin. They present as solitary (<2 cm) skin-coloured papules or nodules that may be mistaken for other lesions or develop within naevus sebaceus. Typically asymptomatic, they can cause pain on ulceration or on the sole of the foot. Simple surgical excision is the treatment of choice because it is usually curative and provides suitable specimens for histopathology.

Benign lesions associated with sweat ducts **Syringoma**

This is a benign adnexal tumour that typically presents as single or multiple small (<3 mm) skin- or yellow-coloured dermal papules usually around the eyelids, penis or vulva. They affect 1% or less of the adult population and are usually the result of benign growth of eccrine sweat gland elements or eccrine differentiation of pluripotent stem cells. Four variants have been recognized:¹⁰

localized, Down syndrome associated, generalized/multiple (including eruptive syringomas) and familial variant.

On histology, they are a superficial dermal lesion characterized by abundant small ducts with a double-cuboidal epithelial lining and a sclerotic surrounding stroma. A PAS-positive secretion is present within the ductal lumen and the classic comma-shaped appearance of ducts on this lesion represent ducts that have elongated tails of epithelial cells upon sectioning. There are multiple management options from simple excision through electrocautery, cryotherapy, laser therapy, topical agents, topical or systemic retinoids. Simple excision with minimal margin achieves a definitive histopathology diagnosis (to exclude BCC) and can produce minimal scarring. Scar burden increases with the number of lesions present and therefore there is no perfect treatment for large numbers of lesions.

Cylindroma

Cylindromas are benign adnexal tumours (apocrine or mixed) that present as single or multiple lesions in the head and neck. When single, they are typically sporadic occurring from middle age onwards as slow-growing, rubbery nodules that may reach up to 5–6 cm in size. Mutations in the *CYLD* gene (16a12–q13) are the principal cause of multiple cylindromas and may indicate familial inheritance (Brooke–Spiegler syndrome – an autosomal dominant syndrome with variable penetrance).¹¹ Cylindromas may have a similar origin to related tumours such as spiradenomas and trichoepitheliomas because mixed lesions have been reported. Definitive diagnosis is by biopsy. Histology demonstrates a dermal tumour with islands of basophilic cells demonstrating peripheral palisading, surrounding larger, more pale central cells. Lamina densa-like material (predominantly collagen IV) surrounds the islands and the nests to separate them from the dermal matrix and these often give the lesions a jigsaw-like pattern of polygonal or oval nest shapes. Spiradenomas are related lesions, but are distinct from cylindromas because they have lymphoid invasion. Management is typically through surgical excision, which is curative following complete excision.

Spiradenoma

These are benign, solitary tumours thought to be of apocrine origin that present on the upper, anterior half of the body as pink, red, purple or blue nodules up to 1 cm in diameter. They typically occur in 15- to 35-year-olds, although infantile cases (with superficial dermal nodules) have been reported. They can be episodically painful and while most are sporadic, they can be associated with Brooke–Spiegler syndrome (with trichoepitheliomas, cylindromas, spiradenomas and mixed lesions). Mutations in the *CYLD* gene on chromosome 9 have been associated with their development,¹¹ but spiradenomas also have increased expression of p53. Multiple, giant, linear and grouped forms have all been described. Malignant transformation can occur, particularly in long-standing lesions. Surgical excision is usually curative, but when multiple, radiotherapy and hyperthermic limb perfusion chemotherapy have been used successfully to avoid widespread excision.

Benign dermal tumours

Dermatofibroma

Dermatofibroma (or superficial benign fibrous histiocytoma) is a common cutaneous nodule of the limbs that is typically asymptomatic, but has been reported to be the most common atypical lesion causing pain.¹² Their aetiology remains unknown, but it may be trauma. Their prominent mast cells may release transforming growth factor beta (TGF- β) and other fibrinogenic factors to promote the fibrotic reaction seen histologically.¹³ Syndecan 1 and fibroblast growth factor receptor 2 (FGFR-2) have also been implicated in their formation.^{14,15} Lesions are usually solitary, but may be multiple. When numerous lesions are present (≥ 15) they may be associated with a systemic condition including immune suppression (e.g. HIV), autoimmune conditions (systemic lupus erythematosus (SLE), dermatomyositis, Graves' disease, Hashimoto's thyroiditis, myasthenia gravis), malignancies (leukaemia, myelodysplastic syndrome, cutaneous T-cell lymphoma, multiple myeloma), atopic dermatitis or Down syndrome.¹⁶

Dermatofibromas can be PET avid and may have a similar appearance to many other tumours. Dermoscopy can be useful to display an overlying peripheral pigment network with a central white area.¹⁷ Ultrasound may show a solid hypoechoic nodule. A biopsy is warranted if there is any diagnostic uncertainty. Surgical removal is indicated if lesions are symptomatic, aesthetically unacceptable or when diagnostic uncertainty remains. Excision of the tumour with minimal overlying skin is usually effective in preventing recurrence, but aggressive subtypes (aneurysmal, atypical, cellular, deep/subcutaneous) have a local recurrence rate of up to 20%.¹⁶ Rare cases of metastasis have been described with these variants.

Unusual malignancies of skin

The malignant tumours described in this section are uncommon at best and hence there is a paucity of high-level evidence to guide treatment. Accurate histopathology evaluation is vital and given the complexity and the rarity of these tumours, referral for expert review may be considered. Mohs micrographic surgery has been reported for many of these tumours, particularly in sites where obtaining a wide margin may be problematic. In general, lesions successfully treated by Mohs tend to be smaller and although results appear to be as good as standard wide surgical excision, the number of cases and/or length of follow-up are limited in most reported series. In cases where the completeness of excision is uncertain, DRAPE (delayed reconstruction after pathology evaluation)¹⁸ should be considered.

Merkel cell carcinoma

Merkel cell carcinoma (MCC) is an uncommon aggressive cutaneous malignancy that is increasing in frequency (Figure 11.3). The mnemonic AEIOU has been proposed to summarize the most common clinical characteristics (asymptomatic, expanding

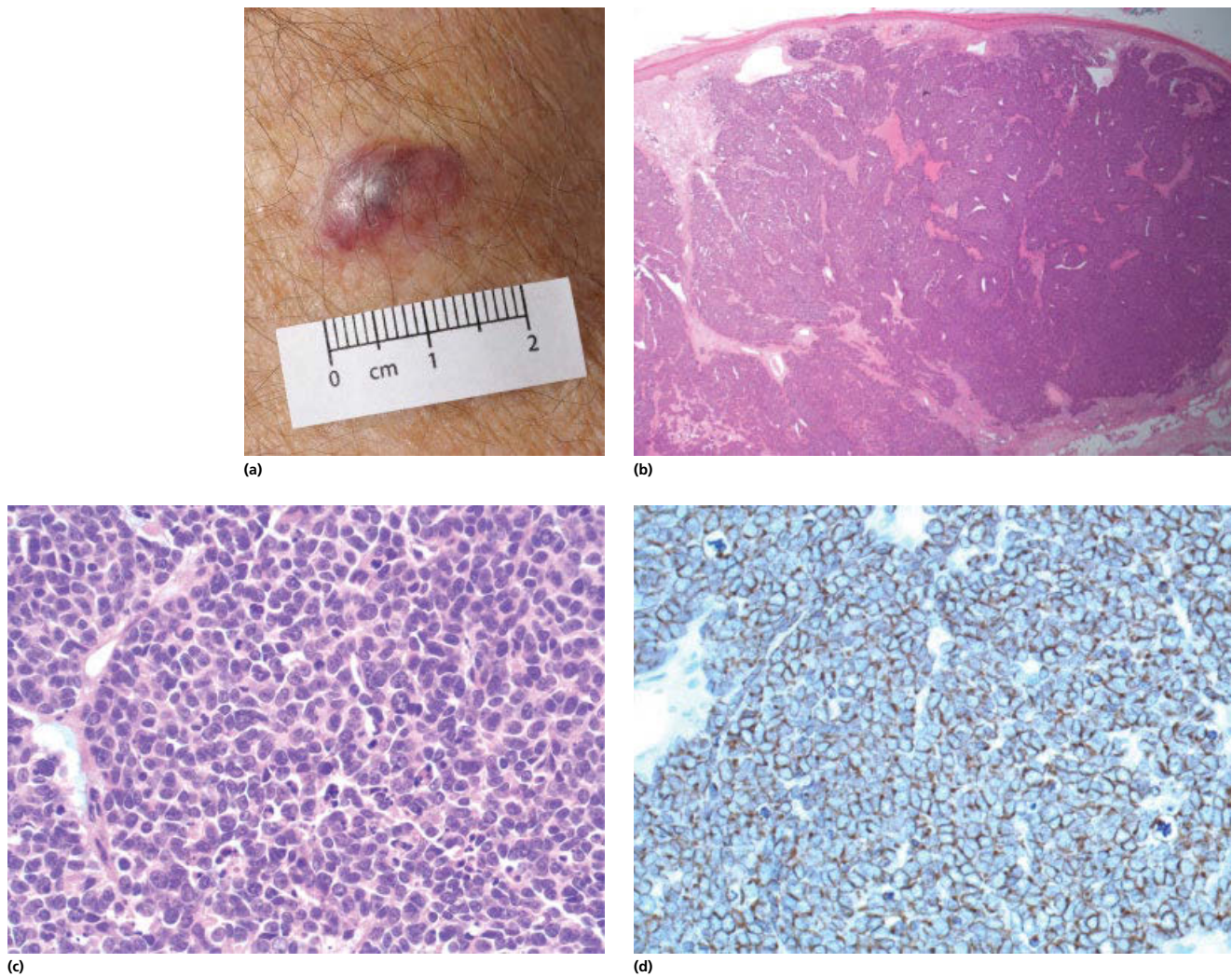


Figure 11.3 (a) Merkel cell carcinoma arising in the anterior lower leg of a 72-year-old man. The lesion is raised and pink/purple in colour. (b) Histopathology showing an intensely basophilic, cellular dermal tumour. (c) Higher power view demonstrates nuclear moulding, minimal cytoplasm, numerous mitoses and apoptosis consistent with a 'small cell undifferentiated malignancy'. (d) Cytokeratin 20 (CK20) immunolabelling shows the perinuclear 'dot' of the Golgi apparatus and nuclear membrane.

rapidly, immune suppression, older than 50 years and UV-exposed site on fair skin).¹⁹ Correspondingly, the lesion affects the sun-exposed areas of elderly males, is related to chronic UV exposure and immunosuppressed patients are overrepresented. The cell of origin is unknown but is unlikely to be the Merkel cell, a mechanoreceptor found in skin. Almost all cases are associated with Merkel cell polyomavirus. The characteristic presentation is a rapidly growing firm non-tender flesh-coloured nodule. The most common site is the face (25%); the head and neck, trunk and limbs account for the remainder.¹⁹

The histological features are of a neuroendocrine tumour with three main patterns. Most common is the solid type, a densely blue (basophilic) tumour with paving of rapidly dividing tumour cells. The trabecular (cells arranged in cords or columns of invasive basophilic cells) and diffuse (poor cellular cohesion reminiscent of lymphoma-like tissue growth)

patterns are less common. The potential for coexistence of MCC within BCC or SCC must always be considered and can be especially difficult to diagnose in desmoplastic non-melanoma skin cancer variants. The diagnosis is confirmed by immunohistochemical labelling with cytokeratin 20 (perinuclear Golgi dot).

Staging of MCC

Prior to 2010, there were at least five competing staging systems in common use for MCC, limiting comparisons between centres worldwide. In 2010, the American Joint Committee on Cancer (AJCC) published a revised TNM staging system focusing on the size and degree of invasion of the primary tumour, whether micro, macro or in-transit disease is present regionally and the presence and site of distant metastases (Table 11.1). Preoperative staging should include fine-slice CT scans of the chest, abdomen

Table 11.1 American Joint Committee on Cancer staging for Merkel cell carcinoma²¹

T Staging for MCC	
Tx	Primary tumour cannot be assessed
T0	No evidence of primary tumour (e.g. metastasis with unknown primary)
Tis	<i>In situ</i> primary tumour
T1	≤2 cm maximal tumour dimension
T2	>2 cm but ≤5 cm maximum tumour dimension
T3	>5 cm maximum tumour dimension
T4	Primary tumour invades bone, muscle, fascia or cartilage
N Staging for MCC	
Nx	Regional lymph nodes cannot be assessed
N0	No regional node metastases or in-transit metastases
cN0	Nodes negative by clinical exam (no pathology nodal exam performed)
pN0	Nodes negative by pathology exam
N1	Metastases in regional lymph nodes
N1a	Micrometastasis
N1b	Macrometastasis
N2	In-transit metastasis
M Staging for MCC	
M0	No distant metastases
M1	Metastases beyond regional lymph nodes
M1a	Metastases to skin, subcutaneous tissues or distant lymph nodes
M1b	Metastases to lung
M1c	Metastases to all other visceral sites
Stage by TNM for MCC	
0	Tis, N0, M0
IA	T1, pN0, M0
IB	T1, cN0, M0
IIA	T2/T3, pN0, M0
IIB	T2/T3, cN0, M0
IIC	T4, N0, M0
IIIA	Any T, N1a, M0
IIIB	Any T, N1b/N2, M0
IV	Any T, Any N, M1

and regional lymph node fields with PET scanning if available. Not all MCCs are PET avid, especially if already irradiated.²⁰

Other poor prognostic indicators include age >70 years, male sex, truncal or head and neck site (especially lip), nodal or distant disease at presentation and adverse histopathology features (small cell size, infiltrative pattern, high mitotic rate, depth of invasion and lymphovascular invasion).²²

Management

Management remains controversial, particularly regarding the primary tumour. MCCs are highly sensitive to radiotherapy but current management recommendations are based on relatively small case series. Larger series typically suffer from a lack of uniform staging, treatment or follow-up. In general, surgical excision is favoured for small lesions in favourable sites. For larger lesions radiotherapy alone to the primary site may be more

appropriate. If concern remains about the completeness of margins after definitive surgery, adjuvant radiotherapy is indicated. MCCs demonstrate a marked propensity for lymph node spread, therefore sentinel node biopsy is recommended by many authorities, but the indications for its use remain unclear and significant false negative rates have been reported. Prophylactic management of the nodal basin is associated with significantly reduced regional recurrence.²³ The role for sentinel node biopsy is to identify the appropriate nodal basin and select patients for nodal irradiation or completion lymphadenectomy, which provide equivalent regional control. For tumours less than 10 mm in size the risk of lymph node involvement is less than 10%. For lesions greater than 20 mm the risk may be 40% or higher.²⁴ Sentinel node biopsy is reasonable for lesions larger than 10 mm although for smaller lesions, it may be justified where the tumour is thick, has lympho-vascular invasion, an elevated mitotic rate or the patient is young. Given the significant risk of developing distant disease in patients with lymph node involvement or tumours greater than 20 mm in size, adjuvant systemic chemotherapy (carboplatin and etoposide) concurrent with radiotherapy may be indicated, however many patients are elderly and therefore potentially unfit for such therapy.²⁵

Most recurrences occur within 2 years of diagnosis. Locoregional recurrence is not uncommon. Palliative chemoradiotherapy may have some benefit for locoregional and distant recurrence most commonly seen in liver, bone, lung and soft tissue.

Adnexal carcinomas

Malignant neoplasms of the pilosebaceous unit are very rare. They are classified with a bewildering array of terms, many of which are revised on a regular basis. The more common types include microcystic adnexal carcinoma, sebaceous carcinoma and porocarcinoma. Overall survival is approximately 70% at 5 years.^{26,27}

Microcystic adnexal carcinoma

Microcystic adnexal carcinoma (MAC) is an infiltrative tumour usually of the head and neck (particularly central face) in older persons. The presentation is usually innocuous as a slow-growing skin-coloured plaque. Wide excision is indicated as the extent of the tumour is often considerably more than the clinical examination would suggest. Properly managed, local regional or distant recurrence is rare. Radiotherapy may have a role when surgical excision is compromised or not possible.²⁸

Sebaceous carcinoma

This occurs predominantly in the head and neck in older patients with a predilection for the eyelid. If adequately excised, recurrence is rare.

Mohs surgery may have a role in managing this tumour, given its common involvement of the eyelid, but as with other unusual cutaneous malignancies, the evidence for its use is largely case series with few patients and/or relatively short follow-up.

Porocarcinoma

This arises not infrequently from a long-standing benign apocrine/eccrine poroma. Most involve the lower limbs in elderly persons as slow-growing lesions that increase their growth rate just prior to diagnosis. Lymph node involvement occurs infrequently. Sentinel node biopsy has been reported, however its use is still undefined. With wide excision, long-term survival is reasonable.²⁹

Extramammary Paget's disease

This is a rare tumour classified as an adenocarcinoma of apocrine glands but considerable debate remains about the cell of origin. The classic site is the perineum (vulva, penis, scrotum and perianal area). More rarely, the axilla and eyelid are affected. Up to one third of affected patients have an occult malignancy

(bladder, prostate, kidney, colon and rectum). Approximately two thirds of patients have *in situ* disease only (Figure 11.4).

The typical history is of a slowly progressive lesion (often mistaken for eczema or similar rash) in patients over 70 years. The disease is frequently extensive at diagnosis and may be multifocal. Careful examination, combined with punch biopsies to confirm the extent of the disease and the presence/absence of invasion are warranted for larger lesions. Exclusion of an underlying primary malignancy is appropriate.³⁰

Wide surgical excision with clear margins is the treatment of choice. Local recurrence may occur in up to 50% of patients. Lymph node involvement occurs in up to two thirds of patients with invasive disease. Sentinel node biopsy has been reported but its role is undefined. Lymph node involvement, dermal involvement and lymphatic and/or vascular invasion are poor

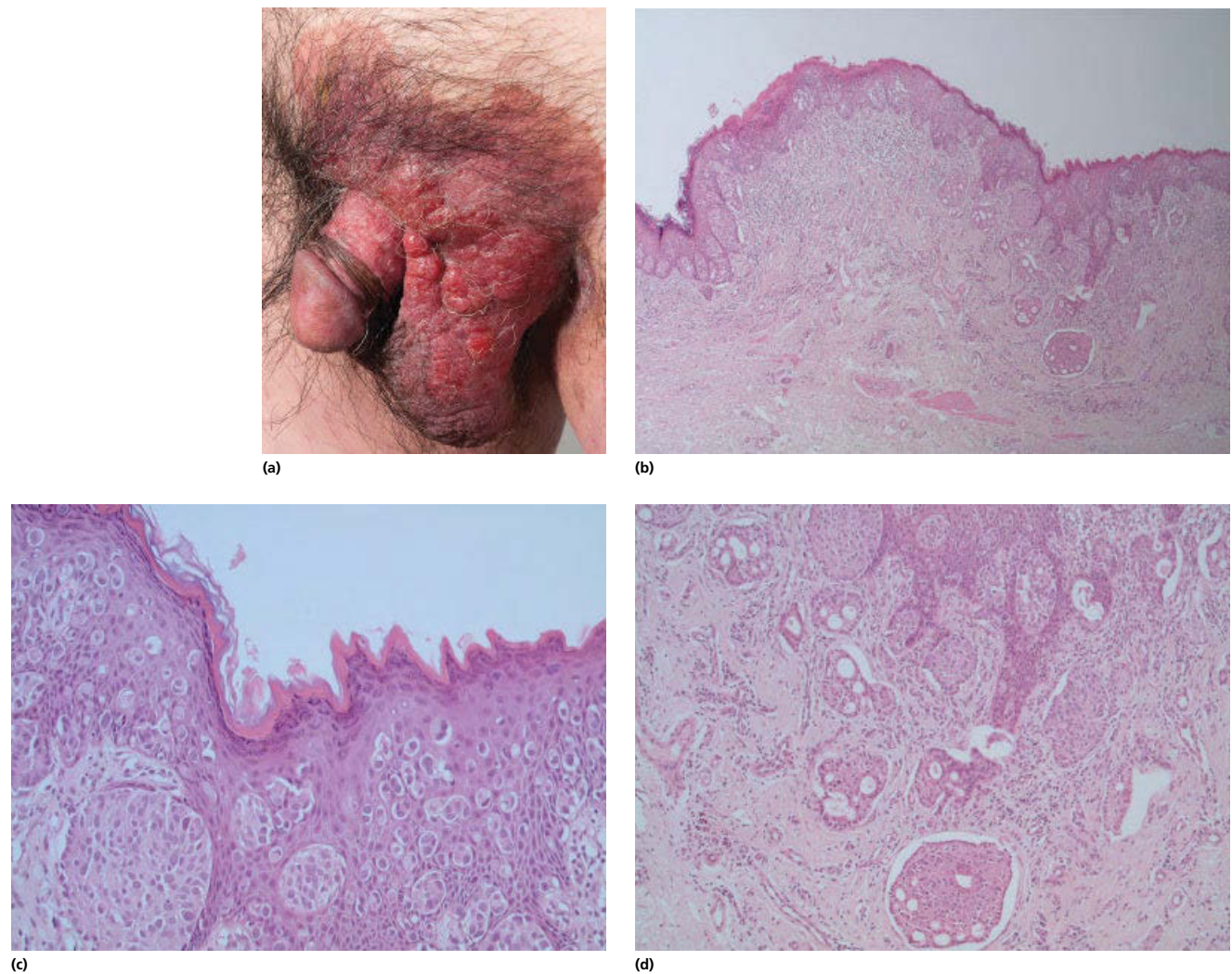


Figure 11.4 (a) Extensive extramammary Paget's disease (EMPD) with invasive adenocarcinoma developing in a 71-year-old male. The patient had a 'rash' for over 7 years. Extensive work-up failed to disclose any occult malignancy. (b) Histopathology demonstrates nested and single malignant cells in the epidermis, with malignant glands in the upper dermis (bottom right). (c) Nested and single malignant cells in 'Pagetoid' distribution in the epidermis. (d) Invasive adenocarcinoma (bottom) with *in situ* EMPD in the epidermis above.

prognostic factors. The role of radiotherapy is controversial. Radiotherapy as monotherapy may be appropriate when surgery is contraindicated however it is not clear if it is as effective as surgery. The literature is also unclear as to whether adjuvant radiotherapy after complete surgical excision offers major benefits. In patients with extensive *in situ* disease, topical imiquimod or photodynamic therapy have been used with relative success.

Overall survival at 5 years is 60% for patients with invasive disease. Long-term follow-up is recommended due to the significant risk of locoregional recurrence.

Other rare adnexal carcinomas

- Pilomatrix carcinoma occurs in the head and neck of females, probably from a benign pilomatrixoma. Wide excision is indicated.
- Malignant chondroid syringoma (malignant mixed tumour) occurs on the extremities and trunk, following an unpredictable course with spread to regional lymph nodes and distant sites. Wide excision is indicated.
- Hidradenocarcinoma occurs in the head, neck and extremities of elderly patients and pursues an aggressive course with regional and distant metastasis.
- Malignant cylindroma is a very rare tumour, probably arising from a benign cylindroma in older patients, which frequently metastasizes widely.
- Eccrine spiradenocarcinoma develops in elderly patients, usually from a pre-existing benign spiradenoma. Wide excision is usually curative as lesions rarely metastasize.

Soft tissue sarcomas of skin

Tumours of mesenchymal origin arising in skin account for less than 5% of all soft tissue sarcomas. Excluding Kaposi's sarcoma, dermatofibrosarcoma protuberans (DFSP) accounts for two thirds of cases. Leiomyosarcoma of the skin and cutaneous angiosarcoma account for an additional 20%. The remainder include fibrosarcomas, myxosarcomas and atypical fibroxanthoma (AFX). In general, these lesions are characterized as having low malignant potential with a significant risk of local recurrence.³¹ They present as small, innocuous-appearing, slowly growing skin lesions. Invariably the diagnosis is made retrospectively following biopsy. Lesions previously described as malignant fibrous histiocytoma (MFH) have been subjected to considerable revision in recent years. In many cases they have been reclassified using an alternative nomenclature – unclassified pleomorphic sarcoma (UPS). UPS is considered by many to be a final common phenotype for various undifferentiated tumours and therefore is reserved as a diagnosis of exclusion.³² Management of these lesions will not be considered further as they behave in a far more aggressive fashion as with MFH/UPS elsewhere. Wide resection with adjuvant radiotherapy is the usual primary management.

Dermatofibrosarcoma protuberans

Dermatofibrosarcoma protuberans (DFSP) is an uncommon, dermally derived tumour³¹ with a marked tendency to local recurrence (Figure 11.5). Haematogenous spread is uncommon

(<5% of cases) and lymphatic spread is rare. Approximately 90% of tumours are low grade, however the remaining 10% display higher grade sarcomatous transformation (most commonly fibrosarcoma). DFSP affects both sexes equally and is typically a tumour of the third and fourth decades, although paediatric cases have been described. Nearly half the tumours arise on the trunk, the proximal limbs approximately 40% and the head and neck accounts for the remaining 10–15%.³³

Most present as small, slow-growing, innocuous, indurated, skin-coloured plaques that may become raised, pink and eventually violaceous with telangiectasia over time. The lesion is attached to skin and advanced cases demonstrate the characteristic cutaneous nodules that give the tumour its name. Advanced lesions may ulcerate, bleed and become fixed to underlying structures. The more malignant form of DFSP, with fibrosarcomatous transformation presents in an identical fashion.³⁴ A less aggressive variant, the Bednar tumour more commonly seen in Black persons is an uncommon, pigmented form of DFSP (5% or less) where scattered melanosome-containing cells are seen in lesions that are otherwise indistinguishable from standard DFSP by histopathology. They have a slightly better prognosis than standard DFSP.

DFSP demonstrates a monotonous population of spindle cells arranged in a typical storiform pattern with bland-appearing tumour projections invading away from the major tumour, which can be difficult to identify histologically and presumably explains the high rate of local recurrence seen following limited excision margins. In most cases the diagnosis is straightforward with routine histology. Immunohistochemistry is useful to distinguish DFSP from other lesions but dermatofibroma, melanoma, leiomyoma and leiomyosarcoma and atypical fibroxanthoma cannot always be excluded. Over 85% of DFSPs label with CD34 but not factor XIIIa (labels the majority of dermatofibromas), vimentin or S-100, however DFSP with fibrosarcomatous transformation labels with CD34 in 50% of cases.

DFSP cells are characterized genetically by either a supernumerary chromosomal ring or linear translocation t(17:22)(q22;q13) in over 90% of cases. This alteration results in a fusion product involving platelet-derived growth factor B (PDGF-B) and the collagen 1A1 gene (*COL1A1*). This fusion protein leads to constitutive activation of the PDGF receptor resulting in cell growth.

The risk of distant or regional lymph node metastasis at presentation is very low, therefore preoperative imaging should be confined to evaluation of the primary tumour site by either MRI or CT scan. Planned margins of 2–4 cm usually result in tumour clearance and an acceptable rate of local recurrence (approximately 5% compared to rates >50% with incomplete/close margins).

DFSP with fibrosarcomatous change is reported to have a higher rate of local (38%) and distant (15% versus <5%) recurrence. In over 80% of cases involving fibrosarcomatous change, distant recurrence follows local recurrence, emphasizing the need for complete surgical excision.^{35,37} Even with wide margins of excision, recurrent lesions have a subsequently higher rate of local recurrence (11%).

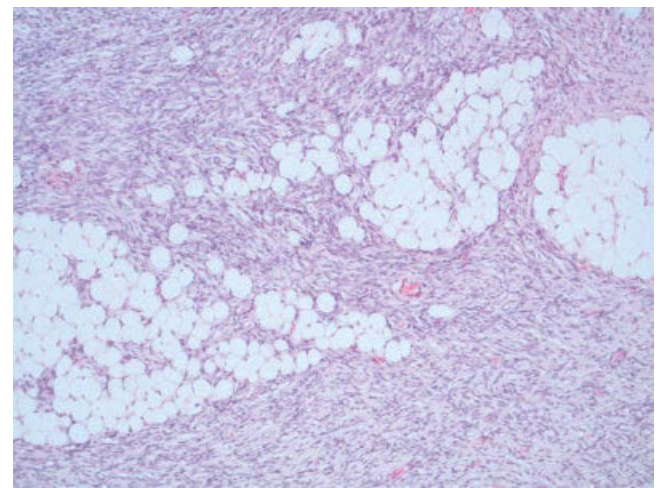
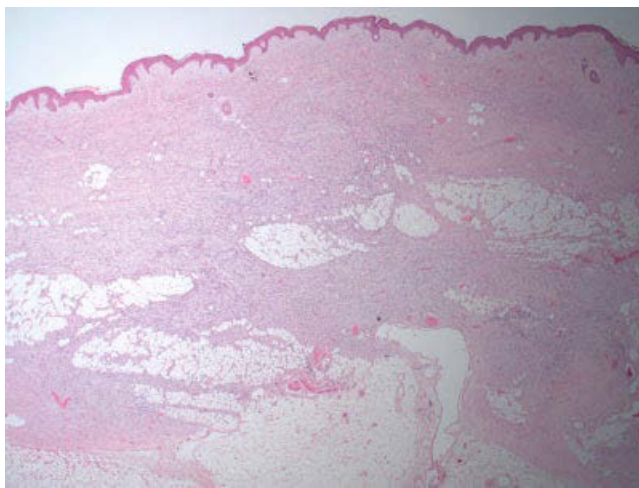


Figure 11.5 (a) Recurrent dermatofibrosarcoma protuberans (DFSP) in a 44-year-old female. Typical pink thickened appearance seen in the initial stages. (b) Histopathology demonstrating cellular dermal and subcutaneous spindle cell proliferation with occasional storiform patterns (irregular whorl-like shapes) evident. Tumour is centred on the subcutis and appears to 'lift' subcutis into dermis. (c) Bland spindle cells wrap around and entrap subcutaneous lobules. Note the low-grade cytology.

DFSP is relatively radiosensitive but the role of adjuvant radiotherapy is currently undefined. In principle it should be reserved for cases where a complete excision is not possible or with extensive tumours. Specifically, patients with close margins should be managed by further excision if possible, rather than adjuvant radiotherapy. Targeted therapies to address the constitutive activation of PDGF-B in DFSP are currently being assessed. Initial reports of imatinib (a tyrosine kinase inhibitor that inhibits the PDGF receptor) for advanced or recurrent disease indicate a response rate approaching 50%. It may also have a preoperative role to improve tumour resectability.

Most recurrences occur within 3 years, however very late recurrences also occur. Follow-up by close physical examination and imaging of the primary site should be concentrated in the first 3 years. Ongoing annual follow-up after that time would seem reasonable.

Atypical fibroxanthoma

Atypical fibroxanthoma (AFX) is a rare and controversial tumour. Since first described over four decades ago, AFX is now recognized as a tumour of intermediate malignant potential that rarely metastasizes. It has considerable histological similarities to MFH, now classified as undifferentiated pleomorphic sarcoma (UPS). The majority of pathologists recognize both entities however at times it may be difficult to distinguish between them. UPS has significantly higher rates of local and distant recurrence and mandates more aggressive management. The risk of metastasis with AFX is very low (<5%) and some authorities argue that metastasizing AFX should be reclassified as UPS.

AFXs occur predominantly in the head and neck (80%) and/or sun-exposed areas of elderly males (median age 70). AFX of the limbs occurs in younger patients (median age 40). The histological appearances can be varied and the diagnosis is essentially

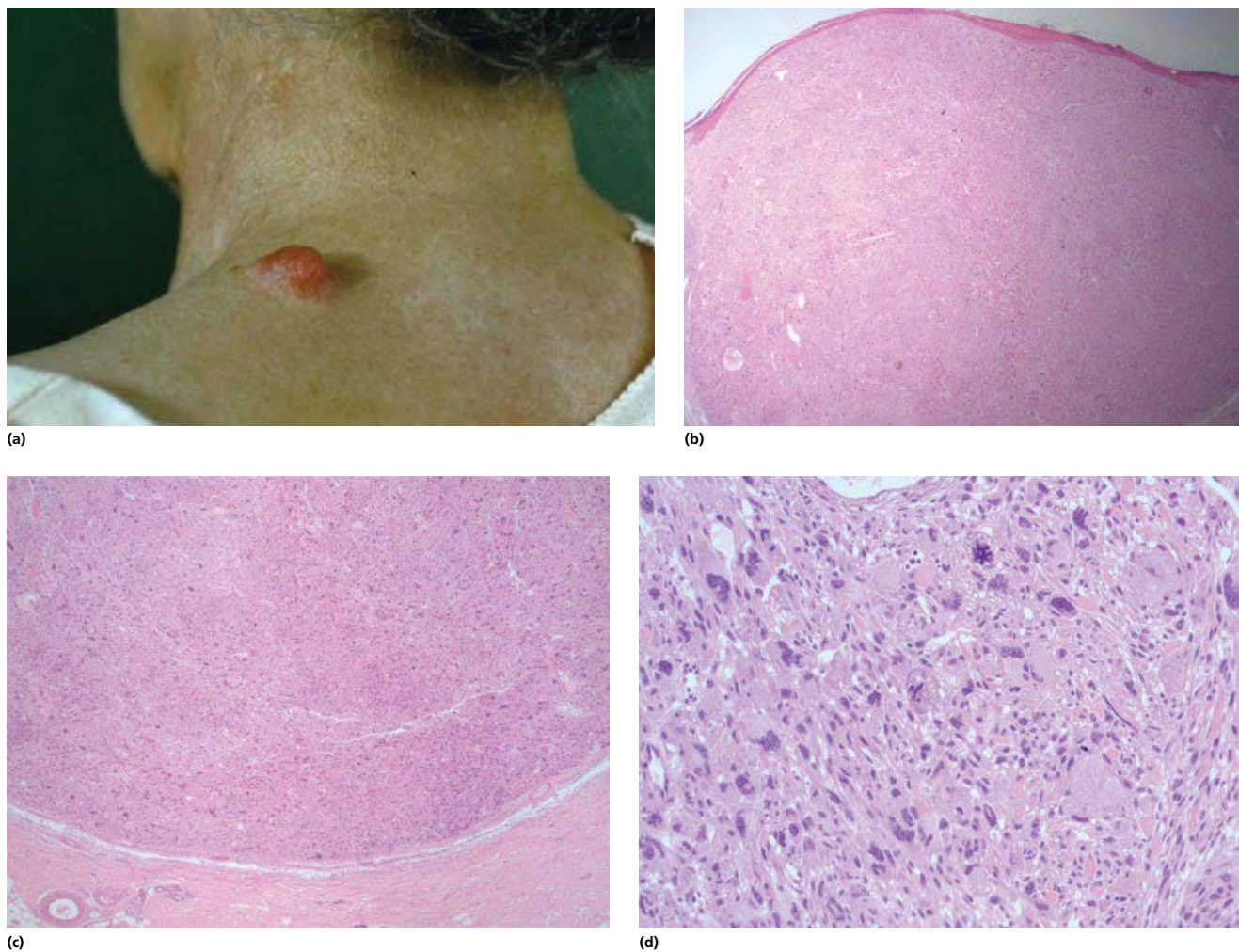


Figure 11.6 (a) Atypical fibroxanthoma (AFX) in a 65-year-old female. A raised pink lesion had grown slowly over at least 12 months. (b) Histopathology demonstrating a cellular dermal spindle cell tumour with attenuated overlying epidermis. (c) High-grade cytology with 'pushing' appearance and circumscribed deep border in deep dermis (not subcutis). (d) AFX usually appears as a high-grade spindle cell malignancy, with tumour giant cells and atypical mitoses that are out of proportion to low-grade clinical behavior.

one of exclusion. Immunohistochemistry may be of value in differentiating lesions such as melanoma or SCC (Figure 11.6).

Management is by wide excision (1 cm or greater) but there is no evidence on which to base margins. Local recurrence is uncommon (<10%) with adequate margins of excision and most recurrences occur in the first 2 years. Invasion beyond the dermis and local recurrence are associated with increased risk of both local and distant recurrence.³⁸

Cutaneous leiomyosarcoma

Leiomyosarcomas of skin are an uncommon group of tumours that can be further classified as superficial (cutaneous or dermal) or subcutaneous. The average age of onset is 40 with no gender bias. Over half present as innocuous, slow-growing nodules in the head and neck with the remainder in the extremities. Cutaneous leiomyosarcomata are relatively innocuous low-grade lesions and probably do not metastasize. Local recurrence is rare

following wide excision. Some suggest that they should not be regarded as malignant. Subcutaneous leiomyosarcoma has a higher rate of local recurrence and up to one third may metastasize.³⁹ There is little objective evidence on which to base surgical management. Margins of 2–5 cm have been reported but the critical issue is completeness of histological margins.

Cutaneous angiosarcoma

This is a heterogeneous condition that may develop in skin, the breast or in irradiated or chronically lymphoedematous tissue and in visceral sites. The most common (>75%) cutaneous site is the head and neck (particularly scalp). Most patients are male with a median age in the 70s. Most cutaneous angiosarcomas present as a small bruise becoming irregular, slightly raised with multifocal macules and papules that are purplish in colour (Figure 11.7). One third of patients have evidence of metastatic disease at presentation and work-up with

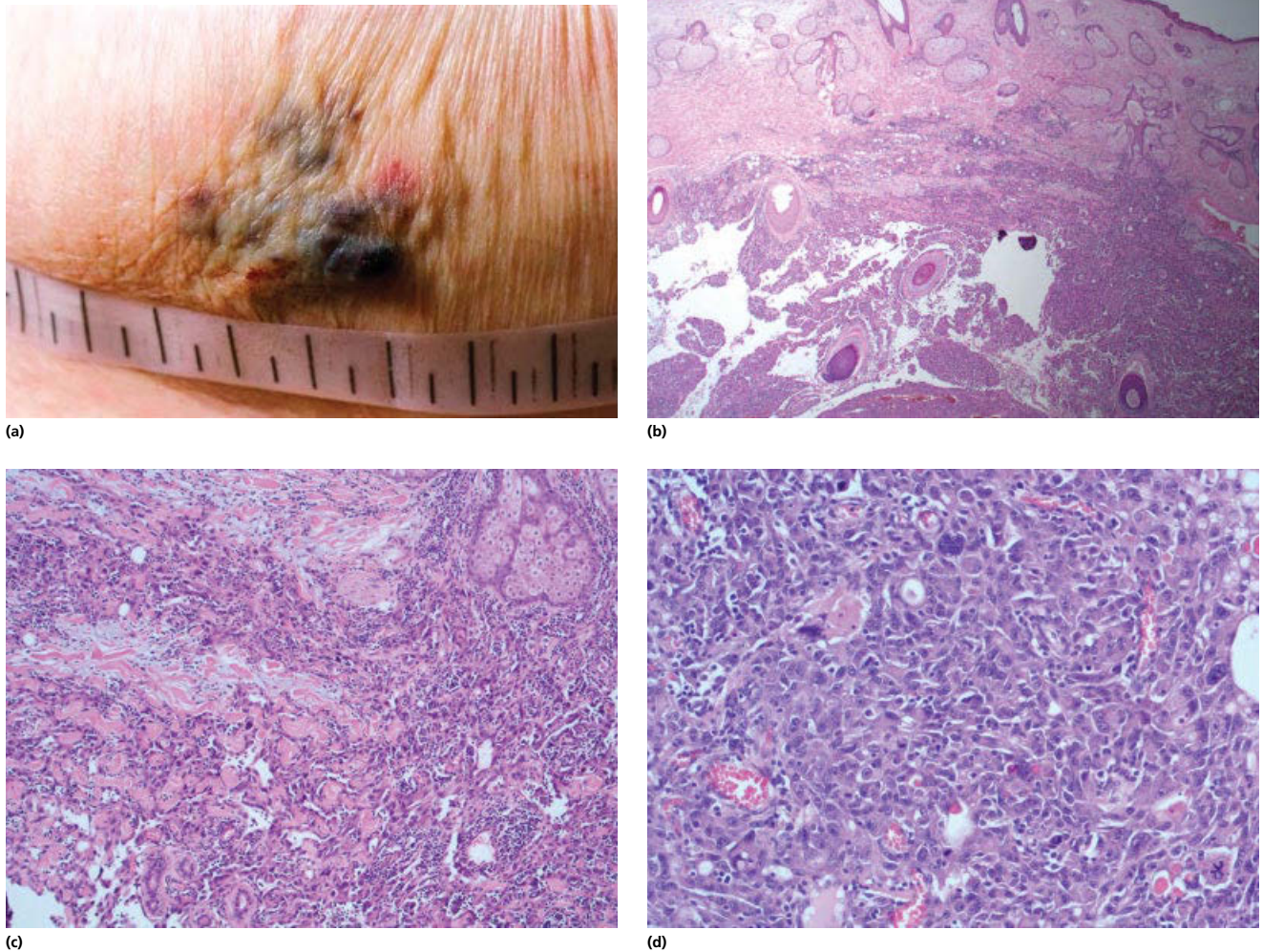


Figure 11.7 Typical appearance of an angiosarcoma developing in a 77-year-old woman. (a) The lesion is purple in colour and multifocal in appearance.

fine-cut CT chest and liver and whole-body PET scanning where available, is appropriate. Very wide excision is indicated, but angiosarcoma is frequently multifocal, leading to a high (50%) local recurrence rate despite clear margins. Wide-field adjuvant radiotherapy incorporating draining lymph node fields reduces the risk of local recurrence and anecdotal evidence suggests it may prolong survival.⁴⁰ The role of sentinel node biopsy is undefined. Most recurrences (local, regional and/or distant) occur within 2–3 years. The 10-year survival is approximately 20–30%.

Summary

The correct diagnosis and management of atypical tumours in clinical practice can be a daunting prospect. Revisions to tumour names, classifications and the emergence of mutational profiling has added to the challenge of managing patients with

these lesions. In general, atypical lesions are a testament to the potential for any cell or stem cell type to develop benign tumours or to undergo malignant transformation. Although the text is not exhaustive, we have sought to cover some of the more important and clinically relevant lesions. These are provided so that the clinician will have sufficient information so as to critically appraise any histopathology diagnosis provided and thereby ensure the appropriate management of their patient. More common tumours such as melanoma are capable of mimicry and therefore the diagnosis of melanoma or other common mimics should always be entertained when there is any discrepancy between the clinical picture and the results of histopathology. The appropriate management of malignant atypical lesions may require a dedicated multidisciplinary team within a tertiary or quaternary health centre. Careful consideration should be given to referring patients with atypical tumours that are at significant risk of recurrence and/or metastasis.

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CHAPTER 12

Allotransplantation

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Historical background

Vascularized composite allotransplantation (VCA) involves transplantation of a vascularized graft, which is derived from different embryogenic origins and composed of a variety of heterogeneous antigenic tissues, across a genetic mismatch.¹

Trying to replace lost limbs and other organs is an old desire of humanity. The first historical mention of VCA dates back to Saints Cosmas and Damian (AD 286), who transferred an Ethiopian Moor's limb to a churchman.² Later, successful solid organ transplantations constituted the initial milestones of VCA. The first successful human allograft was performed in 1957 by E. Peacock. The allograft was composed of an en-block digital flexor tendon mechanism. However, this transplantation did not contain skin.³ In the 1960s, immunosuppression was limited, which led to the unfortunate rejection of the world's first hand transplant attempted by Robert Gilbert in 1964. Two weeks after the surgery, the graft was explanted and re-amputation was performed.¹ This complication delayed the next clinical hand transplantation, but studies about immunosuppression and experimental models continued to evolve to solve the problem. Once the clinical efficacy of cyclosporin (CsA) had been demonstrated in solid organ transplantation, studies began performing limb allograft transplantation in rats, dogs and rabbits. Hewitt reported the first successful rat limb allotransplantation in 1985.⁴ He also showed that transplanted bone marrow was functional and caused immune chimerism.⁵ During the same time period, Achauer mentioned the potential use of allografts for facial reconstruction in 1985.⁶ The first successful face transplantation in rats was performed by Siemionow and colleagues.⁷ Later, many models of VCA were developed and reported in both small and large animals.⁸ Pharmacologic research continued during this time and new drugs were developed for immunosuppression. In 2003, Siemionow reported the first and the only rat transplant model of life-long donor-specific tolerance using a 7-day protocol of anti-alpha/beta T-cell receptor monoclonal antibody and CsA.⁹

During the last 50 years, various clinical and experimental composite tissue allotransplantations have been performed, including: limb, trachea, flexor tendon apparatus, penis, nerve, knee, hand, face and abdominal wall.¹⁰

Nomenclature

An *autograft* is a tissue that is transplanted from one location to another in the same individual. When the graft is transplanted from a genetically identical donor, such as transplants between syngeneic animals and human monozygotic twins, it is called an *isograft*. An *allograft* is transplantation of tissues between genetically different individuals of the same species. *Xenograft* is transplantation between different species. Transplantation may also be described in terms of the site where the tissue is transplanted. *Orthotopic* transplantation means the recipient site is anatomically similar site and *heterotopic* transplantation means the recipient site is different from the site of origin.

Despite the encouraging results, debate continues over the immunosuppression-related risks. Thus, the ultimate goal of allotransplantation is to obtain *transplant tolerance*, which is rejection-free acceptance of transplanted organs or tissues without need for long-term immunosuppression. *Donor-specific tolerance* is defined as the state of donor-specific hyporesponsiveness in the recipient in the absence of immunosuppression. The state of tolerance to one organ or tissue (such as tendon) while simultaneously rejecting another (such as skin) is called *split tolerance*.¹¹ One of the best methods of obtaining tolerance is haematopoietic stem cell chimerism. In *chimerism*, tissues from two genetically different organisms coexist in one organism. There are two types of chimerism: *macrochimerism* and *microchimerism*. Macrochimerism occurs when bone marrow is transplanted to a conditioned patient, engrafted and produces all lineages. Microchimerism arises from the migration of donor leukocytes from the allograft to the recipient and in microchimerism donor haematopoietic stem cells do not engraft, but are produced and migrated systemically.^{12,13}

Basic transplantation immunology

The most important antigens contributing to allograft rejection are the major histocompatibility complex (MHC) antigens. The MHC proteins are encoded in chromosomes and have different nomenclature between species: HLA in humans, SLA in swine and RT1 in rat. There are two major classes of MHC antigens. Class I antigens are expressed on nearly all nucleated cells and serve as the primary target for cytotoxic (CD8+) T lymphocytes. Class II antigens are expressed primarily on vascular endothelium, antigen-presenting cells (APCs), B cells and activated T cells. Both class I and class II molecules have a specific site at which foreign peptide antigens can be presented after they have been processed by the cell. Following allotransplantation, foreign antigens are recognized by host T cells after being processed by host APCs. This is called indirect presentation. In addition, host cells can recognize donor MHC antigens, which is called direct presentation.¹⁴

The allograft rejection or allorecognition is primarily performed by T lymphocytes. The T-cell lineage marker is called TCR and there are two different types of T cells: $\alpha\beta$ T cells, which are responsible for the allograft rejection, and $\gamma\delta$ T cells, which are responsible for infections and antitumor activity. Mature $\alpha\beta$ T cells are subdivided into two groups: *T helpers* induce immune response and are CD4 positive, whereas *cytotoxic T cells* are responsible for cytotoxicity and are CD8 positive. Following allotransplantation, rejection begins with activation of T cells. The first step of T-cell activation is recognition of foreign antigen by T cells (signal 1). The second step is activation of co-stimulatory signals (signal 2). The third step is T-cell differentiation, which is performed by secretion of interleukin 2 following co-stimulatory signals (signal 3). Activated T cells are responsible for cell-mediated rejection of the allograft.¹⁵

The types of allograft rejection may occur and may be regulated by different mechanisms. Hyperacute rejection occurs within a few minutes after transplantation. This immune response is an antibody-mediated rejection process with the most frequent reason presensitized antibodies against MHC antigens. Acute allograft rejection is a cell-mediated immune response and is observed in all composite tissue allotransplantation cases. It is primarily attributed to T-cell activation. There is not enough information about antibody-mediated rejection (AMR) in VCA, but vascular rejection in solid organs is attributed to AMR. Chronic rejection is a common characteristic of solid organ transplantation; however there currently are insufficient data available to define the specific changes of chronic rejection in a VCA. It is characterized by fibrotic changes in the allograft, which may be caused by non-immunogenic factors.

In order to define and classify rejection, various classification systems have been developed. The most widely accepted classification system is the Banff Classification. In this system, the defining features used to diagnose acute skin rejection include: inflammatory cell infiltration with involvement of

epidermis and/or adnexal structures, epithelial apoptosis, dyskeratosis and necrosis. This classification system provides proposed schemas and represents the international consensus on this topic.^{15,16}

Transplant immunology is the most important area of research in VCA and many current studies are attempting to increase our knowledge of rejection mechanisms, routine immunosuppressive regimes, dosages and immune tolerance-inducing strategies. Today, the only method of graft rejection prevention is chronic immunosuppression. Unfortunately, there are several drugs and protocols, which are used experimentally and clinically. However, the ultimate goal of transplant immunology is to provide donor-specific tolerance.

The practice of immunosuppressive administration can currently be divided into three phases: induction, maintenance and rescue therapies. Induction immunotherapy is used as a means to overcome the ischaemic injury and surgical trauma an allograft undergoes during the transplant process. Maintenance therapy is defined as immune suppression that is typically much lower in dose than induction therapy and therefore applicable in a chronic setting. Rescue therapy is similar to induction therapy in that it employs high-dose immune suppression and is usually instigated in response to a rejection episode.¹⁵

Immunosuppressive therapy has well-documented side effects which may give rise to conditions that shorten life. The incidence and severity of such side effects are well described and this would allow an informed decision to be made as to whether these outweigh the potential benefit from VCA.

Experimental models of allotransplantation

Experimental VCA models help us to determine the technical feasibility of various immunosuppression and immune tolerance strategies and to evaluate their effects.

Animal models

During the last 20 years, many VCA models were developed for testing the feasibility of transplantation of different body components as well as for testing the effects of various immunosuppressive protocols. The experimental models can be divided into two main categories, namely small and large animal models, including non-human primate models. Another classification of experimental models depends on the type and content of the transplanted allograft:

- Skin-containing VCA models (groin flap, abdominal wall, etc.)
- Hindlimb allograft transplantation models
- Immunomodulatory VCA models (vascularized bone marrow, thymus)
- Facial allograft transplantation models (full face, hemiface, midface, etc.).

The basic experimental models and their specific features are summarized in Table 12.1.

Skin and soft tissue VCA models

These models include allografts containing skin, subcutaneous tissue, fat and muscle (Figure 12.1). The most frequently used skin-containing VCA model is the groin flap. This flap is also one of the first models used in allotransplantation research.¹⁷ This model contains skin, fat and lymphoid tissue harvested on superficial epigastric vessels or femoral vessels. The groin flap is frequently used in immunologic studies as it is technically simple and has low mortality. However, a disadvantage stems from the small size of the skin paddle. Other soft tissue flaps include both skin and various muscles such as the semimembranosus the pectoralis flaps.^{18,19} Another soft tissue model is the total abdominal wall transplantation, in which the entire abdominal wall skin is harvested on bilateral femoral vessels. This model was developed to investigate the effect of skin

amount on development of chimerism.²⁰ Figure 12.1 shows the skin and soft tissue models of VCA.

Hindlimb allotransplantation models

The rat hindlimb transplantation model was described in 1978.²¹ In this model, a circumferential skin incision at the mid-thigh level is made on the donor and superficial epigastric vessels, femoral vessels saphenous nerve and femoral nerve are dissected and thigh muscles are sharply divided. During muscle transection, the sciatic nerve is dissected. Next, the femoral bone is cut with a saw. The recipient is prepared using the same technique. Following division of the donor femoral vessels the limb is transferred to the recipient. First, the femoral bone is fixed with an intramedullary rod and wire suture. The femoral vessels are subsequently anastomosed and the femoral, sciatic and saphenous

Table 12.1 Experimental models of vascularized composite allotransplantation (VCA) and their specific features

	Experimental model	Tissues included, specific features
Skin-containing VCA models	Groin flap as vascularized skin transplant model	Skin, fat and lymphoid tissue. Simple, used in immunologic studies, small skin component
	Semimembranosus muscle and epigastric skin transplant model	Skin, fat, muscle and lymphoid tissue. Used for microcirculatory, physiologic and immunologic studies
Hindlimb models	Total abdominal wall transplant model	Skin, fat and lymphoid tissue. Contains large skin component
	Rat hindlimb transplant model	Popular, but technically challenging animal model containing all tissue components including vascularized bone marrow
Immunomodulatory VCA models	Groin–thigh osteomyocutaneous transplant model	Alternative model to hind limb transplant less challenging technique, low mortality
	Vascularized femur transplant model	Femoral bone, bone marrow, cartilage and muscle, applied for bone marrow-induced chimerism studies
	Bilateral vascularized femoral bone transplant model	Larger bone marrow content, applied for tolerance induction studies
	Composite vascularized skin/bone transplant model	Combination of groin flap with vascularized femur transplantation, enables follow-up of rejection
	Vascularized osteomyocutaneous iliac transplant model	Iliac bone, bone marrow, abdominal wall musculature and wide skin island
	Vascularized sternum transplant model	Sternum, skin and muscles. Contains bone marrow
	Thymus transplant model	Used for evaluation of effect of thymus on tolerance induction
	Osseomusculocutaneous sternum, ribs, thymus, pectoralis muscles and skin transplant	Includes all three important immunologic tissues (bone marrow, thymus and skin) for immunology and tolerance studies
	Full face/scalp transplant model	Includes ear, neck and facial skin without eyelids and nose; high mortality
	Hemiface/scalp transplant model	Unilateral ear, neck and facial skin. Used for immunologic studies in facial transplantation
Facial allograft models	Hemiface calvaria tarsnplant model	Temporal muscle and parietal bone included to hemiface scalp model. Contains bone marrow
	Hemiface mandible tongue allotransplantation model	Mandibular bone and tongue is included into hemiface model. Contains bone marrow
	Maxilla transplant model	Response of maxillary bone to transplantation
	Midface transplant model with sensory and motor units	First model to evaluate motor and sensory recovery after face transplantation
	Total osteocutaneous hemiface transplant model	Includes entire hemiface: vascularized nose, premaxilla, eyelids, upper and lower lips, external ear and facial skin for immunological, histological and biological evaluation of different facial tissues
	Composite face and eyeball transplant model	Evaluation of optic nerve regeneration and evaluation of orbit content

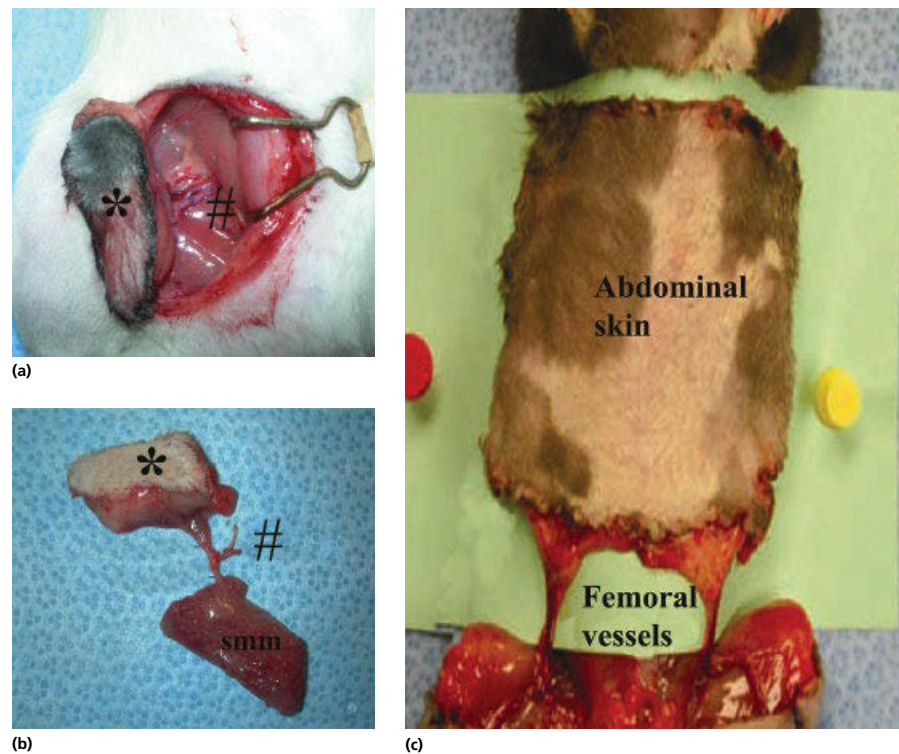


Figure 12.1 Skin-containing vascularized composite allotransplantation models. (a) Groin flap (*) raised on vascular pedicle (#) of the femoral artery and vein. (b) Semimembranosus muscle and epigastric skin flap model. * Indicates the epigastric flap, # indicates the vascular pedicle and 'smm' indicates the semimembranosus muscle. (c) Total abdominal wall transplant model harvested on the bilateral vascular pedicle of the femoral artery and vein.

nerves are coapted. Muscles and skin are closed anatomically. Hindlimb transplantation in the rat is a complex procedure involving connection of bone, muscles, nerves, vessels and skin. The rat hindlimb allograft transplant is the most popular animal model tested in preparation for human hand transplantation. This underlies the importance of having a fast, simple surgical procedure (with short anaesthesia) that provides good recovery for animals and consequently robust statistics.

In order to obtain a fast and simple limb allotransplantation method, Chang *et al.* described a groin–thigh osteomyocutaneous flap, which is composed of skin (groin), muscle (thigh) and bone (two thirds femur), based on the femoral vessels and superficial epigastric vessels.²² The advantages of this model are shortened operative time, easy monitoring of graft rejection, decreased morbidity and mortality rates, and capability for earlier postoperative ambulation.

Immunomodulatory VCA models

These models include vascularized bone and bone marrow transplantation and immunologic tissue transfers, such as thymus, in order to induce chimerism and immunotolerance (Figure 12.2). Vascularized femur transfer is composed of femoral bone, bone marrow, periosteum, cartilage and muscle tissues. This was initially described by Tai *et al.*²³ This model was used to investigate the effects of vascularized bone marrow transplantation on chimerism induction. A bilateral vascularized

femoral bone transplantation model was subsequently described by Agaoglu and Siemionow.²⁴ Bilateral femoral bones were harvested based upon the abdominal aorta and the inferior vena cava and after transfer connected to the same vessels in the recipient. This model included the same tissue types but with a larger bone marrow content, which was shown to induce higher levels of donor-specific chimerism in the recipient. Another vascularized bone marrow transplantation model was the composite vascularized skin/bone allograft model.²⁵ This model was basically a combination of groin flap with vascularized femur transplantation. The allograft was carrying both: the skin component to follow-up the rejection of the flap and bone marrow component to induce the chimerism. In order to reduce the morbidity and increase the allogenic load, a vascularized osteomyocutaneous iliac flap was described.²⁶ This flap included iliac bone, abdominal wall musculature and a wide skin island, harvested on iliolumbar artery and vein. It has a large skin island with bone marrow content, making this model almost an ideal composite tissue model. Unfortunately, it is a technically challenging model and, despite high bone marrow content, the chimerism levels were reported to be low.

Another bone marrow transplantation method is vascularized sternum transplantation. Sternum, including presternal skin and muscles, was first transplanted by Santiago *et al.*²⁷ The allograft was harvested on the thoracic aorta and superior vena cava.

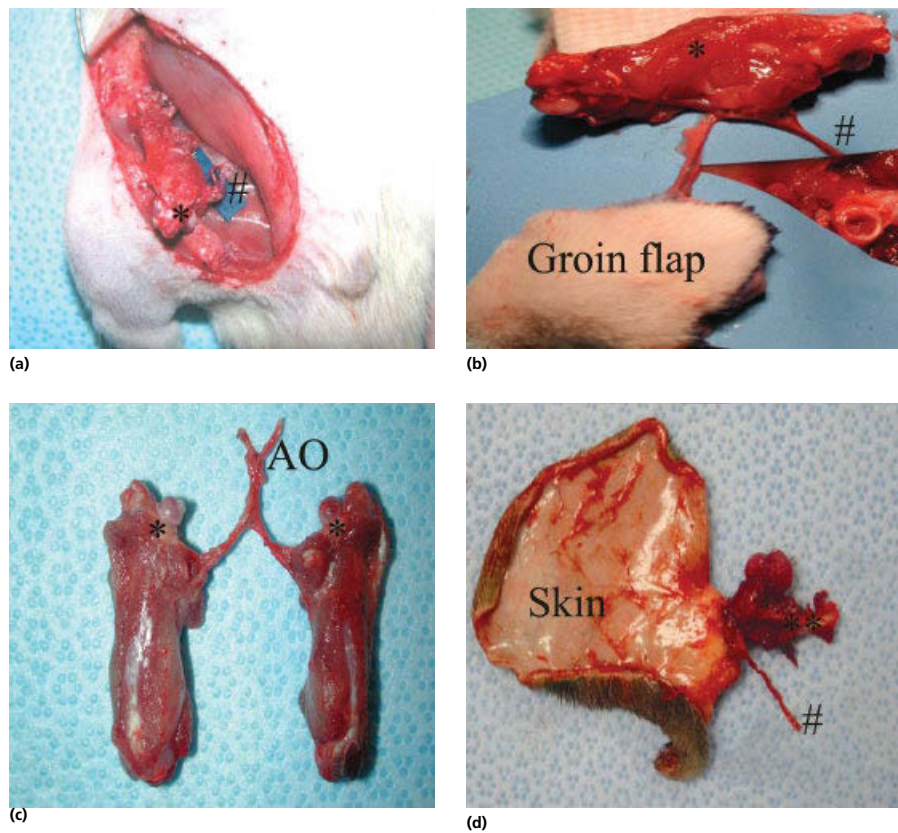


Figure 12.2 Immunomodulatory vascularized composite allotransplantation models. (a) Vascularized femur transfer. * Indicates the femoral bone, # indicates the vascular pedicle of the allograft. (b) Bilateral vascularized femoral bone transplantation, * indicates the femoral bone, AO indicates abdominal aorta as the vascular pedicle of the allograft. (c) Composite vascularized skin/bone allograft model. * Indicates the femoral bone, # indicates the vascular pedicle of the allograft. (d) Vascularized osteomyocutaneous iliac flap. ** Indicates the iliac bone segment, # indicates the vascular pedicle of the allograft.

When the bone marrow-containing models are compared, it is seen that the limb model contains 48.75×10^6 bone marrow cells. On the other hand, iliac, femur and sternum models contain 25, 50 and 7.5×10^6 bone marrow cells, respectively. The results of these experimental studies also revealed that chimerism levels are directly related to the amount of bone marrow cells.

Another method of tolerance induction in experimental studies is to include thymus transplantation. The first thymus transplantation was described by Jiang *et al.* in 1999.²⁸ Later composite osseomusculocutaneous sternum, ribs, thymus, pectoralis muscles and skin allotransplantation model were developed.²⁹ This model includes three important immunological tissues (bone marrow, thymus and skin) and has the advantage of carrying marrow cells for chimerism induction, skin for monitoring of rejection as well as thymus for tolerance induction.

Facial allograft transplantation models

Because the face has many different anatomic structures and units, many facial allograft transplantation models have been developed to evaluate transplantation of facial structures (Figure 12.3). Siemionow *et al.* were the first to develop a full-face scalp allotransplantation model.³⁰ This facial allograft

transplant, which includes bilateral ear, neck and facial skin without eyelid and nose skin, is based on the bilateral common carotid artery and external jugular vein. However, because the operation was challenging, with a high rate of mortality, a hemiface scalp allotransplantation model was then developed in the Cleveland Clinic Laboratories.³¹ This model included scalp, the unilateral ear, neck and facial skin – excluding eyelid and nose skin – and was based on the unilateral common carotid artery and external jugular vein. The hemiface scalp allotransplantation model is generally used for immunologic studies about facial allograft transplantation. Yazici *et al.* developed a hemiface calvaria model by adding temporal muscle and parietal bone to the prior hemiface scalp model.³² This model included parietal bone with bone marrow cells, which were viable up to 100 days post transplant as confirmed by histology. The facial allograft model, containing mandible with bone marrow cells, was later described by Kulahci *et al.*³³ In this heterotopic allotransplantation model including hemiface mandible and tongue, Kulahci *et al.* showed high levels of donor-specific chimerism. In maxilla allotransplantation, the maxilla was dissected along Le Fort II osteotomy lines based on the common carotid artery and

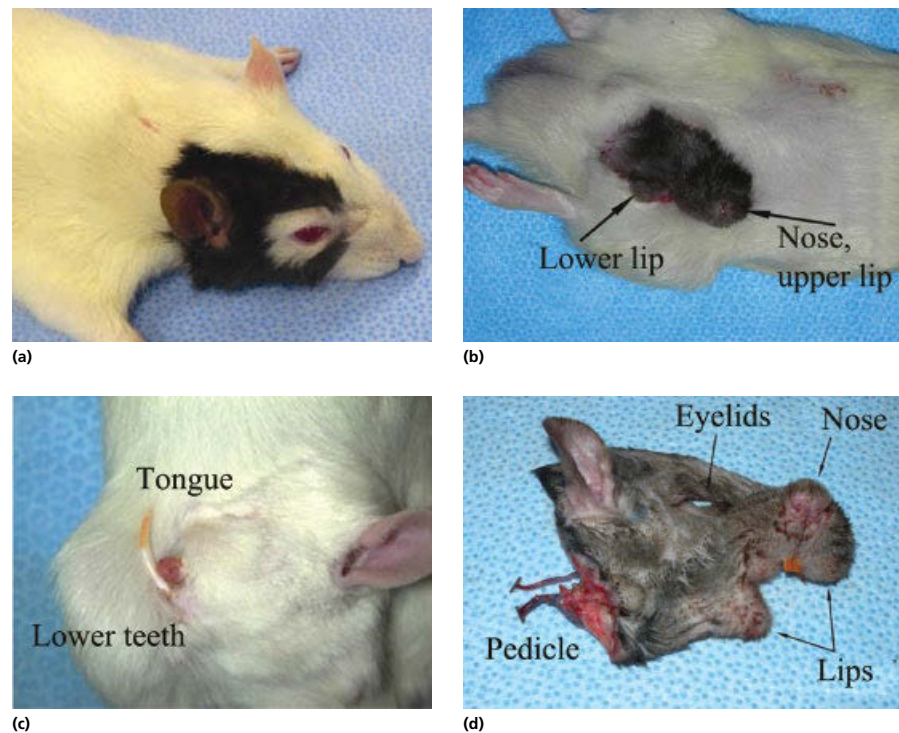


Figure 12.3 Facial VCA models. (a) Hemiface and scalp allotransplantation model. (b) Hemiface mandible tongue allotransplantation model. (c) Midface transplant model with sensory and motor units. (d) Total osteocutaneous hemifacial allotransplantation model.

external jugular vein and transplanted. This model was developed to evaluate the maxillary response to allotransplantation.³⁴

The first model to evaluate motor and sensory recovery after facial allotransplantation was developed by Zor *et al.*³⁵ This composite midface transplant model with sensory and motor neuromuscular units included premaxilla, mystacial pad and nose with infraorbital and facial nerves. At 100 days post transplant, somatosensory-evoked potential (SSEP) and motor-evoked potential (MEP) tests revealed that sensory and motor recovery reached 67% of normal latency values for infraorbital nerve and 70% for facial nerve latency values. Recently, a total osteocutaneous hemifacial allotransplantation model was developed in order to extend the application of the face/scalp transplantation model in the rat.³⁶ This model included all hemifacial structures, such as vascularized nose, premaxilla, eyelids and upper and lower lips, external ear and facial skin. This new model, including all the hemifacial soft tissues as well as the premaxillary bone segment with nose components, enabled immunological, histological and biological evaluation of different facial tissues and structures in one complex composite allotransplantation scenario. The most recent advancement in facial allotransplantation models is the development of composite face and eyeball allotransplant model with optic nerve.³⁷ This experimental model provided evaluation of optic nerve regeneration and the effect of allotransplantation on composite facial tissues, including orbital content.

Large animal models and other VCA models

Other VCA models have been developed for the evaluation of posttransplant changes of various organs. Of these, perhaps the most important one is the cremaster muscle allotransplantation model, which was used to evaluate the microcirculatory changes following allotransplantation.³⁸ Laryngeal and penis allotransplantation models are other models that have been described.^{39,40}

Large animal models have also been developed for evaluation of feasibility and to encourage the clinical application of allotransplantation. Facial allotransplantation models have been described in various large animals such as dogs, rabbits and swine.⁴¹ A swine model of osteomyocutaneous radial forearm flap transplantation was described by Ren *et al.*⁴² Non-human primate models of face transplantation have been studied in cynomolgus monkeys.⁴³ Technical and immunosuppressive requirements for prolonged graft survival are studied in these models. Hand transplantation in non-human primates has been described in baboons and rhesus monkeys.⁴⁴ Both partial and total hand transplantation and immunology-related studies have been performed with these models.

Cadaver studies

Allotransplantation potentially solves challenging reconstructive problems by allowing the surgeon to place an entire *en-bloc* donor part (i.e. hand) onto a recipient. However, all technical aspects of harvesting and implanting this body part needed to be addressed by cadaveric studies. In terms of hand

transplantation, the anatomic and technical challenges have been determined already by experience with hand replantation cases. But facial composite tissue allotransplantation needed cadaver studies in order to determine time estimates, recommended sequence and type of dissection and pertinent anatomical structures. As a result, cadaver face transplantation studies are performed in preparation for clinical face transplantation.

The technical feasibility of face/scalp transplantation was first confirmed in cadaver studies by Siemionow *et al.*⁴⁵ Briefly, the whole face/scalp flap was elevated in the subplatysmal, sub-superficial musculoaponeurotic system (SMAS) and subgaleal

planes within the neck, face and scalp, respectively. In these studies, Siemionow *et al.* described the details of the surgical technique of face/scalp flap harvesting in a cadaver study (Figure 12.4).⁴⁵ They also demonstrated the integrity of the vascular territory following the flap harvest by methylene blue dye injection. As a continuation of this study, Siemionow *et al.* performed a mock transplantation by harvesting a total face/scalp flap in one group of cadavers and transferring the flap to the recipient cadavers.⁴⁶

During the donor flap inset, the anatomical consideration of the flap-anchoring sites and the length of arterial and venous pedicles and nerves incorporated in the flap were evaluated. The

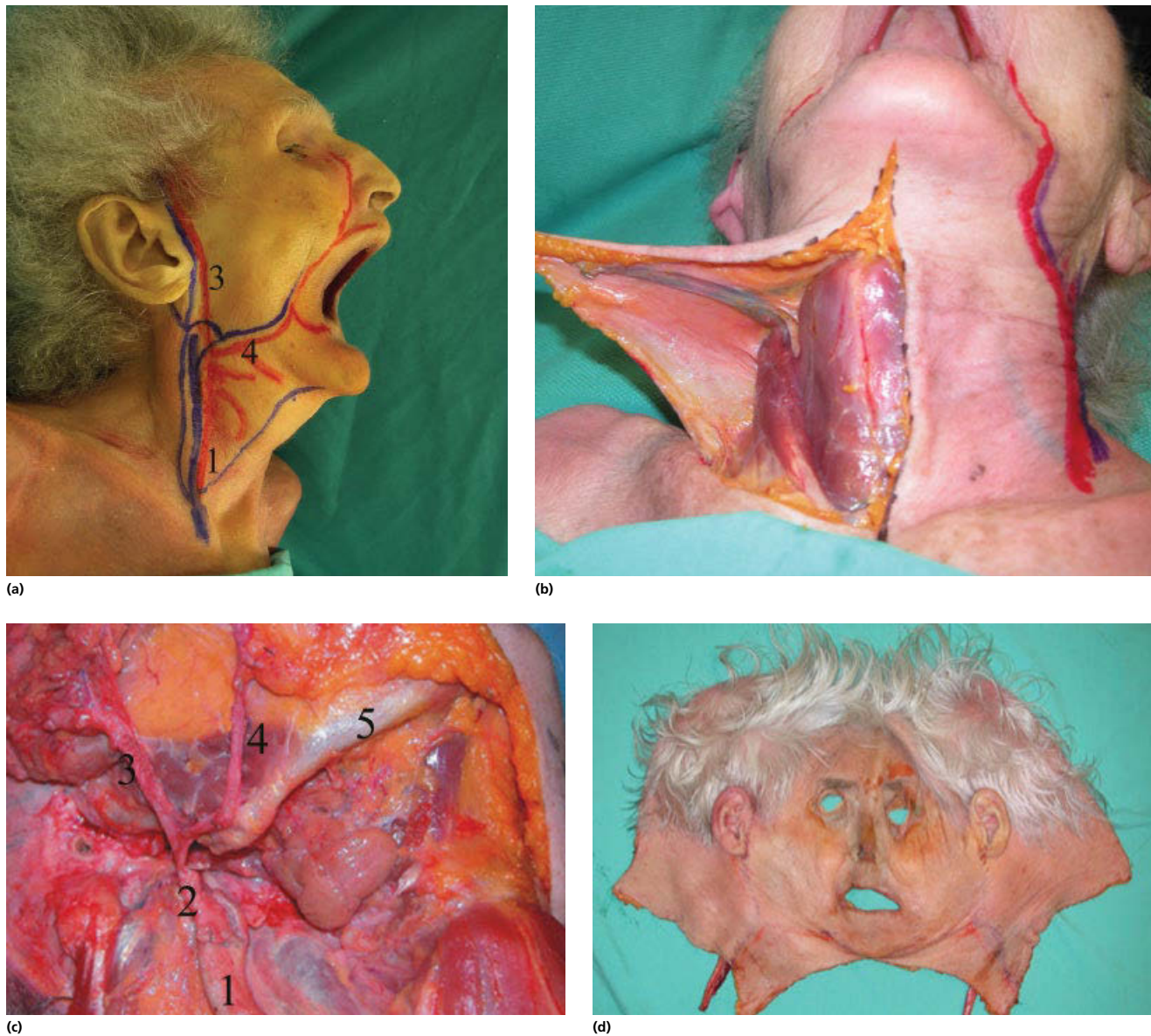


Figure 12.4 Cadaver dissection of subplatysmal, sub-SMAS face/scalp flap. (a) The outline of the underlying vascular structures at the face and neck. (b) Following an anterior neck incision, dissection is performed at subplatysmal level. (c) The arterial network of the face/scalp flap after flap elevation. 1 – common carotid artery, 2 – external carotid artery, 3 – superficial temporal artery, 4 – facial artery, 5 – mandible margin. (d) The harvested total face/scalp flap. Source: Siemionow *et al.*, 2006.⁴⁵ Reproduced with permission of Lippincott Williams & Wilkins.

vascular pedicle was found to be suitable for microvascular anastomoses, and nerve coaptation was possible. The mean total facial flap harvesting time was 235.62 ± 21.94 min. In this study, the sequence of procedures for mock facial transplantation was also described. Later, coronal-posterior approach for face/scalp flap harvesting from donor cadavers was developed to prepare extended length to the neurovascular bundles utilized in facial composite tissue allotransplantation.⁴⁷ It is suggested that the extended length of the neurovascular pedicle will not only provide easier vessel anastomoses and nerve coaptation but also provide the chance for meticulous haemostasis after artery and venous anastomoses.

Despite the continuity of its form and contour, the face can be surgically divided into distinct anatomic units. Each of these units is the object of a separate reconstruction. According to these anatomic principles, Lengele described three main segmental face transplantation types that can be harvested from one or more branches of the external carotid arterial network and includes certain sensory and/or motor nerves:⁴⁸ lower central facial allograft, midfacial allograft and upper facial allograft.

Lower central facial allograft (type I)

The lower central facial allograft includes the donor's nose, lips and chin, from the cutaneous surface to the deep mucosa. It is vascularized by bilateral facial vessels and contains all perioral muscles harvested under subperiosteal dissection. Motor innervations of these muscles arise from zygomatic, buccal and mandibular branches of the facial nerve, whereas sensory nerves including the mental (V3) and infraorbital (V2) nerves arise from the trigeminal nerve. If necessary, dissection can be extended deeper to include the middle part of the mandibular arch to restore bony support to the chin.⁴⁹

Midfacial allograft (type II)

The midfacial allograft contains the nose, upper lip, cheeks and muscles elevating the perioral structures. It is elevated equally on right and left facial pedicles. Although it can consist of soft tissues only, it may also include the anterior part of maxilla and zygomatic arches and variable parts of the anterior palate. Graft sensitivity is restored by infraorbital nerves (V2) with motor reinnervation relying on the zygomatic and buccal rami of the facial nerve.⁵⁰ Another midfacial allograft, Le Fort II-based osteocutaneous face transplantation, was described by Yazici *et al.*⁵¹ Using six cadavers, in combination with MRI and angiography, the authors showed the vascular territories in the masticatory space along with the internal maxillary artery's anterior pterygomaxillary extension.

Upper facial allograft (type III)

The upper facial allograft includes the superficial planes of the forehead, eyelids and root of the nose and the deeper planes of the frontalis, glabellar and orbicularis oculi muscles. It is elevated on the two superficial temporal pedicles and supraorbital

sensory nerves (V1). Motor innervation is provided by frontal and zygomatic branches of the facial nerve.

In addition to these three facial transplantation types, two more types are described by Lengele. According to Lengele, type IV is total face transplantation, which includes all three types, and type V is total face and scalp transplantation.⁴⁹

Face transplantation is potentially indicated only for defects that are not capable of reconstruction by conventional techniques and it is most appropriate that face transplantation be carried out only in patients with massive facial defects. Since these massive defects often include bone deficits, the indications for face transplantation may be more rewarding when bone and soft tissue replacement is needed.

Follmar *et al.* carried out mock osteocutaneous face transplantation in a cadaver model. The face was harvested in the subperiosteal plane and included the Le Fort III osseous segment. The allograft was inset by rigid internal fixation and soft tissue approximation. They showed that osteocutaneous face transplant procedure was technically feasible. On the basis of their experiment and review of prior investigations, the authors have identified 10 points that present technical challenges specific to osteocutaneous transplantation of the face. These included customization of the bony segment, sensory and motor innervation, extraocular movements, dentition, mastication, speech and swallowing, airway, vascular considerations, immunologic considerations and identity issues.⁵²

Baccarani *et al.* presented two face-harvesting techniques in a fresh human cadaver model. In first technique, the skin and soft tissue of the face was harvested by dissections in subgaleal, sub-SMAS and subplatysmal planes. In the second technique, the entire soft tissue and bony structures of the midface were harvested in a subperiosteal plane dissection combined with a Le Fort III osteotomy. Each face was harvested successfully as a bipedicle flap based on the external carotid arteries, the external jugular veins and the facial veins. The authors showed that each technique was a theoretically viable approach to face harvest for facial VCA.⁵³

Wang *et al.* compared two facial flap-harvesting techniques for alloplastic facial transplantation. They aimed to shorten donor graft harvesting time and reduce warm ischaemia. Six paired (SMAS) plane composite face/scalp flaps were harvested using either a superficial temporal artery (STA) or a facial artery (FA) pedicle technique or an external carotid artery (ECA) pedicle technique. They found that STA and FA pedicle flaps provide a shorter harvesting time and potentially safer dissection method for facial transplantation compared with previously reported total facial allograft harvesting techniques based on ECA pedicle.⁵⁴

Later Gordon *et al.* applied orthognathic principles and practice to maxillofacial allotransplantation. They planned the allotransplantation by either using dental cast models, cephalometric analyses, model surgery and occlusal splint fabrication or using a mimic Gunning splint (an edentulous scenario). It was shown that functional and aesthetic results are superior when using orthognathic principles.⁵⁵

More recently, Brown *et al.* incorporated computer-assisted planning and intraoperative navigation to improve precision and efficiency in complex maxillofacial allotransplantation procedures.⁵⁶ Ten mock facial transplantations including all facial skin, mimetic muscles, tongue, midface by means of a Le Fort III osteotomy and mandible by means of sagittal split osteotomies were performed. They showed that preoperative computer planning and intraoperative navigation provided precise osteotomies and a good donor–recipient skeletal match, which greatly reduced the need for intraoperative adjustments and manipulation.

Allotransplantation of the orbital globe and periorbital tissues was studied by Bozkurt *et al.*⁵⁷ In this cadaver model, they harvested the periorbital soft tissues, bony orbit and orbital contents by performing a box osteotomy. Both the ECA and the ophthalmic artery were prepared and indocyanin was used to show the perfusion of the flap.

Finally, cadaver studies have been performed to overcome some of the problems that have been confronted during the clinical application of face allotransplantation. The collected experience from prior facial allotransplantation has shown that the recovery of sensory function of the face graft is unpredictable. The most frequent problem in the clinical setting is the lack of healthy donor nerves, especially in central face defects. In their study, Rodriguez-Lorenzo *et al.* investigated the technical feasibility of transferring the supraorbital nerve to the infraorbital nerve in a model of central facial transplantation.⁵⁸ This study showed that supraorbital nerve to infraorbital nerve transfer is an anatomically feasible procedure in central facial allotransplantation and this procedure could be used to improve the restoration of midfacial sensation by the use of a healthy recipient nerve in cases where the recipient infraorbital nerves are not available.

Another nerve transfer study in allotransplantation was performed by Audolfsson *et al.* and evaluated the technical feasibility of performing nerve transfers in facial transplantation for both sensory and motor neurotization.⁵⁹ The first part of this study evaluated the technical feasibility of nerve transfer from the cervical plexus to the mental nerve and the masseter nerve to the buccal branches of the facial nerve. The second part aimed to simulate the face transplantation scenario, focusing on sensory restoration of the midface and upper lip by neurotization of the infraorbital nerve, sensory restoration of the lower lip by neurotization of the mental nerve, and smile reanimation by neurotization of the buccal branches of the facial nerve. The authors concluded that all nerve transfer procedures are technically feasible and could be used in face transplantation either as a primary nerve reconstruction, or as a secondary procedure for enhancement of functional outcomes.

Clinical applications of allotransplantation

VCA has considerable potential in reconstructive surgery. With proper immunosuppression and a very specialized surgical team, almost any missing part of the body can be restored using

principles of reconstructive transplantation. Many clinical VCA applications have been reported and the golden principles of every procedure are the same: proper selection of the recipient, a VCA well adapted to the needs of the recipient, correct immunosuppression and postoperative care. In this section, clinical applications of VCA are summarized.

Upper extremity transplantation

The loss of a hand or both hands is an important impediment for a patient and when replantation is impossible, the only solution remains hand transplantation. Since the first successful hand transplantation in Lyon in 1998, multiple hand transplants have been performed all over the world.^{60,61}

Because of the many years of experience with hand replantation, there are almost no anatomical and technical challenges regarding the procedure. The time estimates, recommended sequence of transplantation and pertinent anatomical structures are already known. Today, bilateral forearm amputation is the globally accepted indication for hand transplantation.

Patients with guillotine-type amputations at the level of the wrist joint and distal forearm are ideal candidates. In order to reduce the warm ischaemia time, two teams of surgeons working simultaneously are preferred. During the graft harvest, it is better to have more than enough length of all donor structures so they can be joined with recipient tissue without any tension. This is especially important for neurovascular structures. Both teams must use a tourniquet to operate in a bloodless field for optimal identification of all the available neurovascular structures, tendons and muscles. Extensive incisions in the forearm must be made for better exposure of the neurovascular structures for dissection. Incisions are not placed directly above nerves and vessels, as swelling may prevent direct closure, and the flaps must be prepared for Z-plasty transposition during closure. Skin incisions must be planned bearing in mind stump scarring and the possibility of creating a zigzag skin-flap closure.

Dissection and identification start at the subcutaneous level and continue down to the bone. All available structures must be tagged. After identification of all available structures, the tourniquet is released to control bleeding. When preparation of all structures of the donor hand is complete, it is perfused with 500 mL of chilled University of Wisconsin (UW) solution through the brachial artery. The sequence for hand transplantation is:

- 1 osteosynthesis;
- 2 vessel repair (anastomosis of the one main artery and two veins);
- 3 muscle–tendon repair/transfer;
- 4 definitive vessel repair;
- 5 nerve repair;
- 6 skin closure and dressing.

During definitive vessel repair, the two main arteries and at least 4–6 veins must be anastomosed. It is important to anastomose not only superficial veins but also deep vena comitantes. This is the guarantee for optimal venous return. All three main forearm/

hand nerves (median, ulnar and radial/superficial radial, depending on amputation level) must be repaired.

Following skin closure using the skin flaps in a Z-plasty manner, postoperative care and physical rehabilitation is started. The most important complications relating to hand transplantation are rejection episodes. Acute rejection episodes also have a negative effect on the outcome of upper extremity allotransplantation.

Immunosuppressive protocols for hand transplantation are similar to those for solid organ transplants and mainly include induction therapy with thymoglobulin or campath, combined with tacrolimus, mycophenolate mofetil and prednisone. Maintenance therapy usually consists of tacrolimus with mycophenolate mofetil and/or prednisone.

Face transplantation

The first successful face transplantation was performed on 27 November 2005 in France.⁴⁹ Since then there have been more than 27 reports of facial composite tissue allotransplantation.

Perhaps one of the most important issues in face transplantation is recipient selection. Today, the only scoring system developed for recipient selection is the Cleveland Clinic's FACES score.⁶²

Matching the donor and recipient is essential to ensure compatibility for the major (ABO) blood groups, as well as a negative cross-match between recipient and donor. Whether tissue matching for HLA poses a significant benefit on graft survival after facial transplantation is not known. Cytomegalovirus immunization status of the donor and the recipient must also be taken into account.

There are some screening issues unique to face transplantation. The transplanted tissue is visible, therefore must be cosmetically matched to the recipient. Broadly this means that the donor and recipient should be of the same sex and ethnicity and of similar age.

The surgery may begin simultaneously in donor and recipient but sometimes following the removal of all scar tissue, the actual defect of the recipient is determined and then the donor operation is started. The allograft is harvested based on a reliable pedicle, which generally includes either unilateral or bilateral facial and superficial temporal vessels. Depending on the reconstructive need, this allograft may contain soft and bony tissue and the level of the dissection may be sub-SMAS or subperiosteal. The motor and sensory nerves are also prepared. The steps can be summarized as: microvascular vessel anastomoses, fixation of the allograft, nerve coaptations, anatomical repair of muscles and other structures, and skin closure.

Physiotherapy and speech therapy (if indicated) may begin as early as 48 h following surgery. In general, rehabilitation consists of supervised controlled passive and active motion exercises, assessment of mastication, smell, speech, swallowing and facial expression. Gentle massage, sensory re-education, and facial acceptance education are encountered. Psychological status (i.e. anxiety, depression), quality of life and self-esteem of

the patient are evaluated. Sensory and motor re-innervations are closely monitored.

From an aesthetic standpoint, the patients are reported to be satisfied with their aesthetic result and have reported that they feel more comfortable in public even knowing that multiple surgical revisions such as scar revision and redundant tissue excision are necessary in order to obtain an optimal aesthetic outcome.

Other clinical VCA applications

Besides hand and face transplantation, there are other clinical applications of VCA, including larynx, trachea, knee, femur, abdominal wall, peripheral nerve, tongue, scalp/external ear, penis, uterus, flexor tendon apparatus, lower extremity and muscle.^{1,2,61} However, most of these applications are experimental, involving small numbers of cases.

Laryngotracheal transplantation

The first successful laryngotracheal allotransplantation was performed in Cleveland Clinic in 1988. The allograft included the entire larynx, and adjacent tracheopharyngeal structures including thyroid and parathyroid glands. Functional results and patient satisfaction was excellent, with normal swallowing and speech restoration. Later, more than 20 laryngotracheal transplantations were performed. One of these transplantations also included the oesophageal part; this was the first oesophagus transplantation.^{2,61}

Knee and femur transplantation

The first femur, knee and surrounding soft tissue transplantation was performed by Hoffmann in 1996. The initial results were good, but the long-term results were not satisfactory in most patients.^{1,61}

Abdominal wall transplantation

Abdominal wall allotransplantation was described by Levi *et al.* Abdominal wall composite allograft composed of both rectus abdominis muscles, overlying fascia, subcutaneous tissue and skin was harvested upon the inferior epigastric vessels. Abdominal wall transplantation patients received simultaneous intestinal or multivisceral transplantations without alteration of the immunosuppressive protocol required for organ transplants. Over 15 abdominal wall transplantations have been performed with satisfactory results.¹

Peripheral nerve transplantation

Mackinnon was the first to perform successful peripheral nerve allotransplantation. The immunosuppressive protocol of peripheral nerves differs from other transplantations. First, it is only necessary for 12–24 months and second it includes FK506, which is known to enhance nerve regeneration. The results of allogenic peripheral nerve transplantation were encouraging.^{2,61}

Outcomes

A total of 62 recipients have received either single or bilateral allotransplantation to date. According to the International Registry on Hand and Composite Tissue Transplantation (IRHCTT), acute complications of upper extremity transplantation are related to vascular surgery and are similar to free tissue transfers. There have been at least four cases of limb loss reported during the early (due to non-compliance) and late postoperative period. With a follow-up period of up to 15 years, it has been shown that immunosuppression-related complications include: serum sickness (3%), opportunistic infections (88%), acute rejection episode (85%) and metabolic complications,

such as hyperglycaemia, acute or chronic kidney disease, hypertension, Cushing syndrome and avascular hip necrosis (70%). Despite the immunosuppression-related complications, the functional outcomes of upper extremity transplantation are good. According to IRHCTT reports, 31 patients with at least 1-year follow-up have showed improvement in both the Hand Transplantation Scoring System (HTSS) and the Disabilities of the Arm, Shoulder, and Hand (DASH) score. It is reported that protective sensation, grip and pinch functions were provided in all patients.^{63,64}

Twenty-seven recipients have received partial or total face (cutaneous, myocutaneous and osteomyocutaneous) transplantation. Three of these died (one from infection, one from

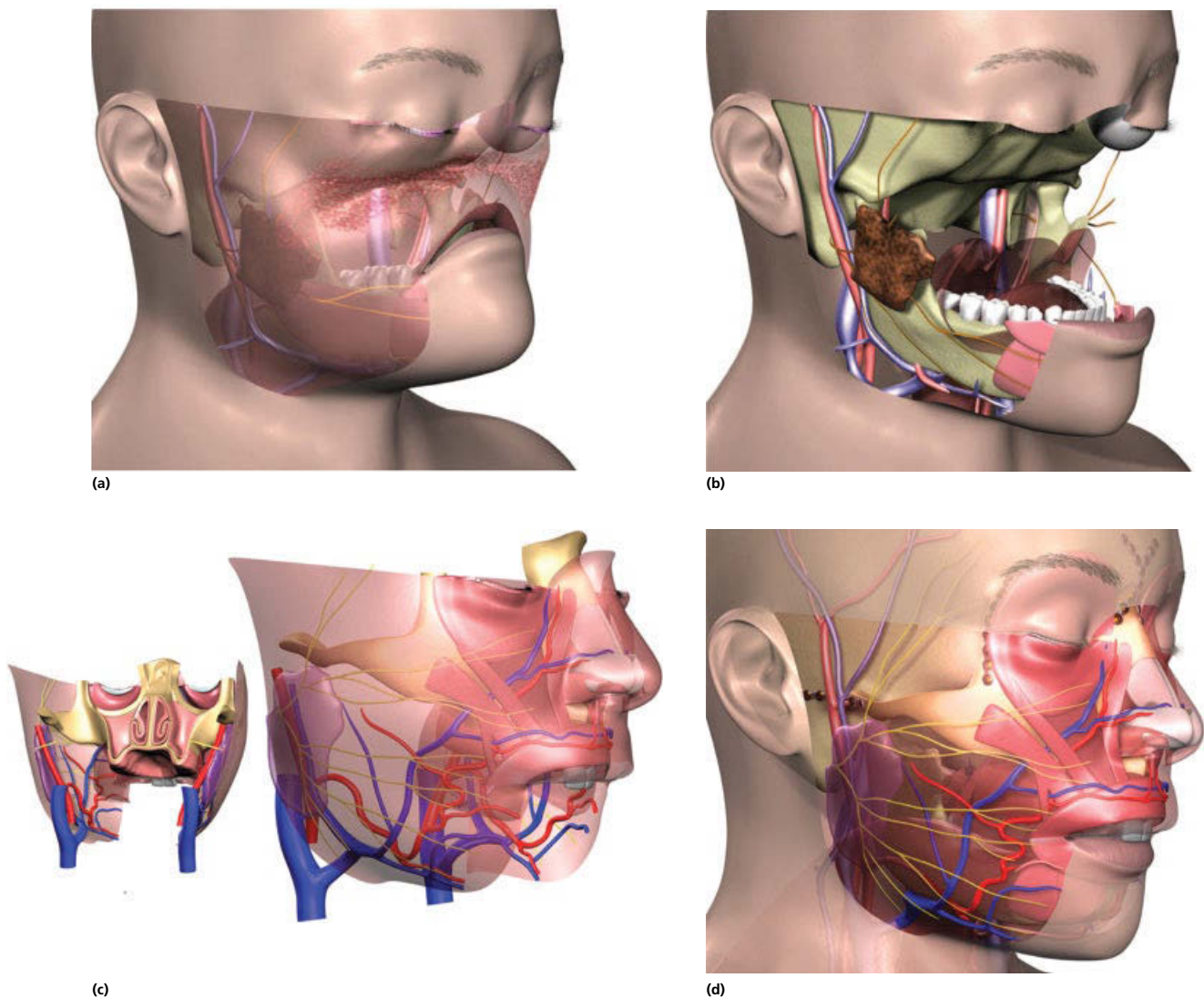


Figure 12.5 Diagrams of the US first near-total face transplant, showing the sequence of the three-dimensional reconstruction that replaced 80% of the patient face. (a) The diagram of patient deformity following gunshot injury and conventional reconstructive operations. (b) Following debridement of all scar tissues, the full facial defect is shown. (c) The face allograft (back view and right anterior view), included 535 cm² of facial skin, functional units of full nose with nasal lining and bony skeleton, lower eyelids and upper lip; underlying muscles and bones including the orbital floor, zygoma, maxilla with teeth, hard palate and parotid glands, arteries, veins and nerves. (d) The diagram showing restoration of the patient's craniofacial defect after face transplantation.

stopping immunosuppression and the third from complications of long-term immunosuppression). In the clinical setting, all patients who received face transplantation accepted their new appearance without signs of psychological problems related to the transplantation.⁶⁵ Good functional and aesthetic results have been obtained. Dubernard *et al.* reported that sensory discrimination and hot–cold sensation returned nearly 18 months following the transplantation. Motor and olfaction functions also returned in face transplantation patients. However, in all reported cases, motor recovery was slower and less optimal compared to sensory recovery.

Siemionow *et al.* reported good functional outcomes for the first near-total face transplant (Figure 12.5).⁶⁵ The patient presented with return of breathing through the nose, being able to drink from a cup and eat solid foods. In addition, good sensory recovery was confirmed by restoration of sensation, which was close to normal. Motor recovery was also satisfactory, with return of essential facial functions such as pucker, smile and pronunciation of the vowels e, i and o.⁵⁰

All face transplant recipients with more than 1-year postoperative follow-up experienced at least one episode of acute graft rejection that was observed clinically and confirmed histologically on skin and mucosa biopsies.

Wound-healing problems, stenosis of Stensen's duct, postoperative ptosis and ectropion of eyelids were observed during the follow-up period and treated surgically. Despite standard antibacterial and antiviral prophylaxis protocols, the majority of recipients have experienced opportunistic bacterial, viral and fungal infections. Insulin-dependent new-onset diabetes mellitus, transient thrombocytosis, acute renal failure, thrombotic microangiopathy, transient steroid-induced confusion, transient leukopenia, posttransplantation monoclonal B cell lymphoma, cervical dysplasia, severe rhabdomyolysis, acute respiratory distress syndrome and diaphragmatic paralysis are the metabolic complications observed in face transplantation patients.^{63,64,66}

Future perspectives

VCA has been shown thus far to be a viable option in a particular subgroup of patients who suffer severe defects not amenable to modern-day reconstructive techniques. The long-term risks of current immunosuppression are the major concerns when debating this experimental surgery as improving 'only' quality of life. Thus, many experimental and clinical studies are currently focusing on newer immunosuppression options, such as donor-specific tolerance.

Transplantation of different structures is another topic of the future development for VCA. Experimental studies of an entire orbit, including the optic nerve, have already been performed and may allow clinical application in the near future. Research regarding transplantation of a whole head (allo-head and body reconstruction) has also begun to appear in the literature.⁶⁷

Although much of this may sound like science fiction, in the future, transplantation of the entire orbit for restoration of vision or transplantation cochlear apparatus for restoration of hearing function may become a clinical reality. We have to keep in mind that just a short time ago VCA procedures such as face transplantation were also considered to be science fiction.

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CHAPTER 13

Radiation therapy and soft tissue response

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Radiation principles

Radiotherapy is the medical use of ionizing radiation, generally as part of cancer treatment to control or kill malignant cells. Ionizing radiation is composed of particles that individually carry enough kinetic energy to liberate an electron from an atom or molecule. Directly ionizing radiation consists of alpha and beta particles. Indirectly ionizing radiation includes photon radiation which may be gamma radiation if produced by radioactive decay or X-rays if produced outside the nucleus.

Alpha particle radiation

An alpha particle is identical to a helium nucleus, containing 2 protons and 2 neutrons. Alpha particles are generally produced by a process of alpha decay. If ingested, inhaled or absorbed into the bloodstream, they can cause severe damage. Radium-226, radon-222 and polonium-210 are alpha emitters. Most alpha emitters occur naturally in the environment and are found in various amounts in almost all rocks, soils and water. This form of radiation is rarely used for the treatment of cancer.

Beta particle radiation

Beta particles are high-energy, high-speed electrons or positrons emitted by certain types of radioactive nuclei. Cobalt-60, strontium-90 and iodine-131 are examples of some of the beta emitters. Iodine-131 is used in the treatment of benign and malignant thyroid disease. Strontium-90 is used in the treatment of metastatic bone cancer and has also been used for treating ocular melanoma. Beta particles are also a source of positrons used in positron emission tomography (PET).

Gamma radiation

Gamma rays are produced by nuclear gamma decay and are emitted from a radioactive source, often following the emission of a beta particle. Gamma rays are less ionizing but more penetrating than alpha and beta rays and can be stopped by a few centimetres of lead. The commonly used gamma emitters are caesium-137, cobalt-60 and technetium-99m. Technetium is

widely used for diagnostic studies, whereas caesium and cobalt are used in the treatment of cancer.

X-Rays

X-Rays, like gamma rays, are part of the electromagnetic spectrum. They are emitted by electrons. X-Rays of varying energies have many uses but are best known for their medical application. Low-energy X-rays (~10 keV) are used in diagnostic radiography to take images of part of a patient's body (e.g. chest or abdomen). The differential absorption of the X-rays by bone, air and soft tissue generates a clear radiograph. High-energy X-rays (photons) are able to ionize atoms and disrupt molecular bonds, thereby causing potential damage to the tissues they pass through. The ionizing capability of X-rays is utilized in radiotherapy, which uses photons to kill malignant cells in the treatment of cancer. In radiotherapy the photons are generated by a machine called a linear accelerator (linac). The linac generates electrons and then speeds them up to almost the speed of light using electrical fields. As the electron is accelerated its kinetic energy is increased until such time as it collides with the target and the energy is released as a photon (Bremsstrahlung radiation).

Mode of action in malignancies

When radiation interacts with cells, ionization of DNA molecules takes place causing tumour cell death. The biological effect of ionizations is controlled by a number of factors, such as the number of ionizations that can cause DNA damage, free radical scavenging processes and the degree of cellular repair. There are two mechanisms by which radiation causes cell damage: direct and indirect.

Indirect effect

Approximately 60% of the human body is made of water; therefore the majority of ionizations generated by irradiation arise in the water molecules. When radiation interacts with water it breaks the bonds that hold the water molecule together, producing

a positively charged hydrogen ion (H^+) and an uncharged hydroxyl radical ($OH^\cdot$). The OH radical is often called a free radical as it has an unpaired electron and is highly reactive, causing cell damage. These free radicals may undergo further reactions to produce hydrogen peroxide, which also contributes to the destruction of the cells. Oxygen plays a key role in this process of radiolysis. Approximately two thirds of the biological damage caused by X-rays or electrons is by indirect effect.

Direct effect

If radiation causes ionization of the atoms that are part of the DNA molecule, or some other cellular component essential for the survival of the cell, then the cell may be destroyed directly by interference with its life-sustaining structure. This is referred to as the direct effect of radiation.

Biological effect on the cell cycle

Cell cycle progression is vital for the growth and survival of a cell or organism (Figure 13.1). The normal cell proliferation cycle goes through two main phases, mitosis (M phase) and DNA synthesis (S phase), which are separated by the G₁ (before cell division) and G₂ (after cell division) phases. G₀ is the resting phase before cell division begins. The average length of a cell cycle is 16 h; this is shorter for cancer cells. Cell cycle arrest normally occurs in response to events that threaten the integrity of the DNA. Cells are most sensitive to radiation in the M and G₂ phases and most resistant in the S phase of the cell cycle. After radiotherapy is delivered, cells may be in different phases of the cell cycle. Fractionating radiotherapy may allow resistant cells time to enter the sensitive phase of the cycle and be more amenable to cell kill. Following radiotherapy, DNA damage can also cause a progressive delay in G₁, S and G₂ phases of the cell cycle. This delay gives the cells time to repair the damage.

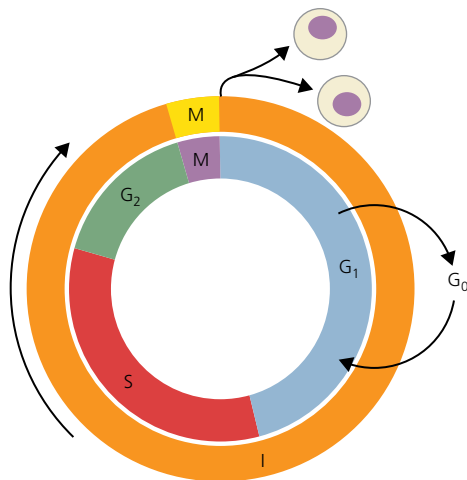


Figure 13.1 The cell cycle. By Cell_Cycle_2.svg: *Cell_Cycle_2.png; Zephyris at en.wikipedia; derivative work: Beao; derivative work: Histidine (Cell_Cycle_2.svg) [CC-BY-SA-3.0 (<http://creativecommons.org/licenses/by-sa/3.0/>) or GFDL (<http://www.gnu.org/copyleft/fdl.html>)], from Wikimedia Commons.

Radiation injury

Radiation kills actively dividing cancer cells efficiently; damage to cancer cells may occur immediately but may take months to take full effect. Along with the cancer cells, some rapidly dividing normal cells (skin, intestines, hair and bone marrow) are also damaged in the process, leading to unwanted acute effects. Slowly dividing normal cells (nerves, brain, bone, vessels) exhibit radiation-induced damage later.

Radiation-related injury to the cells is divided into three main categories:

- **Lethal damage** is a complex, irreparable and irreversible mechanism leading to cell death. The cell is unable to repair the damage and gives rise to a linear component of cell killing.
- **Sublethal damage** following ionizing radiation can be repaired within hours of the injury taking place and would not lead to cell death, unless further radiation exposure was inflicted on the cells. Cellular repair mechanisms can protect them from further injury, which is important for normal tissues, as they are able to repair themselves between fractions of radiotherapy. The approximate time needed for half the sublethal damage to be repaired (half-life) is different for different tissues. The recovery time is approximately 1 h and is thought to be complete by about 4 h in mammalian cells.
- **Potentially lethal damage** will inevitably kill the cells unless manipulated by repair-facilitated cell cycle arrest. Normal cells can readily be repaired in the S phase but not in the M phase. Tumour cells have defective cell cycle controls and therefore do not undergo cell cycle arrest required for repair, in contrast to the normal cells. A minimum of 6 h are required between fractions of radiotherapy to provide sufficient time for these repair mechanisms to work.

Modes of delivery

Radiotherapy delivered by a machine is often referred to as external beam radiotherapy. It may also be delivered by placing a radioactive source inside the body, which is referred to as brachytherapy.

External beam radiotherapy

This is the commonest form of radiotherapy used for the treatment of cancer. Different machines are used for the delivery of varying energies.

The superficial machine produces X-rays that range from 50 to 150 kV (kilovolts) and is used for treating superficial skin lesions. It deposits most of its energy at the surface of the skin and 90% of the dose at a depth of approximately 5 mm from the surface of the skin.

The orthovoltage machine generates X-rays ranging from 150 to 500 kV energy, depositing maximum energy near the surface of the skin and 90% of the dose at a depth of approximately 2 cm from the skin surface. It is also used for treating superficial tissues, skin and bone lesions.

The megavoltage machine produces X-rays of higher energies, usually 1 MV (megavolt) and above. A linear accelerator (linac) is commonly used for the delivery of these high-energy beams. The maximum dose of radiotherapy can be delivered to various depths depending on the energy of the beam used, aiding treatment of deep-seated tumours. Most linacs can also be adapted to treat superficial lesions, such as in cases of skin cancer. This treatment uses electrons instead of high-energy photons. Further technical advances in the design of the linac has permitted the use of intensity-modulated radiotherapy (IMRT) with the use of multileaf collimators (MLC). These are leaves of lead housed in the head of the linac machine, which shape the radiation beam according to the treatment volume and reduce the radiation dose to normal tissue.

Brachytherapy

Brachytherapy is a mode of delivering radiotherapy by using sealed radioactive sources which can be placed either inside a cavity containing the tumour (intracavitary) or directly in the tumour itself (interstitial). Other forms of brachytherapy include intraluminal, intraoperative, intravascular and surface brachytherapy.

The main radioactive sources used for brachytherapy are iridium, caesium and iodine.

The advantage of brachytherapy over external beam radiotherapy is the ability to deliver higher doses of radiation to the tumour and lower doses to the surrounding normal tissues. There tends to be a rapid fall-off in dose as the distance increases from the radioactive source, which restricts its use to the treatment of small and well-localized tumours.

Unsealed radioactive sources are also used for treatment and can be delivered either orally or intravenously. However, following treatment, limited-period precautions are necessary to avoid radiation exposure to individuals in close contact. Radioactive iodine-131 used in the treatment of thyroid cancer is an example of this form of treatment.

Fractionation

Curative treatment of cancer requires higher doses of radiation in order to produce sufficient cell killing. The total dose is delivered in small daily fractions over a period of time to minimize toxicity by allowing sufficient time for normal tissue recovery while inducing tumour cell kill. The efficacy of fractionation is based on the four Rs of radiobiology: repair of sublethal damage, redistribution of cells within the cell cycle, repopulation and reoxygenation.

By splitting radiation into small daily fractions, normal cells with efficient repair mechanisms are able to repair sublethal damage in contrast to cancer cells, which have suppressed repair pathways. Both normal and malignant cells repopulate between fractions. Prolonging total treatment time over several weeks allows normal cell repopulation, however some rapidly dividing tumours such as squamous cell carcinomas regenerate faster than the normal cells and exhibit accelerated repopulation. Fractionation increases damage to the tumour by allowing

reoxygenation of the tumour environment and redistribution of the cells into radiosensitive phases of the cell cycle.

Conventional (standard) fractionation is the delivery of 1.8–2 Gy per day, given as five daily treatments a week over several weeks. Other fractionating regimes used are as follows:

- *Hyperfractionation*: The same total dose is delivered over the same time as in standard fractionation but more than one fraction is given per day at a lower dose per fraction. This reduces the late effects of radiotherapy.
- *Accelerated treatment*: The total dose is delivered over a shorter period of time compared with standard fractionation in order to overcome accelerated tumour repopulation.
- *Continuous hyperfractionated accelerated radiation therapy (CHART)* combines the above two approaches and is used in the treatment of rapidly proliferating tumours (e.g. lung cancer and some head and neck malignancies). It consists of three smaller fractions of radiotherapy per day, separated by 6 h to allow recovery of normal tissues.
- *Hypofractionation*: Radiotherapy is delivered in a smaller number of fractions with a higher dose per fraction. There is potentially a higher risk of developing late effects in normal tissues with this regime. It is used in the treatment of slow-growing tumours which are generally resistant to standard fractionation schedules and also in the palliative setting where development of late effects are less relevant.

Dose and dose rate

Dose is the general term used to determine the quantity of radiation. The energy imparted by ionizing radiation to matter at a specific point is defined as absorbed dose. The SI unit of absorbed dose is joule per kilogram (J/kg) also known as gray (Gy).

Dose rate is defined as the radiation dose delivered per unit time and measured in grays per hour. Radiation response decreases with decreasing dose rate and this is referred to as dose rate effect. The typical dose rate used in standard radiotherapy is 1 Gy/min.

Effects of oxygen and radiotherapy

Malignant cells are most sensitive to radiotherapy in the presence of oxygen. Hypoxic cells are radioresistant and are usually present in the core of the tumour, whereas the cells on the surface of the tumour have an abundant blood supply and are well oxygenated. As the tumour is exposed to radiotherapy, the outer, well-oxygenated layers of the tumour are rapidly destroyed and the hypoxic inner layers become oxygenated and thus radiosensitive between treatment fractions.

Treatment parameters

Total dose

Total dose refers to the total amount of ionizing radiation absorbed by the patient (expressed in Gy). Every tissue in the body has a tolerance dose beyond which the organ would be

at risk of serious complications. Various dose/fractionation schedules can make it difficult to assess the total dose delivered to a particular organ. Therefore a formula is applied to compare altered fractionation doses to standard 2 Gy per fraction doses to know the exact total dose delivered to an organ. This is referred to as a biologically equivalent dose.

Dose fraction size

Fraction size is a significant factor influencing the development of acute and late effects of radiotherapy. Large fraction sizes increase the severity of late normal tissue damage.

Volume treated

The severity of acute and late normal tissue injury from radiotherapy depends on the volume of tissue irradiated. Tissues with a parallel organization, such as the lung and kidney, have a high functional reserve and exhibit a prominent volume effect. This means that the damage caused would depend on the dose distribution to the whole organ rather than to a small area. On the other hand, serial organs such as the spinal cord and gastrointestinal tract have little functional reserve and it is vital that radiation dose does not exceed tolerance at any point.

Time elapsed

If the overall treatment time is increased, a greater amount of repopulation of the irradiated normal and tumour cells is permitted. This has an adverse effect on local tumour control, especially in rapidly proliferating tumours.

Radiation effects

Acute toxicity

The acute effects of radiotherapy are generally defined as those toxicities occurring up to 90 days from completion of treatment. Acute effects usually manifest in rapidly dividing cells in the area being treated and are thought to be due to depletion of stem cells. Common sites exhibiting early radiation effects are the skin (including hair follicles and sweat glands), mucous membranes of the gastrointestinal tract and haematopoietic tissue.

If photon radiation is used it can take some weeks into the treatment course before erythema becomes evident, as initial dose is deposited in the deeper dermis. If a higher skin dose is required (e.g. when tumour involves the skin or in the treatment of a primary skin cancer), a tissue-equivalent bolus material may be applied over the treated field to increase the surface dose. Adjustments in radiation technique to use electrons or orthovoltage (less penetrating) radiation also increase the skin dose and result in the development of skin erythema within the first week of a fractionated course of radiotherapy.

The rates of onset of damage and recovery from it depend upon the turnover rate of epithelial cells. In a typical six-week course of radical radiotherapy delivered with curative intent one would expect skin erythema and inflammation to develop at the

treated site within the first three weeks. Epilation also occurs early and dry desquamation of the skin may follow as surface flaking of the stratum corneum. Towards the end of the course of radiotherapy moist desquamation of the skin is often seen, as exudate is released from the damaged skin cells. This is more commonly seen at the skin creases of the axillae and groins.

In radiotherapy to the head and neck, mucositis develops over the first few weeks, ranging from patchy isolated areas to confluent areas with ulceration. Taste disturbance is noted and salivary gland function is diminished. The latter results from acinar atrophy and chronic inflammation of the salivary glands.¹

Late toxicity

The late effects of radiotherapy are generally the dose-limiting factor. High total dose, large fraction sizes and large treatment volumes all contribute to a greater risk of late effects. The main mechanisms for the late effects of radiotherapy are believed to be damage to vascular and connective tissue and a reduction in the proliferative capacity of stem cells. Late effects tend to manifest in slowly proliferating tissues (e.g. lung, kidney, liver, heart and nervous tissue).

Late normal tissue damage reflects a failure to regenerate functional tissue, whether through lack of stem cells or radiation-induced dysregulation of the normal healing process. This latter phenomenon is described as a result of recent research into radiobiology and molecular pathology of late radiotherapy effects. Cytokine cascades are activated early at the cell and tissue level after irradiation and remain active throughout the expression of late tissue damage. Skin atrophy, telangiectasia, necrosis and fibrosis occur many months after radiotherapy. Skin atrophy is seen as shrinking and thinning of the skin in the irradiated field.² Telangiectasia results from a failure in the regrowth of normal vasculature. It has been postulated that telangiectasia results from loss of smooth muscle cells in vessels, leading to a loss of capillary pressure control. Loss of endothelial cells as a result of radiotherapy may also contribute to its formation.³ Necrosis is generally seen in atrophic skin often developing as a result of trauma, and reflecting the impaired reaction of the vasculature to tissue injury. Many late effects of radiotherapy can be attributed to fibrinogenesis and excessive extracellular matrix and collagen deposition. Increased knowledge of the relevant cytokines and their actions has provided molecular targets for potential therapeutic intervention.

Osteoradionecrosis

Osteoradionecrosis (ORN) can be defined as an area of exposed bone in a field of radiation that has failed to show any evidence of healing for six months.⁴ The incidence of ORN is believed to have declined considerably over time and reported incidence ranges from 3 to 5%.

The pathophysiology of ORN is described as a cumulative tissue damage induced by radiation. The three 'Hs' principle outlines how radiation results in a hypocellular, hypovascular and hypoxic tissue environment. Tissue breakdown occurs as a

result of cell death and collagen lysis exceeding cellular replication. The result is progression to a non-healing wound.

A key event in the progression of ORN is the activation and dysregulation of fibroblastic activity that leads to atrophic tissue within a previously irradiated area.⁵ High-dose radiation leads to bone death via a combination of mechanisms described as progressive resorption of osteoclasts, periosteocytic lysis, extensive demineralization and accelerated ageing of bone.

Common sites for bone necrosis are the jaw (soft tissue tumours of the head and neck), ribs and sternum (breast cancer), skull (brain tumours and soft tissue tumours of the scalp) and vertebral column (spinal cord tumours).

Factors contributing to the development of ORN include high radiation dose with the highest incidence occurring with radiation dose above 70 Gy.⁶ Large treatment volumes and high doses per fraction also impact on the probability of developing ORN.^{7,8} Poor dentition has been associated with a higher incidence of ORN of the mandible. Patients should have a thorough dental assessment and extraction of any teeth in poor condition prior to commencing a course of radiotherapy to the head and neck in order to optimize oral health. However ORN has been seen in edentulous patients also.

Advanced (stage III or IV) tumours/recurrent tumours, tumours invading bone, and tumours involving floor of mouth, tongue and retromolar trigone have been shown to have a higher risk of ORN.⁹

The presentation of ORN varies with site. In cases of ORN of the mandible patients may present with pain, swelling and trismus. ORN of the temporal bone can present with severe otalgia and offensive otorrhoea. Exposed bone and pathological fracture may also be seen. Imaging with plain radiographs may highlight areas of local decalcification or sclerosis. CT or MRI scanning is used to delineate the affected area prior to definitive surgery. A deep biopsy of the lesion to rule out recurrent malignancy is generally recommended.

Treatment of ORN involves initial measures such as control of any superadded infection, gentle irrigation of the soft tissues, nutritional support and analgesia. Hyperbaric oxygen therapy (HBOT) may also be considered.

Radiation therapy and wound healing

The wound-healing capacity of irradiated skin may be severely impaired. Clearly this has clinical implications for patients requiring surgery after radiotherapy. Approximately 60% of patients with solid malignant tumours receive radiotherapy as part of their treatment, either with curative or palliative intent. Radiotherapy is often used after surgery in malignant disease in cases of narrow or involved excision margins or to nodal basins post lymphadenectomy to reduce the risk of local disease relapse and improve overall survival. Radiotherapy may also be used before surgery to downstage a tumour prior to definitive resection (e.g. in preoperative radiotherapy for rectal cancer and head and neck cancer).

The process of wound healing is well described to occur in three phases: an inflammatory phase which develops within days, a proliferative phase which occurs over many days to weeks and a remodelling/maturation phase which extends from many weeks to years.

Unlike a surgical wound, however, fractionated radiotherapy results in repetitive injury to soft tissues. This results in an accumulation of inflammatory responses and dysregulation of this highly structured process of wound healing.

Cytokines known to be involved in the initial inflammatory response to tissue damage are transforming growth factor beta (TGF- β), vascular endothelial growth factor (VEGF), tumour necrosis factor alpha (TNF- α), interferon- γ (IFN- γ) and pro-inflammatory cytokines such as interleukin 1 and interleukin 8. These are all overexpressed following irradiation and lead to uncontrolled matrix accumulation and fibrosis.¹⁰

In the proliferative phase overexpression of additional cytokines (e.g. epidermal growth factor (EGF), fibroblast growth factor (FGF) and platelet-derived growth factor (PDGF)) leads to dysregulated granulation tissue formation, re-epithelialization and neovascularization.

Matrix metalloproteinases (MMPs) are responsible for the remodelling phase and regulate extracellular matrix synthesis. Irradiation can lead to dysregulation of MMPs leading to disorganized collagen deposition by fibroblasts.

There has been much interest in the postoperative complication rates of reconstructive surgery after radiotherapy and the optimal timing of surgery and radiotherapy. Much of the published literature is in reconstructive head and neck surgery and breast surgery.

It is understood that previously irradiated patients have a higher risk of postoperative complications.¹¹ In cases of microvascular free flap transfer TGF- β 1 has been shown to be overexpressed in free flaps for as long as six months after radiotherapy. Extracellular matrix remodelling has also been detected in the free flap for up to six months after radiotherapy completion.¹² The alteration in graft bed extracellular matrix organization after radiotherapy and prior to surgery has been shown to impact significantly on free flap survival in the pre-irradiated field.¹³

However several studies have been unable to demonstrate a significant difference in flap failure rate between irradiated and non-irradiated tissues. A large study by Bengston *et al.* used a prospective database to review 354 patients with a total of 368 free tissue transfers limited to the head and neck over a 4-year period. Postoperative complications in 167 patients who received preoperative radiotherapy were compared with 187 patients who did not undergo radiotherapy preoperatively. No significant differences in complications or flap loss between the two groups were noted. Total flap loss occurred in 5.3% in the irradiated group and 5.0% in the non-irradiated group. Major wound complications requiring additional surgery were seen in 16% of the radiotherapy group and 11% in the non-radiotherapy group. Interestingly, no significant difference in the timing or

dose of preoperative radiotherapy; previous neck dissection or anastomotic type was shown in failed versus successful flap.¹⁴

Similarly a smaller study of 100 patients undergoing fibular free flap reconstruction of the mandible retrospectively compared three groups: non-irradiated, preoperatively irradiated and postoperatively irradiated patients. Although 54% of patients experienced at least one postoperative complication, there were no differences among the three groups in the overall proportion of patients with complications of any severity: 54% versus 65% versus 46%. No case of complete flap loss occurred.¹⁵

It has been postulated that this failure of studies to show a significant difference in flap failure rates after preoperative radiotherapy in head and neck cancer may be due to a lack of statistical power owing to the very high success rate of microvascular free flap surgery.¹⁶

A recent small retrospective study of 12 previously irradiated patients with nasopharyngeal carcinoma who underwent salvage surgery and reconstruction from 2002 to 2007 found no intraoperative or significant postoperative complications. Four different types of flaps were used (two free and two pedicled) and all flaps survived.¹⁷

The timing of radiotherapy has been the subject of much debate with regards to the risk of delaying wound healing and reducing the success of microvascular free flaps when radiotherapy is used preoperatively. This has been retrospectively investigated for sarcoma patients where 119 patients with sarcomas of the head and neck; upper and lower extremities were reviewed with approximately two thirds receiving preoperative radiotherapy and one third receiving postoperative radiotherapy. Free flap loss rates were significantly lower in the preoperative radiotherapy group (0% versus 8.7%). Late recipient site complications also occurred less often in patients receiving preoperative radiotherapy (6.8%) compared to postoperative radiotherapy (26.1%).¹⁸

A review of 140 patients at the M.D. Anderson Cancer Centre who underwent mandibular resection and reconstruction with a free fibular flap compared preoperative radiotherapy and immediate reconstruction, preoperative radiotherapy and delayed reconstruction, postoperative radiotherapy and no radiotherapy. Complications included orocutaneous fistula, osteoradionecrosis and partial or complete flap loss. Forty-two per cent of all patients studied had at least one complication, but the incidence of complications was relatively equal between the groups receiving pre- and postoperative radiotherapy.¹⁹

Our practice in cases of preoperative radiotherapy for head and neck malignancies is to recommend surgery takes place within six weeks of completion of radiotherapy. Published data supports this approach showing an increase in flap loss, infections and delayed wound healing when more than six weeks elapses from the last radiotherapy session and surgery.²⁰

In breast cancer there has been much published literature on the optimal timing for breast reconstructive surgery in relation to receiving postmastectomy radiotherapy (PMRT). Baumann *et al.* studied 189 patients having delayed lower abdominal free

flap breast reconstruction. Eighty-two patients had surgery less than 12 months after PMRT and 107 had surgery 12 months or more after PMRT. The total flap loss rate was 2.6%, with all flap losses occurring in those receiving surgery within 12 months of PMRT. This group also trended towards a higher incidence of microvascular thrombosis, infection and wound dehiscence.²¹

However, a large metanalysis of 1105 patients found that delaying breast reconstruction until after PMRT had no significant effect on outcome.²²

Supportive management

Many of the early effects of radiotherapy are evident during a treatment course. Patients are given written advice on skin care management which includes the use of a daily emollient applied after each fraction of radiotherapy. Avoidance of direct sunlight on the treated area is imperative, as is avoiding the use of potentially irritating chemical containing compounds on the skin (e.g. body lotions, perfumes, talcum powders, etc.). If moist desquamation develops emollients are not helpful and paraffin gauze dressings may offer some comfort. Early detection and treatment of superadded skin infections with oral antimicrobials is recommended.

Oral mucositis often requires analgesia. Optimal pain control is essential to ensure patients are able to take adequate oral nutrition. Analgesia is given stepwise according to need using the World Health Organization (WHO) analgesic ladder. Analgesic mouthwashes can also be used. Early dietetic and speech and language assessment of these patients is important and if necessary parenteral feeding may be used if there is insufficient oral intake.

Xerostomia can be experienced early in the treatment course and is a well-documented late effect of radiotherapy after treatment of the salivary glands in head and neck malignancies. There are limited satisfactory treatment options to ameliorate this symptom. Artificial saliva sprays and gels may offer some relief. Due to the potential significant consequences of xerostomia on quality of life, which include increased rates of dental caries and oral candidiasis, and difficulty chewing and speaking, parotid-sparing radiation techniques are now used such as IMRT. Amifostine (a radioprotective agent) has also been shown to partially preserve parotid function after radiotherapy.²³

The management of late radionecrosis involves optimal wound care: adequate debriding and cleansing, accurately assessing the wound, choosing the appropriate dressing based on the wound assessment, and encouraging granulation tissue formation and re-epithelialization. Alginates, foams, gauzes, hydrogels, hydrocolloids and transparent films are the major moisture-retentive dressings available.²⁴

HBOT is frequently used to treat osteoradionecrosis and radionecrotic wounds. Standard wound care should also be followed.

The aim of HBOT is to reverse the radiation-induced hypocellular, hypovascular and hypoxic tissue environment. HBO consists of exposing a patient to intermittent short-term 100%

oxygen inhalation at a pressure greater than 1 atmosphere. HBO increases the oxygen concentration gradient so that the area around the necrosed hypoxic tissue has a high concentration of oxygen. This stimulates angiogenesis and reverses the hypoperfusion and hypoxia, giving tissues the propensity to heal. Initial reports of the benefit of HBO therapy were published as early as the 1970s. More recently a Cochrane Database Systematic Review of 11 trials concluded that for patients with late radiation tissue injury affecting the head, neck, anus and rectum HBOT is associated with improved outcome.²⁵

Early studies of pentoxifylline in combination with tocopherol have been shown to be efficacious in reversing radiation-induced fibrosis. Pentoxifylline may also have a role in the treatment of ORN. This oral drug has an anti-TNF effect and vasodilator action, which improves microcirculation and enhances oxygen delivery to tissues. Encouraging results have been shown in a small phase II study combining pentoxifylline with tocopherol (vitamin E) and clodronate for the treatment of ORN of the mandible, with all patients experiencing complete recovery within a median of nine months.²⁶

As the molecular basis of late radiation-induced tissue damage is now better understood, in particularly the specific cytokines central to the process, experimental molecular targets for therapeutic intervention are evolving. Injection of multipotent cells, topical administration of active substances to reduce the side effects of radiotherapy and the use of growth factors (e.g. TGF- β 1, 2 and 3), basic FGF, PDGF and VEGF are all areas for future development.¹⁰

Carcinogenesis

There is much evidence to support the carcinogenic property of ionizing radiation. Namely studies from atomic bomb survivors in Japan, the Chernobyl nuclear accident and people treated with high doses of radiation for cancer or other, benign conditions. In atomic bomb survivors the malignancies seen were leukaemias and carcinomas of cells that line the body. There was no increase in the incidence of sarcomas (soft tissue tumours) in these patients. The risk of developing a radiation-induced malignancy varied significantly with age, with young children found to be 15 times as sensitive as middle-aged adults.

A contrast is seen with second malignancies induced by radiotherapy. These cancers are of cells lining the body but are often seen in tissues or organs not directly targeted and that therefore have received lower doses of radiation. Sarcomas are noted to arise in high-dose areas of radiation that lie within or immediately adjacent to the radiation fields.²⁷

Up to 10% of invasive cancers are caused by radiation, both ionizing and non-ionizing radiation (ultraviolet, radiofrequency radiation). The probability of radiation causing a cancer is a stochastic effect, meaning the probability with which it occurs increases with effective radiation dose, but the severity of the cancer is independent of dose. The neoplastic transformation can be divided into three major independent stages: morphological changes to the cell, acquisition of cellular immortality

(losing normal, life-limiting cell regulatory processes), and adaptations that favour formation of a tumour.

Those cancers that may develop as a result of radiation exposure are indistinguishable from those that occur naturally or as a result of exposure to other carcinogens. The impact of lifestyle factors such as smoking, alcohol consumption and diet significantly contribute to many of the same malignancies. There is some evidence that these effects may not simply be additive but multiplicative.²⁸

A recent UK study estimated that 1346 cases of cancer, or about 0.45% of the 298 000 new cancers registered in the UK in 2007, were associated with radiotherapy for a previous cancer.²⁹ The largest numbers of radiotherapy-related second cancers were lung cancer (23.7% of the total), oesophageal cancer (13.3%) and female breast cancer (10.6%). The highest percentages of second cancers related to radiotherapy were among survivors of Hodgkin's disease and cancers of the oral cavity, pharynx and cervix uteri. Following initial radiotherapy for breast cancer, excess risks were seen for cancers of lung, oesophagus, bone and soft tissue.³⁰ Cancers most strongly linked to radiation include bone and soft tissue sarcomas, but skin, brain, thyroid, breast and stomach cancer can also occur (Figure 13.2). In most cases the risk of second malignancy is higher if the radiation exposure occurs earlier in life or during periods of rapid growth of a tissue, such as bone sarcomas during adolescence. Cells that have matured and are no longer proliferating appear to be less susceptible to the effects of radiation. Adolescent girls receiving mediastinal radiation therapy for Hodgkin's disease have been found to develop breast cancer more often than their adult counterparts.

Radiation-induced sarcoma

Radiation-induced soft tissue sarcoma is a rare malignancy with an overall incidence of less than 1% for patients with cancer who are treated with radiation and survive 5 years. Radiation-induced



Figure 13.2 An elderly woman who developed a biopsy-proven cutaneous squamous cell carcinoma within a radiation field given to her thyroid to treat a benign thyroid goitre 20 years earlier. Areas of fibrosis and telangiectasiae consistent with late radiation effects are also seen.

sarcoma has been defined as a lesion of different histology to that of a non-irradiated sarcoma, arising within a previous radiotherapy field, usually after a latency period of more than 4 years.³¹ Although the threshold dose for radiation-induced sarcoma is not certain, most published data report a dose of 40–60 Gy. Dose–response relationships have been found between radiation dose and sarcomas. A 40-fold increase in the risk of bone sarcomas was observed following 60 Gy or more.³² High-dose radiation appears not to increase the risk for leukaemia and thyroid cancer. Stereotactic radiosurgery data support that there may be a lower risk for induction of a secondary malignancy because of a very high probability of complete cellular kill in the high-dose radiation field. Cells lose their ability to proliferate and, therefore, they are unable to sustain a malignant transformation.

Although advances in the delivery of radiotherapy appear to reduce the risk of late radiation toxicity, the newer technologies, such as intensity-modulated radiation therapy (IMRT), involve the use of more fields and consequently expose more normal tissue to low-dose radiotherapy. To deliver a specific dose to the centre of the tumour, IMRT requires the accelerator to be energized longer, which results in higher radiation doses. Therefore, it has been estimated that the incidence of radiation-induced sarcoma may increase by 0.5% with IMRT.³³

Hereditary cancer syndromes

Genetic factors can predispose to a higher risk of developing radiation-induced second malignancy. About 5% of cancers are thought to arise in the context of a known hereditary cancer syndrome. Inherited gene mutations may lead to multiple tumours within the predisposed individual. Somatic cells may sustain transforming mutations following treatment with radiation and certain chemotherapeutic agents and lead to secondary cancers. Retinoblastoma and Li-Fraumeni syndrome are examples of such syndromes.

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CHAPTER 14

Burns: acute care and reconstruction

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Introduction

The skin is the largest organ in the human body, accounting for approximately 2 m² in an average adult. Burn injury to the skin can range from being trivial to one of the most severe insults that the human body can sustain. Every relatively small burn can have devastating lifelong functional and aesthetic sequelae. The aphorism 'a burn is for life' is true for the majority of burn patients.

Burns and their treatment have been depicted since prehistoric times, with cave paintings and subsequently writings by Hippocrates, Celsus and Galen. Ambrose Paré described burn wound excision and Dupuytren described six degrees of depth of burn injury that are still in use today. The twentieth century has brought great advances in burn care, including the scientific understanding of fluid loss and resuscitation, the hypermetabolic response to trauma, the control of infection and development of topical antimicrobial agents, the value of early burn wound excision and wound closure with autologous and allogeneic skin, cultured keratinocytes and the use of artificial skin substitutes.¹

Epidemiology

It is estimated that burn injury affects approximately 250 000 people in the United Kingdom per year. About 175 000 of these people attend emergency departments and 13 000 patients with burn injuries require admission. Approximately 1000 patients a year have burn of a severity to require formal fluid resuscitation. There are approximately 300 burn deaths per year in the UK.² The UK figures are representative of most of the developed world although the United States has a higher incidence, with approximately 1.4 million people sustaining a burn injury each year. Approximately 54 000–180 000 patients in the USA require inpatient care. Burns are a major problem in the developing world, with a much higher incidence and mortality, and

represent a significant burden of unmet healthcare need in many countries.³

About half of all admissions to burn units in the UK are children between the ages of 1 and 5 years and the incidence of injury in this age group remains static. The other vulnerable group are the elderly and the incidence of burns in this cohort of patients is increasing with increasing numbers of older people. The incidence of major burn injury in the developed world is on the decrease.

Most burn injuries are preventable. Prevention has traditionally been either via education or legislation. There have been numerous successful educational campaigns that have improved safety at home and in the workplace and that have also modified people's behaviour. Legislation has also been effective in prevention of burn injuries. Examples include sprinkler systems and smoke detectors in public and commercial buildings, fireguards, transport and storage of flammable materials and, more recently, controlling domestic water heaters.⁴

The burn team

The management of severe burn injuries requires integration of health professionals providing multidisciplinary care working to a common goal. The management of the complex nature of burn injury requires many different skills, which are usually not found in a single specialist. This means that comprehensive care of such patients involves a large team of professionals that includes plastic surgeons, intensive care specialists, anaesthetists, specialist nurses (burn, intensive care, children's), physiotherapists, occupational therapists, psychotherapists, psychologists, dieticians, pharmacists and social workers, all working together as the burn team. The multidisciplinary approach also involves collaboration between clinicians and basic scientists to establish a feedback loop of clinical and basic scientific inquiry. This care is best delivered in specialized burn units, where the multidisciplinary team of experts can all contribute to patient-centred evidence-based care.⁵

Burn classification

A burn can be defined as coagulative destruction of the surface layers of the body. Burn injuries can be classified according to their depth, aetiology and percentage body surface area involved. The combination of the above classifications allows quantification of the severity of the injury.

Depth

The skin is made up of the epidermis and dermis with the adnexal structures, such as the hair follicles, sweat and sebaceous glands residing in the deeper parts of the dermis. These adnexal structures are important as they are the source of proliferating epithelial cells (keratinocytes), which resurface wounds when the skin has been injured. Loss of the barrier function of the skin allows invasion of microorganisms and systemic sepsis. Increased fluid losses from the injured skin also occur until re-epithelialization occurs (Figure 14.1).

Burn injury to the skin can be classified as partial or full thickness. If the epidermis and the superficial part of the dermis have been injured (superficial partial-thickness injury), the majority of adnexal structures are preserved, epithelialization is rapid (10–14 days) and the risk of hypertrophic scarring is low. If the burn extends down into the deeper parts of the dermis more adnexal structures are destroyed, epithelialization is slower (3–6 weeks) and there is a high incidence of hypertrophic scarring.⁶ Full-thickness burns involve destruction all constituents of the skin and usually will require surgical intervention to achieve wound healing.

Aetiology

Burn injury can be thermal, electrical and chemical. Cold injury has a similar pathophysiology and radiation injury can also damage the skin.

Thermal injury

Thermal injury accounts for approximately 90% of all burns. The depth of thermal tissue injury is related to the temperature and duration of application.

Wet heat injuries such as scalds due to hot liquids are the commonest type of injury. They account for 70% of burns seen in children and are also common in the elderly. Scald injuries tend to cause partial-thickness injuries that will heal if treated conservatively.

Dry thermal injuries are typically caused by flame, direct contact or by radiant heat.

Flame burns are common in adults and are often associated with smoke inhalation injuries. They tend to be either deep partial thickness or full thickness and usually require surgical intervention. Radiant heat injuries are commonly sustained in house fires where patients are confined in a hot environment. Contact injuries occur by direct contact with a hot object. Prolonged contact with a moderately hot object such as a radiator can cause injury and is common in people who may have been unconscious or suffered a blackout for a period of time, such as the elderly, people with epilepsy, drug addicts and alcoholics. Contact burns tend to be deep and require surgical intervention.

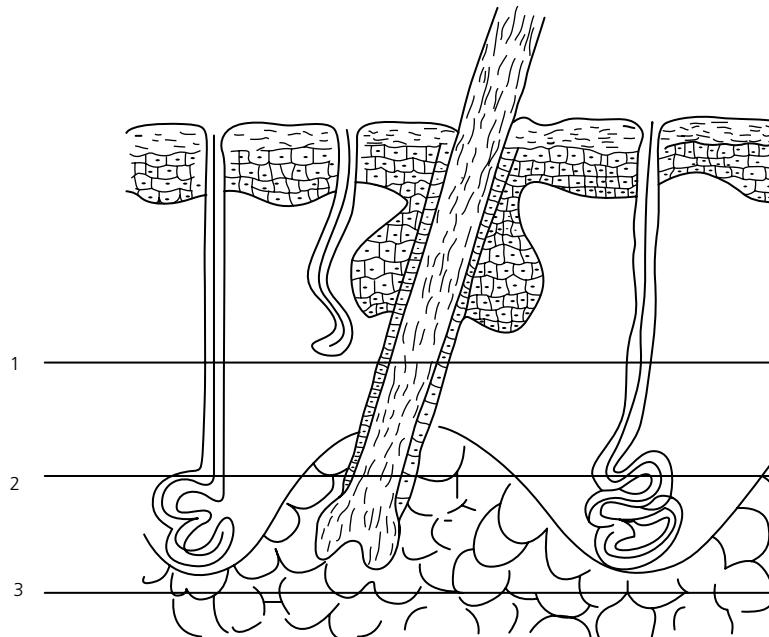


Figure 14.1 Depth of burn wound. 1: Superficial partial-thickness burn – most adnexal structures intact. 2: Deep partial-thickness burn – only deep adnexal structures intact. 3: Full-thickness burn – all structures damaged.

Electrical injury

Electrical injury accounts for less than 5% of all injuries seen in a burns unit. They can be devastating irrespective of their size and can consume significant amounts of healthcare resources. They affect children and often working men and can have significant morbidity and long-term functional sequelae.

Injury severity is determined by voltage, current, type of current, duration of contact and path of current flow. The injury caused by electric current disrupts cell membranes to produce direct tissue damage. In addition it can also generate heat when passing through tissues that act as a conductor. In general, most tissues are good conductors, particularly nerves and vessels. These tend to suffer cell membrane disruption that leads to a progressive injury. Skin and bone are poor conductors although skin conduction does vary with humidity, temperature and sweating. The heat generated occurs around substances that are poor conductors, such as bone, which in turn damage the surrounding local tissues. Clinically there are often 'entry' and 'exit' points where the electric current has travelled from one point of the body to another.⁷

Electrical injuries can be subclassified into high- and low-voltage injuries, depending on the voltage of the electrical source.

Low-voltage injuries are those caused by <1000 V, such as those due to domestic electricity. They tend to occur indoors and cause small, deep contact burns at the points of entry and exit. The injuries tend to be localized. The alternating current can interfere with cardiac conduction and may lead to arrhythmias.

High-voltage injuries (greater than 1000 V) lead to extensive tissue damage and often to loss of limbs. Cardiac asystole and arrhythmias can occur. Muscle injury leads to rhabdomyolysis, compartment syndromes and renal failure. Fluid resuscitation is complex due to the 'hidden' nature of the injury. This type of injury has an associated high mortality.

Approximately 15% of such patients have an associated traumatic injury due to a fall from a height or being thrown. Compression fractures can occur due to tetanic muscle contraction following the electric shock.⁸

Flash injuries occur when current arcs from a high-voltage source. No current passes through the body but the heat from the arc can cause flash burns to exposed parts of the body (hands and face). The resultant burn is usually partial thickness unless the clothes ignite and give rise to a flame burn, which tends to be deeper.

ECG monitoring is required for all high-voltage injuries or if a low-voltage injury has given rise to cardiac arrest, an arrhythmia or a period of loss of consciousness. If an initial ECG is normal and there has been no history of loss of consciousness, cardiac monitoring is not required.⁹

Ocular sequelae are thought to occur in up to 20% of patients with electrical injury, although the exact pathophysiology remains unknown.¹⁰

Long-term neurological sequelae are common with both low- and high-voltage injury and can be very varied in their presentation, ranging from peripheral neuropathy to complex

neuropsychological problems and autonomic nervous system dysfunction.

The exact mechanism of neurological injury is as yet unexplained, but it is noted that resolution is better for early-onset lesions compared to late-onset ones.¹¹

Chemical injuries

Chemical injuries account for approximately 3% of patients seen in a burn unit. They mainly occur as household or industrial accidents. All chemical burns will involve the denaturation of proteins, and the severity of injury will depend on the concentration, quantity, duration of contact and mechanism of action of the chemical involved.

Mechanisms of action would include reduction, oxidation, corrosion, protoplasmic poisoning, vesication and desiccation. Although the exact mechanisms of tissue injury may be different, traditionally agents have been classified into acids or alkalis as the clinical pictures in each group are similar.

Acid burns produce damage by coagulative necrosis, causing precipitation of protein, which tends to be local and short lived. Alkali burns tend to cause progressive liquefactive necrosis, allowing deeper penetration and a prolonged effect. Cement burns are alkali burns, although they can also combine with sweat to produce an exothermic reaction. In addition, cement powder is very hygroscopic and will give rise to a desiccation injury.¹²

In principle, the management of chemical burns is the same whatever the agent involved. The area must be thoroughly irrigated with water after removal of any potentially contaminated clothing. Copious irrigation dilutes the chemical and limits tissue damage. This is usually performed by putting the patient in a shower (present in most emergency departments), allowing large volume and adequate drainage of the solution. Lavage for 30 min to 2 h may be required, although care must be taken to prevent hypothermia. There is no standardized measure of adequacy of lavage, although measuring the pH of the effluent can be helpful.¹³

Ocular injuries also require copious irrigation, followed by referral to an ophthalmologist.

For most chemicals there are no specific neutralizing agents and copious irrigation is the mainstay of initial care, although there are a few specific injuries where such agents are required.

Hydrofluoric acid is used widely for glass etching and in the electronics industry. It is very corrosive and toxic. The fluoride ion rapidly penetrates through the skin, causing progressive tissue damage and intense pain. It chelates calcium ions and can lead to hypocalcaemia, which can be fatal. After initial irrigation, calcium gluconate gel is applied topically to chelate the fluoride ions.¹⁴

Phosphorus has both civilian and military uses. White phosphorus ignites spontaneously in air and can be neutralized by immersion in or irrigation with water. Phosphorus particles can be removed from the wound and a solution of 0.5% copper sulfate applied, which turns the particle black to aid the identification and removal.¹⁵

Pathophysiology

Burn injury results in activation of an inflammatory cascade that can result in a local and a systemic response. Inflammatory mediators are released via the arachidonic acid pathway and activation of inflammatory cells. These mediators include histamine, prostaglandins (PGE₂), prostacyclins (PGI₂), leukotrienes, thromboxanes, kinins, serotonin, catecholamines, free O₂ radicals and platelet activating factor. Local action of these mediators includes endothelial gapping, increase in microvascular permeability and oedema formation. In addition, microvascular stasis and thrombosis can occur, which lead to progressive injury and cell death which produces clinical deepening of the burn wound.

Local effects

Zones of injury

The initial local effect of a burn injury can be divided histologically into three differential zones of tissue damage and blood flow:¹⁶

- *Zone of necrosis* – Central tissue necrosis due to destruction of tissue by thermal injury.
- *Zone of ischaemia* – A microvascular injury surrounding the zone of necrosis. The microvascular injury can be progressive and can lead to a clinical increase in burn wound depth.
- *Zone of inflammation (hyperaemia)* – Manifested by increased microvascular permeability that surrounds the zone of ischaemia.

Systemic effects

In burns involving over 20–30% of a patient's total body surface area (TBSA) the release of inflammatory mediators into the circulation will result in a systemic response. The systemic response is propagated by the inflammatory mediators described below, but in addition tumour necrosis factor alpha, interleukins 1, 6, 8, 12, 18 and interferon gamma have been implicated as mediators of a systemic response. The systemic effects are manifested in all organ systems and can lead to organ dysfunction and organ failure.¹⁷

The burn wound is thought to be the engine of the systemic response. Early aggressive wound excision and closure has been demonstrated to attenuate the systemic response.

Metabolic effects

Major burn injury involving over 40% of a patient's TBSA is associated with a hypermetabolic response characterized by an increased metabolic rate, muscle protein wasting, peripheral lipolysis, central fatty deposition, insulin resistance, immunosuppression, increased risk of infection and multiorgan dysfunction. It can persist for 1–2 years post burn and in children can reduce growth velocity. Such patients have increased resting energy expenditures, tachycardia and increased cardiac work with increased myocardial oxygen consumption. The peripheral lipolysis and central fatty deposition can lead to impaired liver function.¹⁷

The hypermetabolic response is mediated primarily by catecholamines and corticosteroids with a 10- to 50-fold increase of plasma catecholamine and corticosteroid levels that lasts up to nine months post injury. There are significant changes in homeostasis throughout systems mediated by a variety of other mediators and hormones. These include cytokines whose levels peak after injury and only approach normal levels at 3–6 months post burn. Larger TBSA injuries result in greater and more persistent inflammatory responses that are associated with increased cytokine levels and a greater hypermetabolic response.¹⁸

Serum hormone levels and acute phase proteins are all abnormal during hospital stay. Sex hormones and endogenous growth hormone levels are decreased, as are serum insulin-like growth factor 1 (IGF-1), IGF-binding protein 3 (IGFBP-3), parathyroid hormone and osteocalcin.¹⁷ Elevated levels of catecholamines, glucagon, cortisol and gluconeogenic hormones following burn injury lead to increased glucose production in the liver. Levels of insulin and fasting glucose are elevated and there is significant reduction in glucose clearance, leading to hyperglycaemia.

The resting metabolic rate of patients with burns >40% TBSA is twice normal levels. This reduces to 130% of normal level when the wounds are healed, but remains elevated for a long time, with levels being 110–120% at 12 months post injury.¹⁷

The hypermetabolism leads to muscle protein wasting, with loss of lean body mass, decreased strength and weakness that retards rehabilitation. Ten per cent loss of total body mass is associated with immunosuppression, 20% loss of total body mass with decreased wound healing and 40% loss of total body mass increases risk of death.¹⁹

Sepsis can increase resting energy expenditures and protein catabolism by up to 40% and due to post-burn immune dysfunction these patients are more susceptible to sepsis, leading to a vicious cycle of further muscle and protein catabolism and immune dysfunction.

Management

The management of a burn depends on accurate assessment of many variables. These include the age of the patient, comorbid factors and the size, depth and anatomical location of the injury. Once these have been assessed a treatment plan can be formulated.

In general, the aim of burn care is to restore form and physical and psychosocial function. This involves early aesthetic wound closure and optimal physical and psychosocial rehabilitation.

The vast majority of burns are small, do not need specialist care and can be managed on an outpatient basis. If there are concerns about safeguarding, patient reliability, follow-up or comorbidities then inpatient care should be considered.

Initial treatment consists of pain management, removal of clothing and debris, cooling the wound, cleaning the wound, tetanus prophylaxis and an appropriate dressing.

Cooling the wound with room-temperature or cool tap water provides pain relief and limits ongoing tissue injury. It is only effective for up to 30 min and excessive cooling can lead to hypothermia.²⁰

The management of burn blisters continues to be debated. In general, small blisters can be left, but larger ones should be deroofed and the fluid evacuated. Blister fluid content contains vasoactive compounds that can have a deleterious effect on the microvasculature. Aspiration of blisters increases the risk of infection.²¹

There are numerous types of dressings used in the management of these burns and some are described below.

Management of major burns

The immediate management of the major burn is conducted according to standard trauma or immediate care guidelines such as Advance Trauma Life Support (ATLS), Advanced Burn Life Support (ABLS) or Emergency Management of the Severe Burn (EMSB). These include assessment and stabilization of airway, breathing, circulation, disability, exposure and environment control. They also involve primary and secondary assessments for the depth and extent of the burn and an evaluation for associated life-threatening injuries are performed promptly upon admission (Table 14.1).

Table 14.1 Emergency management of burn injury

First aid	Stop the burning process Cool the burn wound
Primary survey	
A – Airway and cervical spine control	Clear airway Open airway – jaw thrust/chin lift Stabilize cervical spine
B – Breathing and ventilation	Expose chest and ensure equal and adequate expansion Ventilate or intubate if necessary ALWAYS PROVIDE SUPPLEMENTAL OXYGEN CO poisoning may give a cherry pink, non-breathing patient Beware respiratory rate >20/min Beware circumferential chest burns – consider escharotomy
C – Circulation with haemorrhage control	Check pulse Capillary refill test- >2 seconds abnormal check other limbs for hypovolaemia or need for escharotomy Stop bleeding with direct pressure Mental obtundation occurs with 50% loss total blood volume Pallor occurs with 30% loss total blood volume
D – Disability – neurological status	Establish level of consciousness A – Alert V – responds to vocal stimuli P – responds to painful stimuli U – unresponsive Examine pupillary response to light Hypoxaemia and shock gives a restless patient with decreased level of consciousness
E – Exposure + environmental control	Remove all clothing and jewellery Estimate burn surface area Keep patient warm
F – Fluid resuscitation proportional to burn size	Insert a large-bore IV line through unburned skin Take bloods – FBC/U&E/coagulation/ABGS/carboxyhaemoglobin Start fluids using modified Parkland formula: $\frac{1}{4}$ TBSA/kg/h for first 8 h (2–4 mL/kg/%TBSA: $\frac{1}{2}$ in first 8 h; rest in next 16 h) <i>from the time of injury</i> Add maintenance for children Warm Hartman's solution Replace blood losses with blood
Fluids	
Secondary survey	
History	AMPLE (Allergies, Medications, Past history, Last ate, Event)
Mechanism of injury	
Examination	Head to toe Urinary catheter
Monitor adequacy of resuscitation	Pulse, blood pressure, respiratory rate ECG, pulse oximetry, ABG Insert NG tube
Baseline investigations	
X-ray	Lateral C spine, CXR, pelvis
Pain relief	IV morphine titrated incrementally
Wound management	Cling film
Records	Re-evaluate Support and reassure patients, relatives and staff

Box 14.1 Referral criteria for specialized burn service.

- Consider if >3% TBSA partial-thickness burn
 - All burns association with electric shock
 - All burns association with chemical burn
 - All burns association with non-accidental injury
 - All burns to face, hands, perineum, feet
 - All burns circumferential to limbs or trunk or neck
 - All burns with inhalation injury
 - All burns not healed within two weeks.
- Discuss with local burn service:
- All burns with other injury
 - All burns with significant comorbidity or pregnancy
 - All infected burns
 - Any other case that causes concern.

Source: London and South East of England Burn Network Referral Guidelines. <http://www.lsebn.nhs.uk>

There are no universal definitions of severity, however a major or severe burn can be defined as the following:²²

- 25% TBSA or greater in the age group 10–40 years
- 20% TBSA or greater in children <10 years and adults >40 years of age
- 10% TBSA or greater full-thickness burns
- Burns involving eyes, ears, face, hands, feet, or perineum
- High-voltage electrical burns
- Burns complicated by major trauma or inhalation injury
- Burns in association with serious comorbidities.

Initial triage and care is usually provided within an emergency department and referral criteria can be useful for referral on to an appropriate burn service (see Box 14.1).

Burn wound assessment

Estimation of depth

Determining the depth of the burn wound can be difficult. The differentiation between superficial and deep partial-thickness injury is particularly important so that the clinician can predict if the wound will heal spontaneously within 14–21 days or will require excision and grafting to optimize healing and minimize scarring.²³ A false-positive assessment and the patient faces needless surgery; a false-negative one and the patient faces increased length of stay, risks contracture and hypertrophic scar formation.

Superficial partial-thickness wounds are pink, moist and blister. They blanch on pressure, are very painful and are fully sensate on pinprick testing. Deep partial-thickness burns will either be white or red with fixed staining. They do not blanch on pressure and have reduced sensation. Full-thickness burns characteristically have a leathery appearance and are insensate.

Diagnosing a very superficial burn or a full-thickness burn is usually accurate. Intermediate or deep partial-thickness burns can be more difficult to assess and are often indeterminate on first assessment. Accurate clinical diagnosis even by experienced

surgeons is possible only 64–76% of the time and is more inaccurate for inexperienced surgeons.²⁴

Many techniques have been described to aid diagnosis of burn depth, including surgery, biopsy, vital dyes, thermography, ultrasound and numerous other experimental modalities. The only technique currently routinely used in clinical practice is the laser Doppler, which can give an estimate of dermal blood flow and depth of burn injury. Laser Doppler scanning has been reported as predicting healing within 21 days in a number of independent studies in both adults and children, confirming a good correlation between the scans and clinical wound outcome and also predicting outcome ahead of clinical judgement.²⁵

It must be remembered that the burn wound is a dynamic entity and the depth can change over a 48 h period. No technique can accurately predict outcome from a single measurement so that repeated assessments are often required. It is reported that laser Doppler scanning is most accurate after the first 48 h.

Estimation of burn size

Accurate burn size estimation is key to assessment of the severity of the injury. Epidermal burns or erythema should be excluded and the size is estimated as a percentage of the patient's body surface area. It is recognized that initial assessment in the emergency department often overestimates the burn size by over 100%.²⁶ Other sources of error in the assessment include three-dimensional to two-dimensional translation and differences in patients' body mass indices (BMIs).²⁷

The simplest method of assessment uses the patient's hand as accounting for 1% of his or her TBSA, however this can lead to overestimation. The 'rule of nines' is useful for rapid estimation of size and can be converted to the rule of tens for children. In the rule of tens the head, anterior and posterior trunk all account for 20% TBSA, with the limbs each accounting for 10% TBSA.

The Lund and Browder chart (Figure 14.2) is the mainstay for assessment and documentation taking into account different age groups and their body proportions; however this technique has also been shown to overestimate burn size.²⁸ Recently the Lund and Browder chart has been incorporated into software for smart phones which can calculate the size of the injury from a drawing but also calculate fluid resuscitation requirements for the patient.²⁹

In addition using computer-aided 3D documentation systems, a 3D virtual body representation of the patient can be produced, aiding more accurate assessment and documentation. This software is also available for smart phones, aiding the clinician at the bedside.³⁰

Escharotomies

Escharotomy is required in circumferential full-thickness burns of chest, limbs or digits. Circumferential full-thickness burns in the limbs and/or digits act as a tourniquet, impair circulation, produce distal ischaemia and can lead to compartment syndromes, digital and limb loss. Circumferential full-thickness chest burns can restrict chest wall excursion and impair ventilation. Such burns require mid-axial escharotomies.

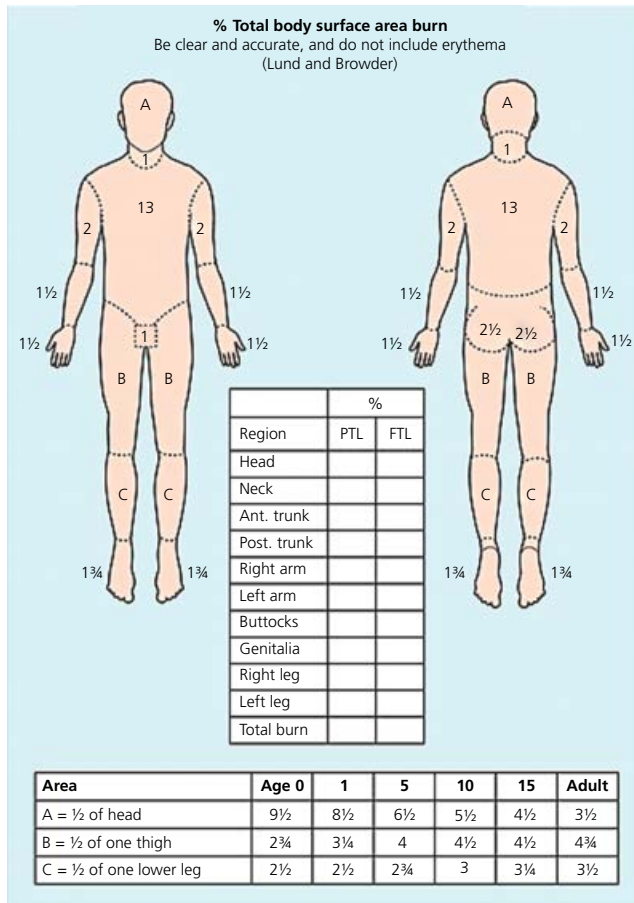


Figure 14.2 Lund and Browder chart. Source: Lund & Browder, 1944. The estimation of areas of burns. *Surgery, Gynecology and Obstetrics* 79:352–358. Reproduced with permission of Elsevier.

Escharotomies have traditionally been performed at the bedside but they are now undertaken in the admission room or operating theatre with an appropriate anaesthetic. The decision to perform escharotomy is a clinical one based on clinical appearance of the wound and the distal extremity. Assessment of distal perfusion includes skin temperature capillary refill, distal pulses (by palpation or Doppler ultrasound) and pulse oximetry. Pulse oximetry above 90% suggests adequate distal perfusion and no need for escharotomy.

The decision to perform chest escharotomy is usually based on signs of impaired mechanical ventilation, decreasing lung compliance, hypercarbia and respiratory acidosis.

A scalpel or electrocautery device can be used to incise through the full-thickness burn eschar down to bulging fat. The incisions should be placed in the mid-axial line and extend into adjacent non-burned or less deeply burned tissue. Since the wounds are full thickness, minimal analgesia or anaesthesia should be required, but extension into less damaged tissue can be very painful.

Complications of limb escharotomy include bleeding, arterial injury (brachial), venous injury (saphenous and cephalic), nerve

injury (ulnar nerve at the elbow, superficial peroneal nerve at the knee) and digital injury (neurovascular bundles and extensors).

Chest escharotomy can be a life-saving manoeuvre in the face of deterioration in ventilation. The escharotomy should be done on the anterior surfaces of the chest with incisions placed above and below the breast and along the anterior axillary lines. In children under the age of 2 years who utilize the diaphragm for ventilation and are 'belly breathers', the escharotomies should continue down onto the abdominal wall if it is also involved.

Compartment syndrome results from increased intracompartmental pressure. Fasciotomy must be considered in patients who have sustained high-voltage injuries, those with associated crush injury and those with very deep burns involving fascia and muscle. Carpal tunnel release should be considered as part of the same procedure in the upper limb. This phenomenon is discussed elsewhere in this book. Briefly, algorithms have been used for surgical decision making as to when to perform an escharotomy, fasciotomy or a carpal tunnel release. These are based on clinical signs and monitoring using the pulse oximeter and the Doppler flow meter.³¹ In addition, the use of compartment pressure monitoring either in the limb or intra-abdominally is a priority if there is any question about the diagnosis.

Smoke inhalation injury

Smoke inhalation injury is a serious associated injury that complicates cutaneous burn injury and carries a significant mortality risk.³² Smoke contains numerous noxious substances that are products of combustion, including soot, hydrocarbons, aldehydes, cyanides and carbon monoxide, which can produce both a chemical and asphyxiant injury.

Inhalation injury can be divided anatomically to upper airways injuries above the vocal cords (supraglottic) and those affecting the airways below the cords such as the trachea, bronchi and lung parenchyma (subglottic). The site of injury determines the diagnosis, management and outcome.³³

Upper airway injuries are usually thermal injuries to the mouth, oropharynx and larynx. Upper airway obstruction can rapidly occur due to oedema, making attention to the airway of paramount importance. These injuries usually resolve spontaneously as the oedema subsides after 3–5 days. These patients may require intubation and mechanical ventilation if the airway is at risk.

Lower airway and parenchymal injuries involve the trachea, bronchi and alveoli. They are thought to be mainly caused by chemical and particulate constituents of smoke. Some constituents of smoke, including carbon monoxide and/or hydrogen cyanide can impair oxygen delivery and extraction to and by the tissues.

The indications for endotracheal intubation include decreased level of consciousness airway obstruction and respiratory failure resulting from lower airway injury. Direct thermal injury to the upper airway can progress to complete airway obstruction within a matter of hours and intubation of such patients can be

very difficult or impossible. In this circumstance an emergency surgical airway or cricothyroidotomy should be considered. All burn patients with significant facial and airway oedema and those with inhalation injury should be assessed by a senior clinician with experience in managing difficult airways. It must be recognized that this group of patients may have a potentially difficult airway. Prophylactic intubation for transfer should also be considered where the airway is potentially at risk.

Lower airway injuries have always been thought to be chemical in nature, however thermal injury to the respiratory mucosa below the larynx involving the trachea and bronchi must occur when inhaling hot noxious gases. Inhalation injuries have a more prolonged course and are often associated with acute lung injury and pulmonary sepsis. The tracheobronchial mucociliary apparatus does not function, resulting in reduced clearance of particulate matter and cellular debris. These get trapped with a fibrinous exudate with formation mucous casts that can obstruct bronchi and smaller airways, leading to distal atelectasis, collapse and secondary infection. Direct alveolar injury can lead to impairment of gas exchange. Respiratory epithelial injury is usually self-limiting and recovers within about 14 days if there are no intervening complications.

Patients with smoke inhalation injury will often develop respiratory failure secondary to complex pathophysiological changes, including ventilation/perfusion mismatch, increased airway resistance, increased alveolar epithelial permeability, decreased pulmonary compliance and increased pulmonary vascular resistance. These changes give rise to impaired gas exchange, including hypoxia and hypercarbia that require mechanical ventilation. The positive pressure mechanical ventilation required following such respiratory failure will often result in increased airway pressures, shear/stress forces in the alveolus, giving rise to barotrauma and acute lung injury.

Carbon monoxide

Carbon monoxide is produced by the partial combustion of carbon-containing compounds and is a common cause of death at the scene in fires. It has a 300 times greater affinity than oxygen to bind to haemoglobin to form carboxyhaemoglobin, and can also directly impair mitochondrial function. Formation of carboxyhaemoglobin seriously impairs oxygen carriage and delivery in the blood. The organs most vulnerable to carbon monoxide poisoning are therefore the cardiovascular system and the brain. Symptoms include headache, nausea and malaise. Cardiac problems include tachycardia, hypotension and arrhythmias. Central nervous system symptoms can include delirium, seizures, unconsciousness, respiratory arrest and death.

Diagnosis can be made by direct measurement of carboxyhaemoglobin levels and measuring the arterial saturation of oxygen analysed using a co-oximeter. Standard pulse oximeter measurement of oxygen saturation provides a falsely high peripheral saturation reading, even with lethal levels of carboxyhaemoglobin, because the oximeter is unable to distinguish between carboxy and oxygenated haemoglobin.³⁴

Carboxyhaemoglobin has an elimination half-life of 4 h on room air but on inspiration of 100% oxygen, via a non-rebreathing mask, this drops to 30 min. The mainstay of management of carbon monoxide poisoning is high-flow (100%) oxygen therapy.

Hyperbaric oxygen therapy improves the clearance of carbon monoxide compared to 100% oxygen therapy and some authors have recommended its use for prevention of delayed neurocognitive syndrome. However, on review of the evidence, the Cochrane group have concluded that hyperbaric oxygen for this indication has not been shown to have any benefit and in particular there is no evidence of benefit in the patient with a cutaneous burn, smoke inhalation and carbon monoxide poisoning.³⁵

Cyanide

Hydrogen cyanide is a cellular poison that binds to the terminal cytochrome on the electron transport chain, inhibiting cytochrome *c* oxidase and making the cell unable to utilize oxygen. This can cause a profound lactic acidosis, accompanied by elevated mixed venous oxygen saturation. Cyanide is produced by the combustion of plastics, paints, wool and silk.

The half-life of cyanide is about 1 h and can be associated with a significant number of smoke inhalation injuries and carbon monoxide poisoning. Signs and symptoms are often similar to those of carbon monoxide poisoning, including central nervous and cardiovascular effects. Burn patients will typically have a metabolic acidosis that is out of keeping with the size of burn and clinical picture. A rapid assay is not available, making diagnosis difficult. A simple aid to diagnosis is an arterio-venous oxygen saturation difference of less than 10%, measured by taking arterial and venous blood gas samples. This indicates an inability of the tissues to extract oxygen from the blood.

Hydroxycobalamin (a form of vitamin B12) is the antidote treatment of choice, rapidly chelating cyanide to form cyanocobalamin. It is safe and rapidly effective but multiple doses are often needed.³⁶ In some healthcare systems it is given to all patients involved in house fires with signs and symptoms of a smoke inhalation injury.

Sodium nitrite and sodium thiosulfate have been used in the past but nitrites produce methaemoglobin, which, although it chelates HCN, also reduces oxygen carriage in the blood and can cause profound hypotension. Sodium thiosulfate, which binds to cyanide and is excreted in the urine, is safer but its action is slow.³⁷

Diagnosis of smoke inhalation

Diagnosis of inhalation injury is mainly clinical. The mechanism of injury, signs, symptoms and physical examination are important in raising suspicion of the diagnosis but are not absolute. Signs and symptoms of an upper airway injury include facial burns, facial swelling, burned nasal hairs, soot in the nose or mouth, intraoral swelling, oedema visible on laryngoscopy, hoarseness of voice and stridor. Lower airway injuries are

suggested by a history of a fire in an enclosed space, loss of consciousness, obtundation, anxiety, facial or intraoral burns, soot in the mouth or nostrils, cough, dyspnoea, wheeze, carbonaceous sputum and signs and symptoms of respiratory distress such as tachypnoea, use of accessory muscles of respiration, cyanosis and impaired gas exchange.³⁸

Patients who have a history of injury in a closed space, facial burns, large burn sizes or advanced age are more likely to have smoke inhalation injury. Investigations such as chest X-ray and arterial blood gases are often initially normal. Later changes evident on the chest X-ray include bronchial thickening, perivascular cuffing, interstitial pulmonary oedema, atelectasis and consolidation. Carboxyhaemoglobin levels are useful, although normal levels do not exclude smoke inhalation injury.

Fibreoptic bronchoscopy is the 'gold standard' for the diagnosis. Grading systems are available for the severity of injury, however in general they are not prognostic.³⁹ False-negative results can occur early on following admission, especially if the patients are hypotensive, so a repeat bronchoscopy is usually performed 48 h after admission. Recently, authors have reported the successful use of CT scanning within a few hours of injury. This is said to be predictive of outcome in terms of duration of ventilation and intensive care stay.⁴⁰

Xenon-133, a radioactive tracer, has been used to visualize small airway injuries and aerosolized technetium 99-m complexed to diethylenetriaminepentaacetate (Tc^{99m} -DPTA) has been used to assess the alveolar capillary membrane. Both techniques are only used for research and not on a regular clinical basis.

Management of smoke inhalation injury

Fluid resuscitation of patients who have smoke inhalation injury associated with a major burn can be very challenging. Such patients have significantly greater fluid resuscitation requirements during the first 48 h post burn.⁴¹ Unfortunately, more fluid administration leads to problems associated with excessive fluids, including compartment syndromes and pulmonary oedema. Conversely, there is no evidence that fluid restriction protects the lungs or improves outcome. Strict attention to fluid administration and balance must be paid to these patients during the resuscitation period.

Pharmacological management with inhaled heparin, acetyl cysteine and salbutamol can be used to limit the duration and incidence of cast formation and mucous plugging of the airways. Heparin inhibits fibrin plug formation,⁴² acetyl cysteine is a mucolytic and salbutamol a beta-adrenergic agonist. Retrospective analyses of case series have demonstrated a benefit in children⁴³ but not in adults, possibly highlighting the difference in both anatomy and physiology between children and adults.⁴⁴

Mechanical ventilation is the treatment of respiratory failure and not the treatment of smoke inhalation injury. Patients who have upper airway compromise or impaired gas exchange may require mechanical ventilation. The optimal mode of mechanical ventilation for such patients is yet to be determined.

Conventional ventilation with lower tidal volumes (5–8 mL/kg) and permissive hypercapnia have been shown to improve survival in patients who have acute respiratory distress syndrome and burns under 30% TBSA. Mechanical ventilation for burn patients who have sustained smoke inhalation injury should focus on providing pulmonary toilet, improving alveolar recruitment, reducing alveolar shear/stress and avoiding ventilator-induced lung injury.

High-frequency ventilation is commonly used in the management of smoke inhalation injury, however there is currently little direct clinical evidence of outcome benefit of using either an oscillatory ventilator or a volumetric diffusive respiration ventilator in such patients.³³

Tracheostomy

The role of tracheostomy in patients who have smoke inhalation and cutaneous burn injury remains debated. The route of intubation seems less important than the avoidance of high peak inspiratory pressures and high cuff pressures.

Recent studies in burn patients have demonstrated no difference in outcome related to the timing of insertion or technique used for insertion (open/percutaneous) of a tracheostomy. Prolonged endotracheal intubation prior to tracheostomy seemed to be a risk factor for swallowing problems and subglottic tracheal stenosis following tracheostomy.

In general, early tracheostomy should be considered for patients with extensive full-thickness facial and neck burns and those who are predicted to require a long duration of respiratory support. Tracheostomy should also be considered at about day 14 post injury in patients who are still slow to wean or who have failed attempts at extubation.⁴⁵

Fluid resuscitation

Burn injury produces both local and systemic effects, giving rise to loss of capillary integrity with leakage of fluid and protein from the intravascular to the interstitial space with the formation of oedema. In addition, intracellular oedema can occur following a drop in the transmembrane electrical potential of cells, giving rise to an increased intracellular sodium concentration caused by a postulated circulating shock factor. In burn size of 25% TBSA or more the oedema is generalized and occurs in non-burned tissues such as the gut, lung, muscle, heart and brain. The amount of oedema in all tissues is directly proportional to the burn size. In major burn injury this fluid loss can lead to hypovolaemia and shock and therefore fluid resuscitation is critical to the survival of such patients.⁴⁶

The increase in capillary permeability is transient and occurs in the first few hours following injury, caused by the systemic inflammatory mediators. The capillary leak usually resolves within a few hours to days, however in some patients it can be prolonged. Oedema continues to develop for at least 24–36 h post injury due to loss of plasma colloid osmotic pressure and

resultant decrease in the colloid osmotic pressure gradient. Correction of the hypoproteinaemia with infusions of albumin or plasma is thought to promote and prolong the development of oedema.⁴⁶

Burn shock is characterized by specific physiological changes that include an initial decrease in plasma volume and cardiac output, producing oliguria. A 'myocardial depressant factor' or inflammatory mediators have a role in the immediate fall in cardiac output observed in large burns.

The primary goal in resuscitation is to restore circulating blood volume and to preserve tissue perfusion and oxygenation. Greater volumes of fluid used in burn resuscitation lead to greater oedema and increases in weight of up to 20%. Significant oedema is characteristic of large burn injuries and is paradoxically aggravated by fluid resuscitation itself. Excessive fluid administration can give rise to 'fluid creep' and compartment syndromes.⁴⁷ Monitoring and constant re-evaluation of the resuscitation process are essential to success. The formulas are only guides and can be unreliable in the young, old, in larger burn injuries, in patients with other injuries or concomitant medical problems.

In principle, to achieve survival, patients with extensive burns need to be given large quantities of fluid, which must contain sodium salts. The total volume of salt-containing fluid required to satisfy obligatory burn oedema and make good urine losses is between 2 and 4 mL/kg/% burn. The actual volume is dependent upon the type of salt solution used. The quantity of sodium ions required for effective resuscitation is of the order of 0.5 mmol/kg/% burn. The resuscitation fluid should preferably be a balanced salt solution with chloride content not exceeding 100 mmol/L and with bicarbonate and/or lactate as the secondary anion. The total water requirement (and sodium concentration of the resuscitation fluid) varies depending on the treatment of the burn wound. The amount of sodium-free water given should be minimized to that required to prevent hypernatraemia.⁴⁸

Burns greater than 15% of the TBSA in an adult and 10% in a child have traditionally been thought to require formal fluid resuscitation; however, large burns are commonly resuscitated in the developing world with oral rehydration using Dioralyte or Moyer's solution (4 g NaCl plus 1.5 g NaHCO₃ per litre of water). In the developed world this would only be recommended in exceptional circumstances, however in the future this may be a way of avoiding excessive fluid administration.

The optimal fluid and formula for resuscitation is still debated and the value of colloid is still unclear. Fluid resuscitation is a dynamic process and all formulas are only guidelines. Constant monitoring of the patient, re-evaluation of progress and adjustment of infusion volumes are the key to success.

The choice of formula depends on local circumstances, experience and expertise. A formula should be easy to understand and regular use will lead to familiarity and confidence. Commonly used resuscitation formulas are given in Table 14.2.

The Parkland formula is commonly used to calculate fluids for the first 24 h (2–4 mL of Ringer's lactate or Hartmann's

Table 14.2 Formulas for fluid resuscitation and ongoing metabolic requirements

Crystalloid resuscitation: Parkland formula	
Adults	2–4 mL × weight (kg) × burn % TBSA = volume given in 1st 24 hours (equates to 0.25 mL/kg/%TBSA/h in 1st 8 h) Using Hartmann's solution or Ringers lactate ½ given in first 8 h, remainder in subsequent 16 h Includes metabolic requirement in adults
Children	As above but add maintenance requirements as dextrose saline or feed 4 mL/kg/h for 1st 10 kg 2 mL/kg/h for 2nd 10 kg 1 mL/kg/h for 3rd 10 kg
Ongoing fluid requirements (after 24 h)	
Adults	1500 mL/m ² BSA (maintenance) + 3000 mL/m ² burn BSA (burn losses) per 24 h
Children	1500 mL/m ² BSA (maintenance) + 3750 mL/m ² burn BSA (burn losses) per 24 h

solution per kilogram body weight per percent TBSA burn with half the volume given in the first 8 h post burn). Resuscitation should use the least amount of fluid required to achieve adequate urine output and other physiological endpoints. Recent concerns about fluid creep have instigated further ongoing debate about fluid resuscitation.⁴⁹

The volume and constitution of fluid therapy in the second 24 h period remain debated, with the use of colloid regaining popularity. Colloid requirements of 0.3 and 0.5 mL/kg/% TBSA burn have been suggested to re-expand the plasma volume following resolution of the capillary leak in most but not all patients.⁵⁰

The concerns about the loss of capillary membrane integrity and leakage of infused proteins into the interstitial space, especially in the lung, has led to the avoidance of colloids in the first 24 h post burn. Some authors argue for maintenance of intravascular volume by the use of early albumin infusion and others advocate an intermediate approach, giving colloids at 8–12 h post injury after capillary leak has resolved.⁵¹

Colloid has traditionally been administered as 5% albumin, however the use of other non-protein colloid solutions, such as dextran, pentastarch or hetastarch, in burn resuscitation has also been described. Recently, concerns about hetastarches have led to their disuse.⁵²

A systematic review of randomized controlled trials on the use of human albumin solution in critically ill patients suggested that albumen administration might be deleterious and increase mortality.⁵³

Hypertonic saline has been repeatedly advocated as a burn resuscitation fluid as the hypertonicity of the infused fluid helps shifts water from the intracellular space into the extracellular compartment. This expands the intravascular space and leads to less fluid administration and less oedema. Unfortunately, this promising approach has not found consensus due to difficulties with composition and concentration of the hypertonic solutions and complications such as hypernatraemia.⁵⁴

Fluid resuscitation endpoints

Endpoints in resuscitation are crucial and will differ for different patients and injuries. Clinical endpoints used routinely in the majority of patients undergoing resuscitation include pulse, blood pressure, respiratory rate, capillary refill, core/peripheral temperature gradient, urine output and osmolality.

The fluid infusion rate should be adjusted to achieve a urine output of 0.5–1 mL/kg/h in adults and 1–1.5 mL/kg/h in children, but it has been debated whether this ideal output should be based on actual body weight or predicted body weight.

Measures such as invasive haemodynamic monitoring of blood pressure, central venous pressure, pulmonary artery wedge pressure and cardiac output can also be used in patients with larger injuries and/or with concomitant smoke inhalation to give more information. Oesophageal Doppler measurement of haemodynamic parameters is a minimally invasive technique that is now routinely used to monitor adequacy of fluid therapy in larger burns.

Serial arterial blood gas estimation with particular reference to the base excess and serum lactate offer an accurate evaluation of adequacy of resuscitation. The persistent elevation or failure to correct these markers of tissue perfusion is associated with increased morbidity and mortality.⁵⁵

Complications of fluid resuscitation

Increased fluid requirements are recognized following delayed or inadequate resuscitation, deep burns, petrol burns, high-voltage electrical burns, alcohol intoxication, extensive deep burns and inhalation injury in children and the elderly.^{56,57}

Under-resuscitation can give rise to hypovolaemia, shock, renal failure, ischaemia–reperfusion injury and multiple organ failure. Over-resuscitation can lead to generalized oedema, pulmonary oedema and compartment syndromes occurring in the limbs and the abdomen.

‘Fluid creep’ can be defined as the unpredictable trend toward ever increasing resuscitation fluid volumes in burn patients. The consequences of increased fluid administration include airway swelling requiring prophylactic intubation, abdominal and extremity compartment syndromes that require escharotomy/fasciotomy and increased mortality.⁵⁸

Intra-abdominal hypertension and the abdominal compartment syndrome have become increasingly reported and are associated with a high morbidity and mortality. Intra-abdominal pressures can be measured by transduction of bladder pressures and intra-abdominal hypertension is defined as pressure greater than or equal to 12 mmHg. Abdominal compartment syndrome can be defined as an intra-abdominal pressure greater than 20 mmHg with new organ dysfunction. Abdominal compartment syndrome is associated with haemodynamic instability, oliguria and renal failure, impaired mechanical ventilation with high peak airway pressures, and a worsening metabolic acidosis.⁵⁹

In major burn injuries, particularly those requiring excessive fluid administration, transduction bladder pressure should be monitored regularly.

Methods to reduce intra-abdominal pressures include extension of anterior trunk escharotomies, neuromuscular relaxants and increased sedation in ventilated and possible judicious use of diuretics in patients demonstrating adequate intravascular volumes. Peritoneal dialysis catheters to remove peritoneal fluid have also been used. If the abdominal compartment syndrome is non-responsive then a decompressive laparotomy or laparoscopy should be considered, although mortality following surgical decompression is high.⁶⁰

It is not entirely clear why ‘fluid creep’ occurs but it may be related to clinical inattention to fluid resuscitation, patients surviving larger/deeper burns that require more fluid, the near abandonment of colloid in the first 24h post injury, a physiological cycle of oedema generation and increased use of opioids.⁵¹

Following fluid resuscitation, burn patients have significant ongoing metabolic fluid requirement due to hypermetabolism, pyrexia and evaporative wound losses. The daily requirement can be calculated and should be given enterally if possible.

Ongoing management

Hypermetabolism and nutrition

Major burn injury impairs the ability to regulate core body temperature, leading to profound heat and evaporative water loss. The hypermetabolic response increases core and skin temperatures 1–2°C above normal in compensation. Patients unable to mount this response are either septic or have exhausted physiological reserve.

Wilmore and colleagues showed that the hypermetabolic response can be attenuated by increasing ambient temperatures to 33°C, regarded as a thermally neutral temperature. Maintaining ambient temperatures between 28°C and 33°C reduces resting energy expenditures in burn patients by a magnitude of 2.⁶¹

Early excision and grafting for major burns has been shown to decrease the metabolic rate for patients totally excised and covered within 3 days of injury compared with those patients excised and covered at one week. The reduction in metabolic rate prevents further net protein loss and catabolism. In addition, early excision has been shown to reduce log bacterial counts in quantitative tissue cultures when compared to late wound excision. This is associated with a reduction in the incidence of sepsis. It can be seen by these mechanisms that early excision and grafting both directly and indirectly attenuates the hypermetabolic response.⁶²

Aggressive, early enteral feeding improves outcomes in severely burned patients. Enteral feeding using 25 kcal/kg body weight and 40 kcal per % TBSA burn per day will maintain body weight in burned adults.⁶³ Children require 1800 kcal/m² plus 2200 kcal/m² of burn area per day.⁶⁴ Enteral nutrition preserves first-pass nutrient in the liver, reduces bacterial translocation with associated sepsis and maintains gut motility.

Enteral nutrition is safer and more effective than parenteral nutrition, which is reserved for those with enteral feeding intolerance or prolonged ileus. Parenteral nutrition alone or in combination leads to overfeeding, liver failure, impaired immune response and increased mortality.⁶⁵

There are many nutritional formulas available to calculate energy requirements of burn patients, however it has been demonstrated that a significant number of these will provide more calories than required (Table 14.3).

By measuring resting energy expenditures, actual caloric requirements can be determined. Overfeeding can lead to significant problems, including elevated respiratory quotients, hypercarbia, increased fat synthesis, fatty deposition in the liver and hyperglycaemia, particularly when excessive carbohydrate is given.⁶⁶ This can give rise to problems with ventilation and prolonged weaning.

Indirect calorimetry can be used to measure resting energy expenditure at the bedside and nutritional calorie requirements can be calculated using 1.2–1.4 times measured resting energy expenditures.⁶⁷

Burn patients require a minimum of 1.5–2 g/kg body weight per day of protein intake to maintain lean body mass. High-fat diets result in protein breakdown and limited lean body mass gains when compared with high-carbohydrate diets consisting of 3% fat, 15% protein and 82% carbohydrate. Burn patients

benefit from a high-carbohydrate diet resulting in increased protein synthesis and improved lean body mass.⁶¹

Burn patients demonstrate significant functional limitations and often struggle to progress with rehabilitation. Exercise training can increase lean body mass, improves strength and stamina and improves overall cardiopulmonary capacity. A 12-week resistance and aerobic exercise training programme has been shown to improve muscle strength, lean body mass and power compared with standard of care.⁶⁸

Pharmacological modulation

Recombinant human growth hormone (rhGH) has been shown to significantly improve outcomes in severely burned children when given intramuscularly at a dose of 0.2 mg/kg/day. Growth hormone was shown to decrease skin graft donor site healing times, length of stay, and total cost of care and improve scar quality.⁶⁹

Unfortunately these benefits have not been demonstrated in adults, where an increased mortality rate in non-burned critically ill adults has been reported, although currently it is thought to be safe to use rhGH in paediatric burn patients to attenuate the hypermetabolic response. The most notable side effect is hyperglycaemia, which requires close monitoring.⁷⁰

Testicular steroid production is decreased in severely burned patients and contributes to the loss of lean body mass seen.

Table 14.3 Formulas for nutritional requirements of burned patients

ADULTS		
1. Calculate BMR		
Patient's age:	Males (kcal/day)	Females (kcal/day)
15–18 years	17.6 W + 656	13.3 W + 690
18–30 years	15.0 W + 690	14.8 W + 485
30–60 years	11.4 W + 870	8.1 W + 842
Over 60 years	11.7 W + 585	9.0 W + 656
2. Adjust BMR for stress		
Multiply by a factor for burn size:		
Partial-thickness burn	Add 1% for each 1% BSA (e.g. for 40% partial thickness × 1.4)	
Full-thickness burn	Add 2% for each 1% BSA (e.g. for 40% full thickness × 1.8)	
To a maximum of 50% BSA		
3. Add a combined factor for activity and diet-induced thermogenesis		
Ventilated patient	×1.1	
Bedbound but not ventilated	×1.2	
Mobile on ward	×1.25	
CHILDREN		
The recommended formulas are based on body surface area:		
Child's age	Daily requirements (kcal/day)	
0–1 year	2100 kcal/m ² (SA) + 2100 kcal/m ² (BSA)	
1–11 years	1800 kcal/m ² (SA) + 1300 kcal/m ² (BSA)	
12–18 years	1500 kcal/m ² (SA) + 1500 kcal/m ² (BSA)	
1. Formula for body surface area (SA):	SA (m ²) = √[(height (cm) × weight (kg))/3600]	
Or use surface area nomogram		
2. Formula for BSA in calculation:	BSA = [% burn × body surface area (SA)]/100	

W, weight in kg; BSA, burn surface area.

Testosterone can only be given by intramuscular injections and has a virilizing effect, limiting its use. Oxandrolone, a synthetic analogue, offers only 5% of the masculinizing effects of testosterone, and is safe for both genders. Oxandrolone improves net muscle protein synthesis and protein metabolism in severely burned patients when administered at a dose of 0.1 mg/kg twice daily. It has been shown to improve donor site healing times, decrease length of stay, increase protein synthesis, increase lean body mass, increase bone mineral content and increase muscle strength.⁷¹

Propranolol used at a dose of 0.5–4 mg/kg per day to reduce resting heart rates by 10–20% blocks beta-adrenergic stimulation, reducing the effects of elevated levels of catecholamines. This decreases tachycardia, cardiac work and basal metabolic rate. It also prevents increased peripheral lipolysis, decreases fatty infiltration of the liver and inhibits the release of free fatty acids from adipose tissue. In addition, it increases lean body mass and decreases skeletal muscle wasting.⁷²

Hyperglycaemia leads to muscle protein catabolism, reduced graft take, and increased morbidity and mortality. Intensive insulin therapy of 400–900 mU/mL to maintain euglycaemia for 7 days has been shown to stimulate muscle protein synthesis and increase lean body mass, improve donor site wound healing and reduce hospital stay. Less rigorous control of glucose levels with insulin has demonstrated similar benefits with a decreased risk of treatment-related hypoglycaemia, which approaches 20% with high-dose insulin regimes.⁷³

Other drugs such as fenofibrate, glucagon-like peptide-1 and ketoconazole have also been investigated for attenuation of the hypermetabolic response.⁶¹

Pain

Burn injury is usually associated with significant amounts of pain and anxiety and pain control in burn patients is essential and difficult task that is often inadequately performed. Adequate pain relief must be given both for background pain and procedural pain control. Anxiety can be equally debilitating and can be controlled with anxiolytics. Requirements for pain relief, sedation and anxiolysis vary depending on the individual patient, the size, site and stage post injury. Pharmacological techniques include the use of opiates for pain and benzodiazepines for sedation and anxiolysis. Ketamine, a neuroleptic agent, is particularly useful for procedures especially in children and can be given orally using nurse-led protocols. Centrally acting agents such as the tricyclic antidepressants, clonidine and gabapentin have a role to play in modulation of the pain response. Non-steroidal anti-inflammatory agents are useful but care must be taken in the acute phase and in elderly patients due to the risk of renal impairment and gastrointestinal haemorrhage.

Pain and anxiety can be measured using scoring systems. Regular, ongoing and documented pain assessment is vital to

the ongoing management of burn pain. Children can pose a particularly difficult management problem. Large doses of analgesics and sedatives are sometimes required due to hypermetabolism of the drugs and tolerance. Non-pharmacological interventions such as relaxation therapy, hypnotherapy and distraction can also be useful in helping control pain and anxiety.

Pain specialists are an integral part of the modern burn multidisciplinary team and pain control must be given a high clinical priority.⁷⁴

Sepsis, antibiotics and topical antimicrobials

Infection is a common, serious and potentially life-threatening complication following burn injury. The reported incidence of nosocomial infections varies between 63 and 240 per 1000 patients and 53–93 per 1000 patient days. Infections are associated with multiple organ failure, increased morbidity and mortality.⁷⁵

The burn wound is usually sterile for the first 6–12 h following the injury. It becomes contaminated then colonized with bacteria.⁷⁶ In the first week or so following injury gram-positive bacteria predominate with gram-negative bacterial infections being most common after that time. The source of these microorganisms is thought to be exogenous from the environment or cross-infection from staff. Alternatively, it can be endogenous from adjacent colonized uninjured skin, bacterial translocation from the gut or from direct faecal self-contamination. Burn patients are known to be immunocompromised and to have a high incidence of nosocomial infection. They often require intensive care support, making them prone to ventilator-associated pneumonia and catheter-related infections.⁷⁷

Microbiological surveillance of the burn wound is essential. This involves regular swabbing of wounds and cultures of sputum, urine and blood. Wound swabs inform clinicians as to what surface organisms are present but do not always inform as to what is happening at the eschar/viable tissue interface. Quantitative microbiology is thought to be invaluable in determination of numbers of organisms per gram of tissue but is labour intensive and may be prone to sampling error. It is common to find 10^3 – 10^5 organisms per gram of burn tissue and counts greater than 10^5 are indicative of invasive infection.⁷⁸

The current literature suggests that prophylaxis with systemic antibiotics should not be given to patients with severe burns. This consensus is based on the lack of evidence of benefit and risk of adverse effects. A recent meta-analysis of antibiotic prophylaxis indicated a reduction in the mortality risk in burn patients although the methodological quality of the data analysed was questionable.⁷⁹

Antibiotic prophylaxis may be given to patients undergoing surgical procedures when there is a significant incidence of bacteraemia. The antibiotic given and duration of therapy depend on the time following the injury, the surgical procedure being

Box 14.2 Definition of sepsis and infection in burn patients includes at least three of the following parameters. Source: Greenhalgh et al., 2007.⁸⁰ Reproduced with permission of Lippincott Williams & Wilkins.

- Temperature >102.2°F (39°C) or <97.7°F (36.5°C)
 - Progressive tachycardia (e.g. adults >90 beats per min; children >2 standard deviations [SD] above age-specific normal values)
 - Progressive tachypnoea (e.g. adults >30 breaths per min; children >2 SD above age-specific normal values)
 - Refractory hypotension (e.g. adults: systolic blood pressure <90 mmHg or a decrease >40 mmHg, or mean arterial pressure <70 mmHg; children <2 SD below normal)
 - Leukocytosis (e.g. adult >12 000 white cells/μL, children >2 SD above normal) or leukocytopenia (e.g. <4000 white blood cells/μL)
 - Thrombocytopenia that occurs 3 days after resuscitation (e.g. adults <100 000 platelets per μL; children <2 SD below age-specific normal values)
 - Hyperglycaemia >110 mg/dL (6.1 mmol/L) in the absence of pre-existing diabetes mellitus
 - Inability to tolerate enteral feedings for more than 24 h based upon:
 - ♦ abdominal distention
 - ♦ residual volumes (two times the feeding rate in adults and >150 mL/h in children)
 - ♦ uncontrollable diarrhoea (>2500 mL/day for adults and >400 mL/day for children)
- The infection must be documented by one of the following evaluations:
- Infection is confirmed on a culture (e.g. wound, blood, urine), or
 - Pathologic tissue source is identified (>10⁵ bacteria on quantitative wound tissue biopsy or microbial invasion on biopsy), or
 - A clinical response to antimicrobial administration is documented.

undertaken and the results of microbiological surveillance and advice from a microbiologist.

The diagnosis of sepsis can be difficult in the burn patient. They are typically hypermetabolic, hyperpyrexial, tachycardic, tachypnoeic, and have an elevated white blood cell count. Clinical indicators of burn sepsis are given in Box 14.2.⁸⁰

Examination of the burn wound can reveal erythema, progressive focal necrosis, eschar separation, punctate haemorrhages and progression of burn wound depth. In addition, wound odour and subeschar pus may be present. Ecthyma gangrenosum, an aggressive pseudomonal infection, can present with black lesions in the wound and surrounding skin.

Blood cultures typically have a low yield rate but although approximately 20% of patients in a burn ICU will have positive cultures, these are associated with 20% mortality. Such positive cultures can be difficult to interpret due to contamination with commensal organisms.⁸¹

Vascular access in patients can be a significant challenge and a risk in terms of catheter-related bloodstream infections. Central venous catheters in burn patients are changed every 7 days or so following insertion, either to a new site or over a guidewire, with a recent study showing no difference in the incidence of bloodstream infections between the two methods.⁸² Other studies have shown increasing incidence of sepsis associated with increasing number of line days and line changes with no difference between line sites.⁸³

Intensive care bundles of care have been popularized and have benefits in burn patients in the ICU. Implementing care bundles into the burn critical care setting has been associated with reduction in ventilator-associated pneumonias, bloodstream infection rates and a lower mortality.⁸⁴

Suppurative thrombophlebitis is a rare but serious infective complication of peripheral intravenous cannulas. It typically gives rise to a clinical picture of sepsis and persistent positive blood cultures with skip lesions of infected foci with normal vein in-between. Pus can be expressed from the affected vein and treatment requires excision of the vein.⁸⁵

The management of systemic sepsis involves fluid resuscitation, immediate intravenous antibiotics and source control in the form of excision of infected wounds. Antibiotic therapy should be guided by previous cultures and sensitivities and close liaison with the microbiologist is essential. Care must be taken to avoid the use of multiple broad-spectrum antibiotics that can lead to the emergence of resistant organisms.

Infection control is a very important part of care within a burns unit. Cross-infection should not be tolerated and can be eliminated by effective infection control and cohort nursing. Washing wounds in a shower reduces the bacterial load and topical antimicrobials limit bacterial growth. The introduction of topical antimicrobial agents in burn care in the 1970s was thought to lead to a significant reduction in burn mortality, but recent systematic reviews have highlighted the paucity of data.⁸⁶

Topical agents currently used include silver-containing products active against *Staphylococcus aureus*, *Pseudomonas aeruginosa*, enteric organisms and *Candida albicans*.

Povidone-iodine has broad antibacterial and antifungal activity but a short duration of activity. Mupirocin (Bactroban®) is effective against gram-positive organisms (especially MRSA) with some activity against gram-negative organisms. Nystatin is an effective antifungal agent that can be used topically to reduce fungal wound infection and orally to prevent gut colonization. Hypochlorite has a broad spectrum and is bactericidal against *Ps. aeruginosa*, *Staph. aureus* and other gram-positive and negative organisms. Used at lower concentrations (0.025%) it is toxic to bacteria but not to human cells.⁸⁷

Complications

Patients with major burn injury are a high-risk group for developing complications.

Goal-directed resuscitation, early wound excision and recognition and modulation of the hypermetabolic response have reduced morbidity and mortality. Multiple organ dysfunction in burn injuries >20% TBSA has been reported to have an incidence of 40–60% and mortality rates are proportional to the numbers of organ failures.⁸⁸ The majority of complications are related to sepsis and the systemic inflammatory response, although specific complications can occur in each physiological system.

Organ dysfunction

Patients with a significant burn injury with or without smoke inhalation will exhibit a spectrum of clinical manifestations of systemic inflammation that range from systemic inflammatory response syndrome (SIRS) through to sepsis, severe sepsis and septic shock (Table 14.4).

Tissue damage is the precipitating event for systemic inflammation caused by direct thermal injury or by ischaemia–reperfusion. Tissue injury results in the release of proinflammatory cytokines such as tumour necrosis factor alpha, interleukins IL-1, IL-6, IL-8, IL-12, IL-18 and interferon beta. Other causes of SIRS include sepsis, ischaemia–reperfusion injury, multiple trauma and pancreatitis.¹⁷

Some patients with SIRS will go on to develop multiple organ dysfunction syndrome (MODS), where non-fatal organ dysfunction develops, such as renal insufficiency, inotrope or ventilator dependence. Some of these patients with MODS will go on to full blown multiple system organ failure (MSOF), which is often fatal. It is not clear what factors escalate this chain of events, but gut failure with repeated bouts of bacterial translocation has been postulated as one cause.⁸⁸

Cardiac function alters in a number of ways, including initial reduction in preload with myocardial depression leading to decreased in cardiac output. A hyperdynamic phase follows, characterized by decreased afterload due to a lowered systemic vascular resistance and a subsequent increased cardiac output. Depending on the clinical picture, fluid administration to increase intravascular volume and/or inotropic support may be required.¹⁷

Initially following burn injury coagulation factors are diluted and consumed, leading to derangement of clotting indices and increased fibrin degradation products, although frank coagulopathy is uncommon. The coagulation cascade is activated; fibrin, factors V and VIII increase and antithrombin deficiency is common. Later on there is tendency for patients to be hypercoagulable, leading to deep vein thrombosis and pulmonary embolism.⁹⁰ Thrombocytosis in the later stages of wound healing and recovery is common. Venous thromboembolic prophylaxis should be considered following the initial phase of burn injury. The placement of precious central venous catheters must be carefully considers as the femoral site, which is often the only available vascular access site in a

major burn, has been associated with a significant incidence of thrombosis.

Mechanical ventilation in a burn patient may be required for upper and lower airway inhalation injury, although respiratory failure and hypoxia due to acute lung injury can also occur in burn patients without inhalation injury. This can lead to acute respiratory distress syndrome (ARDS). This clinical condition presents as dyspnoea, severe hypoxia and decreased lung compliance with radiological changes of bilateral diffuse pulmonary infiltrates. Burn patients can sustain lung injury both directly following smoke inhalation and also because of the systemic inflammation triggered by the cutaneous burn injury.

Typically, a patient with a severe burn with or without an inhalation injury will be intubated for airway compromise or for operation. At 4–5 days following injury oxygenation will deteriorate, requiring higher inspired oxygen concentrations to prevent hypoxia. Lung compliance will increase, requiring higher inflation pressures, and at 7 days post burn lung infiltrates will appear on the chest X-ray.

Treatment is mainly supportive until the fibrotic process ceases. Protective ventilation strategies should be employed to reduce barotrauma.⁹¹ Steroid treatment has been shown to be of no benefit in prevention of ARDS following smoke inhalation injury.

Pneumonia is a common complication following smoke inhalation injury and is usually caused by a variety of organisms including *Ps. aeruginosa*, *Klebsiella pneumoniae* and *Staph. aureus*. Prophylactic antibiotics do not prevent infection in patients and are a risk for promoting growth of multiple drug-resistant organisms.⁹²

Diagnosis of pulmonary infection following smoke inhalation can be difficult, as many patients will have a systemic response. However clinical signs and symptoms, deteriorating gas exchange, worsening chest X-ray appearance, sputum cultures and bronchoalveolar lavage are all important features in the diagnosis.

Once the diagnosis is made or considered, early institution of broad-spectrum antibiotics, close liaison with the microbiologist and antibiotic stewardship to match organism sensitivities are crucial.

Long-term airway and pulmonary complications can result from smoke inhalation injury and its treatment.⁹³ These include glottic, subglottic and tracheo-bronchial complications. Pulmonary

Table 14.4 Definitions of SIRS, sepsis, severe sepsis, septic shock and MODS

SIRS Criteria (≥2 meets SIRS definition)	Temp >38°C (100.4°F) or <36°C (96.8°F) Heart rate >90 Respiratory rate >20 or PaCO ₂ <32 mmHg WBC >12 000/mm ³ , <4000/mm ³ , or >10% bands Suspected or present source of infection
Sepsis criteria (SIRS + source of infection)	Lactic acidosis, SBP <90 or SBP drop ≥40 mmHg of normal
Severe sepsis criteria (organ dysfunction, hypotension, or hypoperfusion)	Severe sepsis with hypotension, despite adequate fluid resuscitation
Septic shock criteria	Evidence of ≥2 organs Failing
Multiple organ dysfunction syndrome (MODS) criteria	

Source: Dellinger et al., 2012.⁸⁹ Reproduced with permission of Lippincott Williams & Wilkins.

function test, laryngoscopy and bronchoscopy allow detection and management of the complications. The plastic surgeon must be mindful and alert to these issues when planning surgical reconstruction requiring general anaesthesia.

Gastrointestinal events that initially follow burn injury include splanchnic hypoperfusion, mucosal atrophy, altered absorption and increased intestinal permeability. These all lead to diminished gut barrier function. Feeding intolerance, mucosal ulceration and bleeding from the stomach and duodenum manifest this. Diminished gut barrier function is also thought to lead to bacterial translocation, which stimulates the systemic inflammatory response.⁹⁴

These changes can be attenuated by judicious fluid resuscitation and early enteral nutrition. Early enteral feeding increases splanchnic blood flow, maintains pyloric function and intestinal motility and reduces infective complications.⁹⁵

Poorly perfused organs lead to acidosis, leading to increased fluid administration. Overzealous fluid administration can lead to intra-abdominal hypertension (>15 mmHg) and compartment syndrome (>20 mmHg).⁵⁹

Stress ulcer prophylaxis is also maintained by either reduction of gastric acid production with histamine blockers (ranitidine) or proton pump inhibitors (omeprazole). Gastrointestinal haemorrhage in a burn patient is now uncommon.

Renal failure due to failure of initial resuscitation is uncommon, however is more commonly seen as part of a multiple organ failure syndrome. The incidence is reported to be up to 20%, with an incidence of renal failure requiring renal replacement being 15%. The mortality associated with renal failure following burn injury has been reported to be from 50 to 100%.⁹⁶

In major full-thickness injury and high-voltage electrical injury, red blood cell and muscle destruction can lead to haemoglobinuria, myoglobinuria and acute tubular necrosis. This can be avoided by aggressive fluid resuscitation aiming for a urine output of 1–2 mL/kg per h. Additional therapies include alkalization of the urine and the use of an osmotic diuretic (mannitol).

Later onset renal failure is usually part of multiple organ failure secondary to sepsis and/or the systemic inflammatory response. It can also occur following administration of nephrotoxic drugs.

Management involves treatment of the underlying cause and early renal replacement therapy, either by haemofiltration or dialysis. The mortality associated with renal failure remains high, especially in the group associated with multiple organ failure.

Electrolyte disturbances are common following major burn injury. Hypernatraemia in the resuscitative phase is usually indicative of both over- and under-resuscitation. Hyponatraemia is not uncommon following resuscitation of children and is usually due to over zealous administration of hypotonic solutions such as 5% dextrose or dextrose saline. It can lead to hyperpyrexia, cerebral oedema and convulsions. In the late burn phase

hypernatraemia may occur due to excessive water losses and inadequate replacement. Hyperchloraemia is not uncommon following resuscitation, due to the significant amount of chloride-containing fluids administered. Burn patients have a significant requirement for phosphate and magnesium due to exudative losses and urinary excretion.⁹⁷

Hypocalcaemia can occur following resuscitation and requires intravenous supplementation. Hypercalcaemia is underrecognized, can be associated with renal failure and has been associated with increased mortality.⁹⁸

It is important to ensure that metabolic requirements following a burn are given enterally and that particular attention is paid to fluid balance on a daily basis.

Great care must be taken in positioning and prevention of peripheral nerve compression during the acute care of burn patients. Isolated peripheral neuropathy, either idiopathic or due to entrapment or compression, can hinder functional outcome. In addition, patients surviving major burn injury can develop a generalized critical care neuropathy characterized by global weakness that can prolong ventilator dependence, mobilization and rehabilitation. Risk factors for critical care neuropathy include multiple organ failure, muscular inactivity, hyperglycaemia and use of neuromuscular blockers. Early mobilization and exercise may help prevent or reduce severity of this problem.⁹⁹

Patients surviving major burn injury become osteopenic due to prolonged immobilization and hypermetabolism. This is as a result of alterations in calcium and phosphorus metabolism. Bone formation is reduced, with a reduction in bone mineral density, leading to an increased incidence of fracture, decreased growth velocity and growth stunting.¹⁰⁰

In the recovery phase, heterotopic ossification can cause periarthral ossification and limitation of movement, which can hinder rehabilitation. An aggressive approach of early surgery and wound healing with early active mobilization has reduced the incidence of this problem.

Wound management

Treatment planning

In general, burn injury and its attendant consequences are directly related to the size and depth of the burn wound. It is evident that treatment planning is key to successful management of burn injury.

The management plan depends on the depth and size of the injury, the anatomical distribution, the age and comorbidities of the patient and involves both conservative and surgical options.

Superficial partial-thickness wounds require a conservative approach, whereas an aggressive surgical approach is best for deeper wounds. The aim of such a treatment plan is to improve outcome and reduce morbidity in smaller injuries and to improve survival in bigger injuries.

Superficial partial-thickness wounds

The aim of management of superficial partial-thickness burn wounds is to promote rapid spontaneous re-epithelialization with the minimum number of painful dressing changes. In addition, prevention of infection to minimize deepening of the burn wound and subsequent hypertrophic scarring are also important.

The wounds can be treated with conventional dressings, either impregnated or free from a topical antimicrobial agent, with topical antimicrobials, with biological or semi-biological dressings or by exposure.

Conventional dressings, such as Vaseline gauze or silicone sheets (Mepitel®), can be used to dress the wound while re-epithelialization occurs and are useful for small burns (less than 5% TBSA). These dressings tend to require frequent changes which can be painful. Many synthetic dressings, such as Duoderm®, Omniderm®, Tegaderm® and hydrocolloids have also been used with success to dress such wounds. A recent Cochrane review highlighted the paucity of high-quality data on dressings for superficial and partial-thickness burn injury and that the available evidence was of limited usefulness in aiding clinicians to choosing suitable treatments.¹⁰¹

The antimicrobial properties of silver have been long recognized and many silver-containing dressings are currently used for the management of burn wounds. Traditionally, topical 1% silver sulfadiazine (Flamazine®/Silvadene®) cream has been the mainstay of treatment for these wounds. Following cleaning and debridement of the wound, the cream is applied and covered with a conventional dressing. The dressings are changed once or twice daily until re-epithelialization occurs and the wound is healed.

Acticoat® is a nanocrystalline silver dressing that can provide sustained release of silver for up to 7 days, with studies showing Acticoat to have better antimicrobial activity than silver sulfadiazine. Other studies have suggested Acticoat having fewer adverse effects, reducing healing times, improved ease of application and low frequency of dressing changes.¹⁰²

Treatment with a topical antimicrobial is useful for patients presenting late after injury with a colonized wound.

A biological dressing can be defined as the use of human or animal tissue for temporary wound covering. A semi-biological dressing refers to where a component of the dressing is derived from such tissue (i.e. collagen).¹⁰³

Biobrane® is a semi-biological dressing made up of a fenestrated silicone layer bonded to a nylon mesh that has been impregnated with type I porcine collagen. Repeated studies have shown it to reduce pain, inpatient stay and time to healing.¹⁰⁴ Biobrane® has been used extensively in patients with superficial partial-thickness burns presenting within 24h of their injury. Under sedation or an anaesthetic the burn blisters are cleaned, deroofed and all burned epithelium is removed. Biobrane is then applied to the wound so that it is closely adherent. The Biobrane can be secured by histoacryl glue, sterile Hypafix® tape or with staples. A standard secondary dressing of rolled gauze covered by elastic bandages is then applied. The outer

dressings are removed at 24–48h to inspect the wound. Oral antibiotics with staphylococcal coverage are usually given. As re-epithelialization occurs in 10–14 days, the Biobrane spontaneously separates from the healed wound. If wound infection supervenes, the Biobrane rapidly becomes non-adherent. Purulent exudate produced by the wound is then readily visible. Biobrane is not used in patients with delayed presentation and potential colonization arriving more than 24–36h following injury. Biobrane is also relatively expensive compared to other dressing modalities.¹⁰⁴

Other biological dressings such as allograft skin, xenograft skin (porcine) and human amnion are used in a similar fashion to Biobrane to physiologically close the superficial partial-thickness wound while re-epithelialization occurs. The problems associated with the use of these products include availability, collection, storage, transmission of infection and cost.¹⁰³

Exposure of the burn wound has fallen out of favour but has been extensively used in the past. After cleaning and debridement, wounds are nursed exposed in a warm dry environment and left to crust over. The coagulum formed separates as re-epithelialization proceeds until healing occurs. Exposure is comfortable and dressing changes are not required. Disadvantages include prolonged inpatient treatment, requirement of a specialized humidified environment, electrolyte disturbances and higher infection rates. This technique has now generally been abandoned apart for specific areas that are difficult to dress, such as the face, genitalia and perineum.

Large superficial partial-thickness injuries (>40% TBSA) carry a higher risk of wound contamination and infection, leading to organ dysfunction and morbidity.

The use of allograft applied within 24h of the injury has been reported as improving outcome. Following cleaning and debridement of loose epidermis under anaesthesia, allograft split-skin grafts are placed over the open dermal wound. Similarly a mid-dermal burn injury can be debrided with a dermatome at a depth of 10–15/1000 inch and allograft applied. Standard graft dressings are applied and spontaneous healing should occur with allograft separation as the wound epithelializes.¹⁰⁵

Xenograft skin¹⁰⁶ and Biobrane can be used in a similar fashion but are associated with an increased risk of desiccation, infection and deepening of the burn wound. Topical antimicrobials such as silver sulfadiazine or Acticoat can be used for this type of wound, especially for wounds that present late and are colonized. Dressing changes in these circumstances can be painful. There is a significant incidence of wound sepsis and deepening of the burn wound necessitating skin grafting.

Deep partial-thickness wounds

Deep partial-thickness burns are associated with significant morbidity in terms of time to healing, infective complications and subsequent scarring. Conservative management leading to spontaneous healing usually involves prolonged and painful dressing changes and the resultant scar is invariably hypertrophic.

This leads to disfigurement and functional disability. An optimal approach is early surgery that tries to preserve dermis and achieve prompt wound healing.¹⁰⁷

Burns that are deemed to be deep partial thickness in nature are best tangentially excised and the wound covered with autologous split-skin grafts that usually require meshing. Cosmetically and functionally sensitive areas such as the face and hands need thicker sheet autograft for wound closure.

Allograft, xenograft or other biological or semi-biological dressings may be required for temporary wound closure if donor sites are scarce, to allow time for donor site healing. Alternatively serial wound excision with conservative management of the unexcised wound can be used for larger burns where donor sites are scarce. Unexcised areas are treated with topical antimicrobials until donor sites can be reharvested, usually at 7–14 days.

The unexcised and unhealed areas of burn wound are susceptible to invasive wound infection, with associated increase in morbidity and mortality. Topical Flammacerium® (silver sulfadiazine and cerium nitrate) has been reported as decreasing episodes of invasive wound infection, morbidity and mortality with this conservative method of treatment.¹⁰⁸

Alternatively, deep partial-thickness wounds can be treated with Acticoat; alternate day to weekly dressing changes can be used. Daily or twice daily application of silver sulfadiazine until wound healing is achieved has largely been abandoned for this type of wound. The wounds may take 4–6 weeks to heal, with an a higher incidence of invasive wound infection, deepening of the wound and hypertrophic scarring. More conservative methods are reserved for patients who are unfit for surgical intervention and for smaller burns in functionally and cosmetically unimportant areas.

Full-thickness wounds

Full-thickness burns require excision and will not heal spontaneously unless they are very small. The resultant wounds require closure to reduce the risks of invasive infection and systemic sepsis and invariably require skin grafting. Prompt excision and wound closure reduces morbidity and mortality in patients with such injuries.^{109,110}

These wounds should be excised in a tangential fashion wound and closure obtained with split-thickness autograft. Sheet autograft is used for functionally and cosmetically sensitive areas such as the hands and face. Meshed autograft is used if larger areas need closure. Grafts are secured to the wounds by staples or absorbable sutures and are dressed in the standard fashion. Grafts and wound inspection is undertaken on the second day if the initial wound was infected or heavily colonized or on the fifth day if not.

Patients who are elderly or unfit for surgical intervention can undergo conservative management with topical antimicrobials. The antimicrobial agent – Acticoat® or silver sulfadiazine – is applied regularly until the burn eschar separates and a granulating wound is present. This usually takes approximately

3–4 weeks to occur and sometimes longer. This granulating wound can then be covered with autograft to achieve wound closure. Other topical agents such as Flaminal® or Nexobrid® can also be used to aid eschar separation. In certain cases small wounds less than 5 cm diameter can be left to heal spontaneously by wound contraction and epithelialization from the wound margins.

In larger injuries (>10% TBSA) most authors agree that early excision of the burn wound is the treatment of choice. However, the timing varies from immediate excision soon after arrival in the burn service to staged excision over the first 7 or so days following admission. Unfortunately, no evidence exists as to optimal timing or extent of early wound excision.

Total early excision of the burn wound, within 24 h of injury, and physiologic wound closure with split-skin autograft, allograft and/or synthetic skin substitute approach has been shown to improve mortality in selected adult patient groups.¹¹¹ It needs a coordinated approach from the surgical and anaesthetic teams, and timing of surgery post injury is critical as blood loss in the 24 h post burn has been shown to be half that of surgery after this time.¹¹²

Following total wound excision, the whole wound must be physiologically closed with auto- or allograft or a synthetic skin substitute. Autograft is widely meshed and used with allograft overlay in a sandwich technique. Areas not closed with autograft have allograft applied to close the wound. If wound closure cannot be achieved primarily with autograft, the patient returns to the operating room when the donor sites are ready for reharvesting, at which time the allograft is changed and further autograft is applied. This is usually done in stages on a weekly basis until the whole wound is closed with autograft.

A more traditional conservative surgical approach has been described above for deep partial-thickness burns and entails excision of as much of the wound that can be covered with available autograft, with the unexcised areas of burn treated with topical antimicrobials. This method of treatment has a higher morbidity and mortality in larger injuries and has generally been abandoned.

Preoperative planning is essential with anatomical areas to be debrided, technique used for debridement and choice of donor sites being the key elements in the treatment plan. It is essential to estimate the amount of autograft that will be required, which areas are priorities for autograft coverage and any required mesh ratios.

In smaller burns where donor sites and available graft are plentiful, the focus of treatment should be to minimize donor site morbidity and to maximize functional and cosmetic outcome. Sheet grafts are preferred and preferential harvest of cosmetically hidden areas such as the upper thighs, buttocks and scalp should be considered. The scalp is an attractive donor site, as subsequent hair growth completely hides any marks. In patients with major burn injury, choice of donor sites is limited and skin grafts should be harvested from any available site as

priority is given to cover large areas with available autograft in the smallest number of operations.

Techniques of wound excision

Wound excision is key to accurate removal of non-viable tissue. Preservation of viable dermis is important in partial-thickness injury, whereas in full-thickness injury all necrotic and infected tissue must be removed. The aim of debridement is to leave a viable wound bed of dermis, fat, fascia or muscle that will accept a skin graft.

Enzymatic wound debridement has recently been regaining popularity, with the development of newer agents that have been shown to be effective.¹¹³

The combination of non-surgical debridement techniques with cellular skin replacement therapies seems to be the logical direction of travel for future burn wound management.

Tangential excision of the burn wound was described by Janzekovic in the 1970s and involves repeated shaving of partial-thickness burns in a tangential fashion until a viable dermal bed, manifested clinically by punctate bleeding, is achieved.¹⁰⁷ If the wound extends down into fat, its colour can be helpful as a sign of viability ('red fat being dead fat') in addition to punctuate bleeding and thrombosed vessels. After haemostasis the wound is ready for grafting. Full-thickness wound excision can also be achieved using sharp excision with a knife or with electrocautery. The plane of excision runs between viable and non-viable tissue.

Water jet hydro surgery (Versajet®) has been described for burn wound excision, mainly in partial-thickness wounds.¹¹⁴ Authors cite precision and dermal preservation as the main benefits associated with this technique, with a randomized trial reporting equivalent adequacy of debridement and a shorter surgical time for the Versajet.¹¹⁵

Fascial excision involving surgical excision of the full thickness of the integument is reserved for deep burns extending down through the fat into muscle, late-presentation infected wounds and for failed initial surgery in patients with life-threatening invasive infections. It is undertaken with electrocautery and offers excellent control of blood loss, leaving a fascial wound bed which is an excellent for graft take. Unfortunately fascial excision is mutilating and leaves a permanent contour defect.

Avulsion can be used for conservatively treated deeper wounds. Physical removal of the necrotic eschar by avulsion from the underlying viable wound bed can be rapidly accomplished with minimal bleeding.

Primary amputation must be considered in the management of high-voltage electrical injuries or very deep thermal injuries with extensive muscle involvement and rhabdomyolysis which is life threatening. Limb salvage should always be attempted to preserve length and maximize function of the extremity. Amputation is reserved for patients who have an ischaemic limb or refractory invasive infection following repeated debridement. In other circumstances amputation is undertaken only if all other measures to preserve a useful functioning limb have failed.

Table 14.5 Estimated approximate blood loss given in millilitres per square centimetre burn excised in patients >30% TBSA burn

Day post injury	Estimated blood loss (mL/cm ²)
0–1	0.4
1–2	0.6
2–16	0.75
>16	0.5

Source: Desai *et al.*, 1990.¹¹¹ Reproduced with permission of Lippincott Williams & Wilkins.

Blood loss

Blood loss during burn surgery can be massive and the amount required can be estimated. Blood loss depends on timing of the surgery following injury and the area of wound that requires excision and autografting. To estimate blood requirements pre-operatively the burn size in square centimetres should be estimated. A calculation can then be made if the time post injury is known (Table 14.5).¹¹²

Blood and blood products must be ordered and present in the operating theatre prior to the commencement of surgery. Incorporating this into the World Health Organization patient safety checklist should minimize complications of blood loss.¹¹⁶

Blood loss during surgery can be minimized by the use of sterile limb tourniquets, infiltration of the eschar and donor sites with 1/10⁶ solution of adrenaline (1 mg in 1000 mL N saline) and warm topical phenylephrine (2% solution)-soaked dressings. It is possible to debride and excise significant wounds without the need for blood transfusion.

Operative approach and anatomical considerations

The sequence of wound excision and cover depends on surgeon and institutional preference. In large injuries the sequence usually is the posterior trunk, anterior trunk, lower limbs, upper limbs, and head and neck in order. This sequence maximizes the area covered with autograft early in the course of treatment and allows for early mobilization and ambulation. In general, sheet grafts should be used whenever possible. Care must be taken when mesh grafts are used as contracture can occur in the line of the interstices. Mesh graft interstices on the trunk and breast should be placed horizontally. Care should be taken to preserve the breast or breast bud, especially in females, and to place enough skin with minimal mesh expansion into the inframammary folds and the sternal area to try and reduce subsequent breast deformity. The umbilicus should be preserved if possible.

The buttocks are difficult to manage and skin graft take is poor. They are prone to faecal soiling and shearing and can be the site of repeated bouts of invasive wound sepsis. It is often

worth autografting them in the first operation, but graft take can be disappointing. If the grafts fail it is then best to leave the area for a time when the patient can be nursed prone while the grafts take. This is usually done after all other areas are healed. It is not usually necessary to perform a colostomy to prevent faecal soiling as the faecal stream can be diverted with a rectal tube. The perineum and genitalia are usually managed conservatively, with grafting of any unhealed areas later on in the course of surgical treatment.

Mesh graft interstices on the limbs should run longitudinally along the line of the limb except at the joints. At the knee, ankle, axilla, elbow and wrist joints graft expansion should be minimized if possible and the direction of the interstices should be the same as the axis of rotation of the joint (i.e. perpendicular to the longitudinal axis of the limb).

The skin on the sole of the foot is glabrous skin and is very thick and specialized. It will commonly re-epithelialize despite what initially seems a full-thickness injury. The sole of the foot is best treated conservatively until it is apparent that spontaneous re-epithelialization will not occur. In contrast, the skin on the dorsal aspect of the foot is very thin and often requires grafting. It is important not to use widely meshed skin in this area as any significant hypertrophic scarring can cause difficulties with weight-bearing ambulation and fitting of shoes.

The burned hand requires attention to detail to achieve optimal functional and cosmetic results. The volar aspect of the hand is covered with specialized glabrous skin, which usually heals and it is best to avoid grafting it if possible. The dorsal skin is thin and usually requires grafting in deep burns. In general, sheet graft is preferable to mesh graft and is best secured with absorbable sutures. Mesh interstices should run longitudinally along the hand and digits. The key to functional success is early mobilization of the hand. Initially after grafting the hand is dressed and splinted in the position of safety with the metacarpophalangeal joints flexed at 70–90°, the interphalangeal joints at 180°, the wrist in neutral or slightly extended and the thumb flexed and adducted at the metacarpophalangeal joint. The grafts are inspected at 5 days and, if stable, mobilization can be started. If sheet grafts are used they can be exposed with no dressing during mobilization during the day with splintage at night.

In patients with large burns, repeated application of allograft may be required until it is time to autograft the hands. It can be very difficult to maintain the position of safety of the hand during this phase and K-wires through the metacarpophalangeal and interphalangeal joints may be required to maintain the joints in optimal position.

Deeper burns of the face will often benefit from early excision and autografting. Medium to thick sheet split-skin autograft should be used for the face and applied in cosmetic units to place marginal scars in natural skin crease lines. If available, donor sites should be above the neck for optimum colour and texture match. The scalp is an excellent donor site for grafts destined for the face. The use of skin substitutes such as Integra® have been described as giving superior cosmetic outcomes.

Early excision and closure of eyelid burns reduces the incidence of corneal injury secondary to exposure and should be a priority. When applying autograft to the eyelids, thick split-thickness graft should be used and overcorrection should be performed, putting more skin in than seems to be needed as contraction can lead to corneal exposure.

Wound closure

Following wound excision it is vital to obtain wound closure to reduce invasive infection, evaporative water loss, heat loss and pain. Wound closure can be permanent using autologous split-skin graft and is considered the gold standard. Temporary physiological closure using allograft or skin substitutes allows time for donor site healing.

Although autologous split-skin grafts are considered the gold standard for resurfacing burns. Full-thickness grafts tend not to be used for acute burns and are usually reserved for postburn reconstruction.

Split-skin grafts have a more reliable 'take' and can be harvested either with a hand knife or with a powered dermatome. They can be used as sheet grafts or can be meshed using expansion ratios of 1:1, 2:1, 3:1, 4:1, 6:1 and 9:1. Other skin expansion techniques such as the Meek technique are also invaluable for covering large areas of wound.¹¹⁷

Allograft skin harvested from cadaveric donors is an essential skin substitute used in the management of extensive burns. Appropriate donor selection, screening for communicable disease, consent from relatives and strict exclusion criteria are applied to ensure safety for transplantation. These functions are undertaken in addition to screening, harvesting, processing and distribution by regional or national tissue banks. The amounts required can be estimated by determining the body surface area and burn size in square centimetres.¹¹⁸

Fresh viable allograft skin can also be obtained from living donors, usually parents or relatives of burned children, however in view of the good quality and quantity of allograft skin provided by tissue banks, this method is used infrequently.

Allograft skin is mainly used meshed 2:1, allowing haematoma and seroma drainage. Sheet allograft tends to be used to cover cosmetic areas such as the face as a temporary covering.^{119,120}

Acellular bilaminar skin replacements have been extensively used that provide wound closure with theoretical permanent dermal replacement. An upper silicone layer acts as a temporary epidermis, forming a barrier to microorganisms and controlling evaporative water loss. The deeper layer is a cross-linked bovine collagen and chondroitin-6-sulfate matrix. This matrix is vascularized and incorporated into the wound. It is termed a 'neodermis' and is used to replace dermis in full-thickness burns and modulate postburn hypertrophic scarring. These dermal replacements have produced encouraging results both in single-centre studies and in a multicentred randomized controlled trial. These studies have reported less hypertrophic scarring

with a pliable scar and a reduced requirement for secondary reconstructive procedures. Long-term studies describing benefit are lacking.¹²¹

More recently synchronous application of a dermal substitute and skin graft has been established as a standard procedure. This approach obviates the need for a second procedure for application of autograft. In a pilot study comparing simultaneous transplantation of a dermal substitute and split-thickness autograft or split-thickness autograft alone it was reported that simultaneous application of a dermal matrix was safe, feasible and gave significantly better results with respect to skin elasticity.¹²²

De-epidermalized decellularized sterile human dermis can be used as a dermal replacement for acute care and for postburn reconstruction. In acute cases, the burn wound is excised and prepared as usual. The de-epidermalized decellularized dermis is applied to the wound and a thin (epidermal) split-skin graft is applied over it. This bilayered graft is secured and managed as skin graft. This technique has limited applications in the management of large surface area acute burns due to paucity of skin graft donor sites. It is more been used more successfully in postburn reconstruction.¹²³

Amnion is commonly used by ophthalmologists to cover damaged corneas as it is immunologically privileged tissue, has antiangiogenic properties and contains many growth factors that stimulate epithelial proliferation. Amnion has been used in burn care mainly in partial-thickness wounds. The risk of disease transmission, limited availability, small size and fragility limit its usefulness in burn care.¹²⁴

A variety of xenograft materials (sheep skin, frog skin) have been used as a skin substitute, but the only commonly commercially available product is from domestic swine. It has been processed in a number of ways, including irradiation, lyophilization, glutaraldehyde cross-linking and impregnation with silver to reduce antigenicity and prolong adherence. Porcine xenograft is usually available as de-epidermalized dermis, which is essentially a collagen matrix. Porcine skin xenografts can reduce evaporative water loss, limit infection, and encourage autologous epidermal growth. They are useful as temporary coverings for partial-thickness burns, donor sites and desquamating skin conditions. They are easily available, have a prolonged shelf life and are relatively inexpensive.¹²⁵

Cultured epithelial autografts (CEAs) have been used by burn surgeons for over 20 years. From a 1 cm² autologous skin biopsy, tissue culture techniques in the laboratory can produce enough epidermal cells (keratinocytes) in approximately 3–4 weeks to cover an adult of up to 2 m² body surface area. There have been many reports of successful use of CEA to close burn wounds, however the technique only produces the epidermal layer for wound closure. The cost of production is significant, there is a lag time of three weeks from biopsy to production of adequate quantities, the take rates can be low, and the grafts are very fragile and shear easily and blister even after successful take, due to poor and delayed basement membrane formation. Grafting

the CEA on to an allograft dermal bed as described by Cuono in 1986 improves take and durability.¹²⁶

More recently the use of subconfluent, autologous keratinocyte suspensions sprayed onto excised wounds has been described to treat partial-thickness wounds and excised wounds. In addition, augmentation of epithelialization by spraying interstices of widely meshed skin grafts and also skin graft donor sites has been used. The use of CEA is reserved for patients with major or massive burns with limited donor sites or ones that are difficult to harvest and cosmetically and functionally important.¹²⁷

Donor sites

The splint skin graft donor site is an important and often neglected wound that is vital to the successful care of burn patients. Management of a donor site wound should promote re-epithelialization with a minimum pain and discomfort. In major injury where donor sites need to be reharvested, they become a precious commodity that requires meticulous care to optimize healing and minimize infection so that the donor site can be reharvested in the shortest time possible.

Initial management at the time of harvest will include the use of topical adrenaline to limit bleeding and infiltration of the wound with a long-acting local anaesthetic such as bupivacaine.

Many dressings are available for donor site care and the choice usually depends upon surgeon preference and availability. In general, donor site dressings will involve a primary contact layer such as Telfa™ Clear (polyethylene terephthalate), Mepitel (silicone) or Jelonet® (paraffin gauze) covered with an absorbable layer of gauze and wool secured with a crêpe bandage.

In a recent randomized controlled trial, moist wound healing provided by hydrocolloid dressings was associated with the shortest healing rates when compared to other dressings, including alginate, semipermeable film, gauze, hydrofibre and silicone. In the same trial, patients treated with a gauze dressing had a higher rate of infection.¹²⁸

Rehabilitation

Burn rehabilitation starts on admission to the burn service. Passive measures such as patient positioning, splintage and control of oedema maintain range of motion and function. Active mobilization and promoting functional activities should also be encouraged from the earliest time possible.¹²⁹ Respiratory care and rehabilitation to maintain spontaneous ventilation, cough and expectoration minimize the need for mechanical ventilation and its associated complications.

Consistent repetitive motivational education is a vital part of patient care, with patients being encouraged to share responsibility to optimize their rehabilitative opportunities. Well-motivated

patients can surpass expectations with regard to functional recovery, whereas poorly motivated patients can end up with poor functional outcomes despite best efforts. Early exercise has been shown to be beneficial following paediatric burn injury, enhancing lean body mass and strength, without exacerbating postburn hypermetabolism; however the evidence for this is still lacking in an adult population.¹³⁰

Scar management

Hypertrophic scarring is a common problem following burn injury, with a high incidence in those wounds that take longer than three weeks to heal. They are typically thick, red and raised and can be symptomatic in terms of pain and itching. The hypertrophic scar is thought to result from an overproduction of collagen and an imbalance between the anabolic and catabolic phases of healing. Hypertrophic scarring causes significant aesthetic, functional and psychosocial morbidity and takes 18–24 months to mature.¹³¹

These scars require lifelong care, including moisturizing with creams and sun protection. Sun exposure causes hyperpigmentation, but there is no evidence of an increased risk of developing skin cancer.¹³² Scar maturation can be modulated by scar massage, pressure and the use of topical silicone.¹³¹ Custom-made pressure garments are used but are expensive. Recommendations include the use of continuous pressure of 15–40 mmHg, applied for at least 23 h/day for more than six months.¹³³ However, compression therapy is limited by the ability to adequately fit the garment to the wounded area, and patient discomfort frequently reduces compliance. There is little evidence of efficacy compared to simple elasticated bandages.¹³⁴

Silicone gel sheeting is thought to decrease collagen remodeling and hasten scar maturation, although the exact mechanism of action is unclear and the evidence is not strong.¹³⁵ Side effects of silicone sheeting include local skin reactions, such as maceration and pruritus.

Scar management is usually undertaken and supervised by physical therapists on a regular three-monthly basis in an outpatient clinic in association with the surgical team.

Psychological support

Psychological support of the burn patient is vital and must be a routine part of treatment from the outset of care. Depression and posttraumatic stress disorder (PTSD) are the most common psychological problems and are often linked to preinjury morbidity. Other risk factors include pain, anxiety, female gender and facial scarring. Therapeutic interventions include optimizing social functioning and adjustment as well as therapies directed toward anxiety, depression and PTSD.¹³⁶

Return to work can be considered as one of the ultimate goals of postburn rehabilitation, demonstrating reintegration into society. In a retrospective review 90% of patients returned to

work at 24 months post injury. Only 37% returned to the same job. Predictors of return to work outcome included TBSA, anatomical site of injury, length of hospital stay, preinjury personality and psychiatric morbidity and demographic factors including preinjury employment status.¹³⁷

Burn reconstruction

The aim of postburn reconstruction is to preserve, restore and improve function, comfort and appearance. This requires an organized approach with functional issues taking precedence over aesthetic concerns. Normal and hypertrophic scarring, scar contracture, loss of parts of the body, and change in colour and texture of injured skin are processes common to all burned patients.

Prevention of dysfunction and deformity starts on admission and carries on throughout the acute injury phase. Once wound healing has been achieved, patients go into scar management programmes consisting of the use of pressure garments, massage and moisturizing therapy to optimize scar maturation.

Timing

Surgical reconstruction is usually delayed until scar maturation has occurred and the effects of scar management are well established after about 12 months. As unsightly scars mature over time they become flexible and pliable, and patience on the behalf of both surgeon and patient is recommended.

Timing of reconstruction is guided by the clinical scenario and can be categorized as urgent (immediate), essential (early) or desirable (late). Urgent (immediate) procedures are indicated for problems such as cover for exposed vital structures or to preserve function in vital areas and should be performed after all open wounds are closed. Examples include release of eyelids to protect the cornea, microstomia, entrapment of neurovascular bundles and severe neck contractures.

Essential (early) procedures are performed to improve rehabilitation and function, and usually for mature burn scar contractures that do not respond to splinting or aggressive physical therapy. Examples include neck contractures, joint contractures (e.g. elbow, knee and ankle), mobility-limiting contractures (e.g. axilla, groin) and hand contractures.

Desirable (late) procedures encompass the majority of reconstructions and are performed after the scar has matured and address the size and shape of the mature scar, including abnormalities in colour and texture with the aim of producing the best aesthetic result possible. Examples of late reconstruction include reconstruction of passive areas (e.g. trunk, extremities) and aesthetics (e.g. face, breast).

Following any reconstructive procedure, occupational and physical therapy, scar management and skin care are all essential to postoperative management to ensure optimal outcome.

A realistic approach is necessary to counsel patients' expectations to the probable outcomes of reconstructive surgery and a good patient–surgeon relationship is essential.¹³⁸

Planning reconstruction

Surgical planning should be undertaken following a thorough review of the problems and potential solutions and should include a record of the acute care, a full history and physical examination, a record of problems noting quality and colour of the skin, scars, pigmentation, contractures, atrophy and open wounds. A functional assessment should be made and recorded with the aid of the physiotherapists and occupational therapist, including the contribution of both soft tissue and skeletal deformity. A full donor site inventory should be made so that they can be prioritized according to the patient's reconstructive needs.¹³⁸ Different anatomical and functional sites require different reconstructive options, including skin grafts, skin substitutes, tissue expansion, local, regional and distal flaps.

Outcome

Measurement of outcome in burns patients is complex and multifaceted. As increased numbers of burned injured patients survive, a more comprehensive assessment of outcome deserves high priority.

Mortality and length of stay have traditionally been used as outcome measures, although crude mortality data are not helpful due to lack of risk stratification. The percent TBSA burn at which half of patients admitted to the burns unit die calculated by probit analysis of mortality data is known as the lethal dose 50% (LD%) and is the standard mortality outcome that can be used to compare services.¹³⁹ Length of stay is a good global surrogate measure for outcome and quality of care reflecting the whole patient journey, including assessment, admission, fluid resuscitation, surgical care, nutrition, critical care, physical and psychosocial rehabilitation. Delays in discharge reflect complex rehabilitation, psychological or social issues.

More complex outcome analysis has been proposed that encompasses a wider range of domains, including skin, neuromuscular function, sensory function, pain, psychological function, physical function, community participation and perceived quality of life. The optimal measurement tools of these outcome domains are yet to be fully developed.¹⁴⁰

The future

Prevention should be the ultimate global goal of burn care to reduce the burden of disease, disfigurement and disability. Future advances will concentrate on modelling care to suit a patient's individual genetic makeup and response to tissue injury and trauma.

The application of regenerative medicine techniques both in the acute setting and subsequent rehabilitation and reconstruction will be individually tailored to match bespoke patient needs.¹⁴¹ Tissue engineering holds great promise for all reconstructive surgeons. The combination of regenerative medicine and tissue engineering will allow prefabrication of flaps with specific characteristics, and the availability of 'off the shelf' skin and soft tissue constructs.

Tissue allotransplantation holds great promise in areas of complex 3D reconstruction, such as the face; however, the issues associated with immunosuppressive therapy and its complications must be addressed.¹⁴²

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PART III

Paediatric plastic surgery and congenital disorders

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Congenital melanocytic naevi

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Incidence of phenotypic classification

Congenital melanocytic naevi (CMN) are present at birth, or very occasionally, arise in the first 2 years of life (*tardive CMN*). These lesions grow commensurate with the child, but tardive lesions can appear to grow more rapidly as the full extent of the CMN is often not initially apparent. The incidence ranges from 1 to 2% if all sizes are considered to approximately 1 in 20 000 for naevi projected to be greater than 20 cm in diameter in adulthood.¹ CMN are commonly classified according to maximum projected adult size and the total number of lesions at birth. The most recently proposed classification of size is small (CMN with an estimated diameter in adulthood of less than 1.5 cm), medium (M1, 1.5–10 cm and M2, 10–20 cm), large (L1, 20–30 cm, L2, 30–40 cm) and giant (G1, 40–60 cm, G2, >60 cm).² However, the authors prefer not to employ the term *giant* as it is often unacceptable to patients and families.

The estimates of projected adult size are determined in a variety of ways, none of which are decidedly accurate, but remain the most reliable methods currently available. Some clinicians use a scaling factor applied to the CMN diameter at birth. More specifically, a CMN on the head is predicted to enlarge by a factor of 1.7, on the trunk and upper extremity by a factor of 2.8 and on the lower extremity by a factor of 3.3.³ This approach is not without fault, as CMN often affect more than one region of the body and it does not account for significant differences in growth between the sexes. Other clinicians therefore estimate the projected adult size by drawing the child's CMN onto an adult body map, and estimating the longest non-circumferential diameter.

CMN can present as isolated singular lesions or can be accompanied by a variable number of smaller discrete so-called *satellite* lesions. These lesions should be counted or estimated as each satellite lesion represents an additional CMN.

Histological and clinical appearances of CMN

CMN consist of nests of naevus cells, which differ from normal melanocytes in their non-dendritic morphology and proclivity to form nests. Melanocytic nests are present at the dermo-epidermal

junction, within the dermis and/or subcutaneous tissue, and naevus cells can be localized between collagen bundles and found surrounding dermal appendages, blood vessels and nerves. Small and medium-sized CMN generally present clinically as homogeneous pigmented lesions with well-delineated borders, an increase in skin surface markings and variable hypertrichosis. Larger CMN express colour variegation, are often asymmetric with irregular borders, can have a nodular surface and are usually hypertrichotic (Figure 15.1).

Nodules are often appreciated to arise within the substance of the CMN or can be present at birth. Any such focal change within a CMN should be viewed with suspicion and reviewed by a paediatric dermatologist. Although malignancy should always be considered, the vast majority of nodules are benign and repeated excisions can thus be avoided. Histopathology and genetic investigations can assist in differentiating between benign proliferative nodules and melanoma, in particular array comparative genomic hybridization.⁴

Neurological associations of CMN

Individuals with CMN can be divided into those with a cutaneous phenotype only (either single or multiple), and those with extracutaneous features (either neurological or facial), the latter then termed *congenital melanocytic naevus (CMN) syndrome*. The projected adult size of the lesion, as well as the total number of CMN at birth is known to be associated with adverse clinical outcomes. Neurological abnormalities are the most common complication of CMN, and this association has previously been known as *neurocutaneous melanosis*. The latter term has now been supplanted by *CMN syndrome*, as the neurological complications are not always melanotic in nature. The prevalence of neurological abnormalities increases with the size of the largest CMN and the total number of naevi.^{5,6} CMN distribution, in particular, lesions on the posterior axis, is not associated with an increased risk of neurological anomalies. The previously suggested connection between cutaneous distribution and neurological risk



Figure 15.1 Clinical images of different cutaneous phenotypes of CMN.

was confounded by the fact that the largest lesions are often located on the back.

The most common neurological complication is intraparenchymal melanosis, where foci of melanin-producing cells are found within the brain parenchyma, most notably in the amygdala.⁷ Intra-parenchymal melanosis without other neurological abnormalities on MRI are benign congenital lesions and entirely asymptomatic in approximately half of cases. The other half of cases can exhibit developmental delay. Although delay is most frequently mild, global developmental retardation or behavioural problems, such as attention deficit hyperactivity disorder or autistic spectrum disorder, can be present.⁶ Even when symptomatic, these lesions are not a sign of malignancy or of impending death, and should not be considered for surgical resection. Although rare, progression to malignant melanoma remains a possibility. However, the latter can arise in the CNS even in the absence of visible intraparenchymal melanosis.

Other congenital neurological anomalies such as Dandy–Walker malformation, hydrocephalus and brain or spinal cord tumours can occur, with or without leptomeningeal melanosis. These more complex neurological abnormalities are much more commonly symptomatic with manifestation of seizures and developmental delay, and therefore necessitate more rigorous management, with regular MRI, neurological assessments and frequently, neurosurgical consultation.⁸ Interestingly, it has been shown that neurological symptoms in patients with CMN can be present in the absence of any perceptible radiological abnormality.^{6,9} This finding has been proposed to be related to lesions that cannot be visualized with current imaging techniques.¹⁰ Thus, the authors' current management protocol includes a gadolinium-enhanced MRI of the CNS for any child

with the onset of abnormal neurological signs or symptoms at any age (irrespective of a previously normal scan) and routinely for *all children with two or more CMN at birth, independent of size or site* (Figure 15.2).

Follow-up of children with CMN or CMN syndrome depends on the severity of the cutaneous phenotype, the neurological presentation and the risk of melanoma. Patients should have access to a specialist paediatric dermatology clinic with input from a multidisciplinary team including paediatric plastic surgeon, neuroradiologist, neurologist, neurosurgeon, psychologist and oncologist when necessary.

Malignant melanoma and CMN

Primary malignant melanoma of the skin or CNS is a rare complication of CMN. However, the incidence varies with the severity of the cutaneous phenotype. The cited estimates of the incidence of malignancy have been extremely variable, ranging from 0 to 40%. More recent and reliable data on the incidence of malignant melanoma and the clinical phenotype of CMN associated with its development have been published. Large studies have demonstrated that although there is an increased risk of melanoma in patients with CMN (especially during childhood and adolescence), it is in fact much lower than previously reported estimates, with the overall incidence for all sizes of CMN between 0.7 and 2.4%.^{11–13} However, the risk of melanoma increases with CMN of projected adult size >40 cm and satellite lesions at birth. Although the incidence of malignant transformation in this group has to be definitively elucidated, a recent prospective study

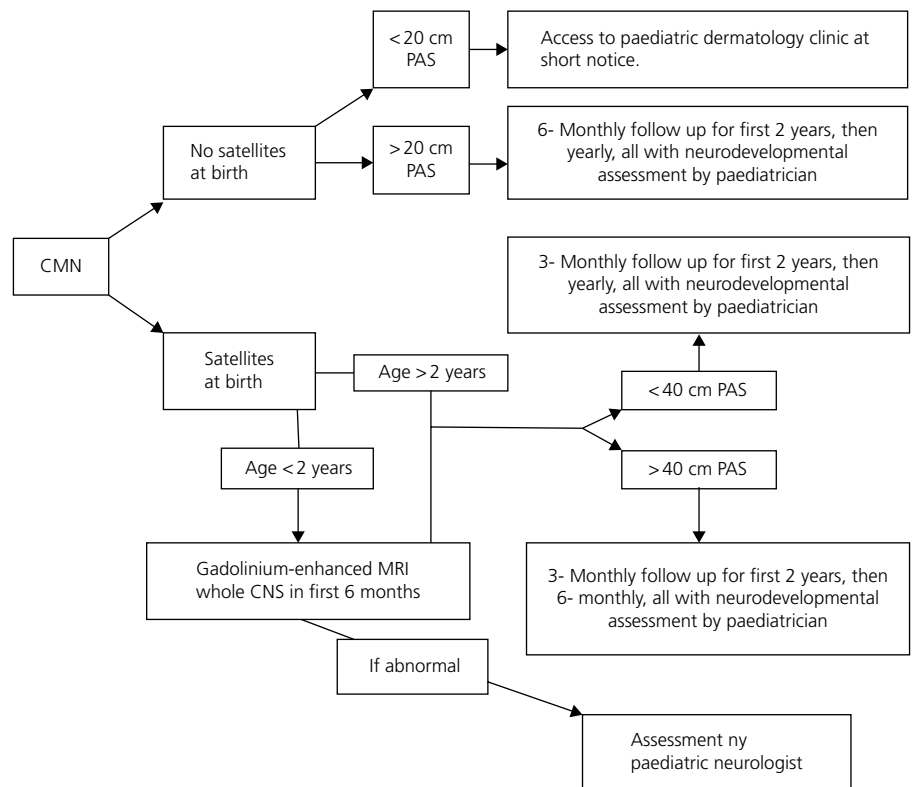


Figure 15.2 Protocol for management of CMN in childhood. PAS, projected adult size.

suggested an estimated incidence of 14% in those with CMN of projected adult size of >60 cm or multiple naevi.¹¹ Authors therefore proposed that patients with CMN of <20 cm projected adult size without satellite lesions at birth should be reassured that the risk of melanoma, at least in childhood, is approximately 1%. When melanoma does arise in these children, it is usually highly aggressive, refractory to therapy and rapidly fatal.^{11,12} The median age of onset is estimated at 7 years.¹²

Genetics of CMN

The causative genetics of multiple cutaneous CMN and CMN syndrome have recently been elucidated. These are engendered by somatic mosaicism for mutations in the gene *NRAS*,¹⁴ and are therefore one of the mosaic RASopathies (genetic disorders of the RAS/MAPK pathway).¹⁵ *NRAS* is a known oncogene involved at the somatic level in the development of melanoma, and although mutations in a variety of oncogenes have been described in individual CMN,^{16–18} no causal association between these findings has previously been made or suggested for the multisystem disorder. Developmental *NRAS* mutations predispose to melanoma in individuals with CMN syndrome by acting as the first insult in the subsequent occurrence of melanoma.¹⁴

Evidence is suggestive, however, of a germline predisposition for the development of CMN, with a higher prevalence

of smaller CMN in family members of affected individuals,^{11,14} as well as rare reports of large CMN in first-degree family members.^{19,20} In one UK study, homozygosity and compound heterozygosity of variants in the *melanocortin-1-receptor* (*MC1R*) gene were found at a higher prevalence in individuals with CMN and certain variants were associated with a more severe cutaneous phenotype.²¹ The *MC1R* is a G-protein coupled receptor expressed on melanocytes, melanoma cells and CMN cells. The *MC1R* gene is responsible for red hair and freckling in humans. Kinsler *et al.*²¹ proposed that the *MC1R* gene is implicated in the pathogenesis of CMN syndrome, based on the observation of a high prevalence of red hair pigmentary phenotype in families of children with CMN. More specifically, certain *MC1R* variants appear to promote growth of CMN during embryogenesis. *MC1R* variants are a risk factor for melanoma development in the general population and may contribute to the increased risk of melanoma in individuals with CMN. The lifetime risk for melanoma is approximately 1–2% when all CMN are considered together as a group, which is comparable in magnitude to the excess risk associated with *MC1R* variants in the normal population.

Recently, characteristic facies in children with CMN has expanded the description of this condition (Figure 15.3). Typical features include a wide or prominent forehead, apparent hypertelorism, eyebrow variants, periorbital fullness, small/short nose, narrow nasal bridge, anteverted nares, broad nasal tip,



Figure 15.3 Facial appearances of children with CMN.

Table 15.1 Prevalence of characteristic facial features of children with CMN

Facial feature	Prevalence (%)
Wide or prominent forehead	74
Apparent hypertelorism	12
Eyebrow variants	36
Periorbital fullness	27
Small/short nose	25
Narrow nasal ridge	18
Flaring nares	15
Broad nasal tip	34
Broad or round face	71
Full cheeks	55
Prominent premaxilla	10
Prominent everted lower lip	47
Prominent or long philtrum	20

The presence of three or more features is required to fulfil the facial criteria for congenital melanocytic naevus syndrome.

broad or round face, full cheeks, prominent premaxilla, open mouth appearance, everted lower lip, and prominent or long philtrum.²² (Table 15.1). In support of the notion that children with CMN display a distinctive facies is the fact that aberration in neural crest development gives rise to melanocytic and facial development in humans. Indeed, a single genetic abnormality may be responsible for CMN, neurological abnormalities and the characteristic facies of these children, by affecting the pluripotent cephalic neural crest cells that ultimately differentiate into diverse cell types including melanocytes, neurons, glia, osteocytes and chondrocytes. The genes on the RAS/MAPK pathway are known to be involved in facial development, and individuals with germline RASopathies have characteristic facies. This clinical finding is therefore consistent with *NRAS* mosaicism affecting neural crest or neuro-ectodermal structures, including pigment cells, cells of the CNS as well as bone and cartilage of the face.

Thus, the term *congenital melanocytic naevus syndrome* has been proposed with preliminary diagnostic criteria in an effort to formally characterize and unite these aspects of the condition. The following two criteria are included, both of which must be present. First, the presence of a CMN of greater than 5 cm projected adult size or more than one CMN of any size at birth. Second, neurological involvement (clinical or radiological) and/or the presence of three or more typical facial features.

Surgical management of CMN

Irrespective of size, long-standing practice for treatment of CMN has been surgical removal. A commonly cited argument in favour of early surgical excision is to decrease or avert the potential for malignant degeneration to melanoma. However, *surgical excision has not been shown to reduce the risk of malignant melanoma formation in patients with CMN*. First, individuals at high risk for malignancy have very extensive CMN associated with numerous smaller CMN, which can total in excess of 400 CMN. *Complete* excision of a very large CMN and *all smaller naevi* remains an impossibility (Figure 15.4). Second, there is some evidence that the behaviour of naevus cells may in fact be altered by surgical intervention. In a recent study,²³ tissue expansion was associated with the development of new naevi, supported by individual case evidence from other centres (Taieb A, personal communication). The latter is thought to occur secondary to either non-naevus cell melanocytes that are activated by some aspect of treatment with tissue expanders or that treatment promotes the spread of naevus cells from the primary naevus, a type of benign metastasis known to occur in CMN and Spitz naevi. Moreover, an increased incidence of darkening of surgically treated areas of CMN and untreated areas of partially excised CMN were noted, and new CMN were found to appear at the edge of the treated area.²⁴ An additional important consideration is that the occurrence of *malignant*



Figure 15.4 Child with a giant CMN and multiple smaller CMN, demonstrating the surgical impossibility of eradicating all lesions.

melanoma is not restricted to the CMN, and thus surgical excision of the primary CMN does not preclude development of melanoma in the CNS or other extracutaneous locations.

CMN can be aesthetically displeasing and as such associated with considerable psychological morbidity. Thus, a secondary argument for surgical excision of CMN is aesthetic concerns. However, there is evidence that untreated CMN often lighten over a period of years (Figure 15.5). Thus, with the exception of tardive CMN, the colour of CMN is darkest at birth. Although this phenomenon is quite variable, and the degree of depigmentation cannot be reliably predicted, the authors recommend observation with serial photographs for at least a year to assess for spontaneous lightening. Of note, scalp CMN appear to reliably display the most considerable amount of change. The latter is further supported by a recent series of spontaneous involution of scalp CMN in childhood.²⁵ Thus, removal of scalp lesions by tissue expansion is no longer advised, given the significant surgical intervention required and notable associated complication rate (e.g. alopecia). Often, however, these lesions extend into neighbouring areas of face, which can be addressed surgically (Figure 15.6). In fact, facial lesions, whether the primary CMN or a satellite lesion, are in the authors' practice routinely excised, as are CMN that can be completely removed by staged serial excision (Figure 15.7). Operative management is also routinely offered to patients with functionally troublesome or particularly prominent areas (e.g. large nodule) or areas suspect for malignancy. Clinical surveillance of CMN remains the principal treatment option for large or giant CMN or those in areas where excision would result in significant anatomic or functional compromise.

Earlier treatment modalities utilized in the surgical management of these lesions included curettage and dermabra-

sion. More recently, laser therapy has been introduced as an additional therapeutic option. One premise for these options has been the suggestion that superficial naevus cells bear the most malignant potential and therefore removal of these cells will effectively reduce the risk.²⁶ However, it is well documented that malignant transformation within CMN most often occurs within the deeper cells of the dermis and subcutaneous tissue.²⁷ Indeed, two thirds of cutaneous melanoma within CMN develops below the dermo-epidermal junction. Thus, the effects of superficial excision can mask the appearance of a melanoma arising within the CMN. Moreover, these techniques are associated with significant morbidity including bleeding, infection (sepsis), scarring and recurrent folliculitis. Most importantly, all lesions treated with superficial removal re-pigment over time, at variable rates ranging from weeks to years. Large CMN extend through the entire dermis, often into subcutaneous tissue. Initially, after elimination of superficial naevus, there is a noticeable decrease in pigmentation, as the deeper naevus cells do not produce melanin. However, once these deeper cells become more superficial they begin to produce melanin, resulting in re-pigmentation (Figure 15.8). Comparison of the colour of re-pigmented CMN following superficial removal is intrinsically biased, as CMN spontaneously lighten over time. The latter is evident when treated and untreated areas in the same individual are compared, where no difference in pigmentation is observed.

Currently, the primary methods in the operative management of CMN include single-stage excision with primary closure, staged serial excision, tissue expansion and excision with either partial- or full-thickness skin grafting. Any of the aforementioned techniques can be used alone or in combination, following consideration of the aesthetic unit(s) to be addressed. Although implemented by some clinicians, free or pedicled flap



Figure 15.5 Spontaneous lightening and regression of an untreated CMN. In this case, lightening became evident after 6 months of age. Illustrated are images of the CMN at age (a) 2 months, (b) 1 year, (c) 2 years and (d) 3 years. Of note, the last image demonstrates CMN regression in addition to depigmentation.



Figure 15.6 (a) Child with a large CMN involving the scalp, forehead, temporal region and lateral aspect of the cheek and ear. In this case, only the part of the CMN on the face was surgically excised. The resultant defect was resurfaced with a split-thickness skin graft harvested from the scalp. (b) Result at 6 months following the procedure.

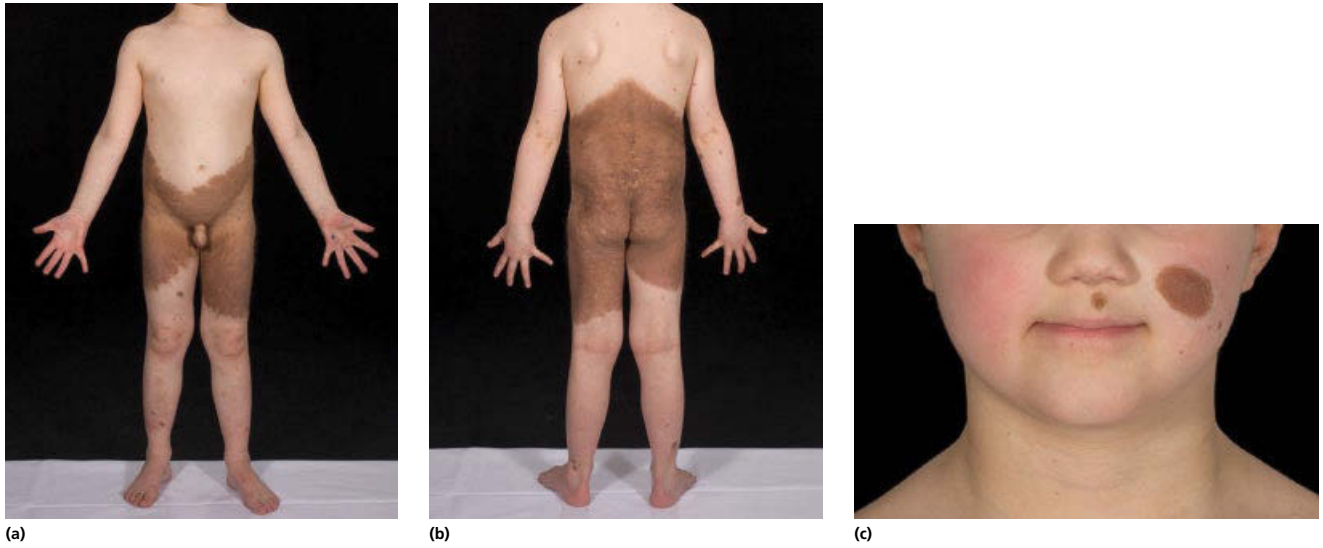


Figure 15.7 Child with a giant CMN and multiple smaller CMN. In contrast to the child in Figure 15.4, this patient would be offered the possibility of excising the two smaller CMN on his face, which are harbingers of the larger so-called *bathing suit pattern* CMN.



Figure 15.8 CMN treated with superficial excision demonstrating re-pigmentation. (a, b) CMN at day 1 of life. (c, d) CMN 2 months following dermabrasion. (e, f) CMN 2 years following dermabrasion, which shows the return of pigment to all treated areas.

coverage (with or without tissue expansion) in an effort to eradicate large or giant CMN is not considered in the authors' surgical armamentarium, given the previously stated points and in view of the considerable morbidity associated with these interventions. In general, unless there is a specific concern with regards to malignancy or functional issue, surgery is delayed until after 2 years of age. Occasionally, however, partial or complete excision with primary closure is effected earlier taking advantage of the skin laxity in infancy. It is important that the full thickness of the lesion be excised, and thus carried down to the subcutaneous tissue layer.

In cases where excision and primary closure is not a practicable alternative, serial excision is considered if complete excision can be accomplished in two or three stages. In cases where closure with skin graft is planned, usually for CMN of the face or hand, either a split- or full-thickness skin graft is selected based on size and location of the excised lesion. For large areas where full-thickness skin graft coverage is planned, pre-expansion of the donor skin prior to harvest minimizes the aesthetic morbidity at the donor site. As previously mentioned, in the authors' practice, facial lesions are routinely excised, sparing areas of naevi involving delicate facial structures that remain difficult to reconstruct without significant risk of iatrogenic deformity (e.g. alar rim, hair-bearing eyebrow). For facial lesions, where a split-thickness skin graft is selected, the scalp remains the donor site of choice. Full-thickness skin grafts are usually harvested from the post-auricular area or supraclavicular fossa (with or without pre-expansion of the area) depending on the size of the resulting defect. Tissue expansion of the area adjoining the CMN to be excised is generally avoided in the forehead. The latter usually does not give a superior aesthetic result to skin grafting and is associated with considerable risk for distortion of forehead musculature and unaffected neighbouring cosmetic units (namely, the eyebrow and hairline).

Conclusion

CMN can be restricted to the skin or associated with neurological abnormalities and/or characteristic facial features (CMN syndrome). Management of children with multiple (>1) CMN or CMN syndrome necessitates a multidisciplinary approach involving the care of a paediatrician, dermatologist, plastic surgeon, neurologist, neuroradiologist, psychologist and primary care physician. Children with more than one CMN at birth (irrespective of size or site) should have an MRI of the CNS and undergo routine neurodevelopmental assessment. Routine surgery with current techniques has not been shown to decrease the risk of malignancy in CMN. Moreover, there is no proven benefit to early intervention and some evidence suggests that surgery may adversely affect the behaviour of CMN cells. Based on these findings, a shift in management of these patients is recommended. In our current practice, surgery is delayed for

at least the first year, with serial photographs taken at yearly intervals to assess spontaneous lightening. Surgical intervention is offered to patients with CMN that are easily amenable to excision or those in aesthetically sensitive areas.

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CHAPTER 16

Vascular anomalies

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Vascular anomalies are disorders of the endothelium, which can be subclassified into vascular tumours and vascular malformations, distinguished by proliferating endothelium and quiescent endothelium, respectively¹ (Table 16.1). Vascular anomalies can affect individuals of any age and patients present to clinicians in a variety of clinical specialties. These lesions often require significant diagnostic expertise and a multidisciplinary management approach. Arguably, in no area of paediatric medicine is incorrect terminology more pervasive than in the field of vascular anomalies. This is largely due to insufficient training in the subject, the wide variety of terms and labels used in the historic literature and the involvement of many different medical specialties, each with their particular interests and assumptions. Correct designation using the standardized classifications and terminology introduced by the International Society for the Study of Vascular Anomalies (ISSVA) is paramount for appropriate patient management.² In the majority of cases, the precise diagnosis can be realized by detailed history and physical examination. In instances where diagnostic ambiguity persists, imaging proves useful, with ultrasound sufficient in most instances. Rarely, is tissue biopsy required.

Vascular tumours

The term *vascular tumour* encompasses a wide range of pathology, including common haemangiomas and other rare lesions such as tufted angiomas and kaposiform haemangioendotheliomas. The majority of *vascular tumours* have a rapid proliferative growth pattern and are composed of solid tissue, forming focal soft tissue masses. In contrast, *vascular malformations* are made up of vascular channels or cysts and are usually more diffuse. The most common vascular tumour is the *haemangioma*. Haemangiomas can be divided into infantile and congenital tumours. Infantile haemangiomas occur in approximately 3–10% of infants, and in up to 30% of premature infants. These lesions are most common in white infants, with a lower frequency in infants of African or Asian descent, and are 3- to 5-fold more

common in females than males.³ In approximately 20% of infants, multiple haemangiomas may be present, with five or more tumours termed *haemangiomatosis*. When five or more infantile haemangiomas are noted, further investigation with abdominal ultrasound is mandatory to exclude the presence of haemangioma in the liver. A rare presentation is *miliary haemangiomatosis*, in which a large number of small cutaneous haemangiomas, usually exceeding 20, are apparent soon after birth and recognized to be associated with internal lesions.

Infantile haemangiomas appear to arise from vasculogenesis, and more specifically, from multipotent derived stem cells, which have the potential to differentiate into multiple cell lineages and form human *glucose transporter isoform 1 (GLUT1)*-positive microvessels in immunodeficient mice.⁴ During the proliferative phase, these cells produce vascular endothelial growth factor, an endothelial mitogen. Indeed, *GLUT1* is uniquely expressed on the endothelial cells of haemangiomas and provides a valuable marker for differentiation of haemangiomas from other vascular tumours and vascular malformations. Haemangiomas have a characteristic evolution, appearing within the first few weeks of life, proliferating for several weeks to months, stabilizing between 6 and 12 months of age, and finally gradually regressing over a period of several years.

At birth, the site of the future haemangioma is usually completely normal, with the median age of appearance at 2 weeks. However in approximately 30% of cases, a nascent lesion may be present, such as an area of telangiectasia or pallor. Rapid growth then ensues, with tumours attaining approximately 80% of their maximal size by 3 months. After 12 months of age, the tumour usually begins the involuting phase. Classically, this stage is characterized by complete resolution of approximately 50% of lesions by 5 years of age, 70% by 7 years of age and 90% by 9 years of age (estimates derived from the study of Bowers *et al.*⁵). However, more recent clinical and histological work has demonstrated that involution is usually complete by age 4 years.⁶ Following regression, infantile haemangiomas can often leave residual telangiectasia, anetodermic cutaneous excess, fibro-fatty residuum, scarring and, rarely, significant structural deformity (Figure 16.1).

Table 16.1 Current classification of vascular anomalies

Tumours	Malformations	
	Slow flow	Fast flow
Infantile haemangioma (IH)	Capillary malformation (CM) Cutis marmorata telangiectatica congenita (CMTTC) Telangiectasias	Arterial malformation (AM) Aneurysm Atresia Ectasia Stenosis
Congenital haemangioma (CH)	Lymphatic malformation (LM)	Arteriovenous malformation (AVM)
Rapidly involuting congenital haemangioma (RICH)	Microcystic	Capillary malformation–arteriovenous malformation (CM-AVM)
Non-involuting congenital haemangioma (NICH)	Macrocystic Primary lymphoedema	Hereditary haemorrhagic telangiectasia (HHT) PTEN-associated vascular anomaly (PTEN-AVA)
Haemangioendotheliomas	Venous malformation (VM)	Combined malformations
Kaposiform haemangioendothelioma (KHE)	Cerebral cavernous malformation (CCM)	Capillary–arteriovenous malformation (CAVM)
Other	Cutaneomucosal venous malformation (CMVM) Glomuvenous malformation (GVM) Verrucous haemangioma (VH)	Capillary–lymphatic arteriovenous malformation (CLAVM)
Pyogenic granuloma (PG)	Combined malformations Capillary–venous malformation (CVM) Capillary–lymphatic malformation (CLM) Capillary–lymphatic–venous malformation (CLVM) Lymphatic–venous malformation (LVM)	

Source: Greene, 2012.²

The majority of haemangiomas are small and do not encroach onto anatomically sensitive areas, and as such, are clinically insignificant. These lesions are readily diagnosed in view of their characteristic appearance, including distinctive cherry-red colour. Occasionally, deep-seated tumours, which do not alter the colour of overlying skin are encountered, often requiring ultrasound to confirm the diagnosis. With these haemangiomas, families should simply be reassured that the tumour(s) will resolve with time and the lesion(s) followed only if there is cause for concern. Although infantile haemangiomas predictably undergo spontaneous involution, during their proliferative phase, they occasionally cause disfigurement and serious complications, including airway obstruction (subglottic haemangioma) or amblyopia (periorbital or orbital haemangioma), cardiac failure and/or hypothyroidism (hepatic haemangioma), ulceration and bleeding.⁷ In the past, such lesions were treated with corticosteroids or laser therapy.⁸ In 2008, Léauté-Labrèze *et al.*⁹ published a landmark paper where the unanticipated antiproliferative effect of propranolol on infantile haemangiomas was described. Propranolol has since proven to be highly effective and generally well tolerated,¹⁰ and is now the systemic treatment of choice.¹¹ The therapeutic action of propranolol is secondary to vasoconstriction, inhibition of angiogenesis and induction of apoptosis. Prior to initiation of propranolol, infants should be hospitalized for monitoring of any adverse effects, which have been reported to include bronchospasm, bradycardia, hypotension, hypoglycaemia, seizures, hyperkalaemia and diarrhoea. Evaluation with a baseline electrocardiogram and/or echocardiogram, vital

signs, blood glucose and electrolyte measurements are recommended. A history of asthma, blood glucose abnormalities and congenital heart disease are considered contraindications for initiation of propranolol therapy. For small superficial haemangiomas, early use of the topical beta-blocking agent timolol can be effective. The use of systemic corticosteroids is now reserved only as a second-line treatment, when propranolol is contraindicated.¹² There is no role for vincristine or interferon previously documented in the literature.

The use of pulsed dye laser should be reserved for treatment of residual telangiectasias following tumour involution. Surgical excision is commonly performed for tumours that are in the involuting phase. This is considered when it is evident that surgical intervention will be necessary and the resultant aesthetic outcome is predicted to be comparable to that anticipated during the involuted phase. Otherwise, resection is delayed until involution is complete. Serial excision or circular excision and purse-string closure can be utilized for surgical resection.¹³ Occasionally, excision can be considered in the proliferative phase, if for instance, outcome would be comparable to surgery effected later or if medical management is contraindicated or has failed.

Infantile haemangiomas can also occur in extracutaneous areas, most frequently in the liver. Internal organ involvement is more common in infants with multiple haemangiomas (>5) and haemangiomatosis. These hepatic tumours can instigate hepatomegaly, cardiac failure and/or hypothyroidism. The presence of a flat segmental facial haemangioma should prompt consideration



Figure 16.1 (a) Haemangioma of the left cheek, lower eyelid, lateral nose and upper lip in an infant. (b, c, d, e) Series of images at ages 3, 4, 5 and 10 years, which illustrate the natural regression of the haemangioma and resolution of the associated anatomic distortion.

of the associated diagnosis of 'PHACES' (the term PHACES comprises posterior fossa brain malformation, *haemangioma*, arterial cerebrovascular anomalies (most common associated finding), coarctation of the aorta and cardiac defects, *eye/endocrine* abnormalities and sternal clefting or supra-umbilical raphe).¹⁴ Evaluation of the brain and cerebral vasculature with MRI is important, although deciding which infants to image remains unclear. Similarly, segmental haemangiomas in the lumbrosacral region and perineum can be associated with ventral-caudal malformations, including omphalocele, imperforate anus, vaginal

and renal anomalies. Radiological investigation (ultrasound and/or MRI) is essential to exclude any associated abnormalities.

In contrast to infantile haemangiomas, congenital haemangiomas are almost always completely developed at birth and divided into *rapidly involuting congenital haemangiomas* (RICH) and *non-involuting congenital haemangiomas* (NICH), depending on their pattern of involution. Mulliken and Enjolras¹⁵ have characterized the disparate growth curves for congenital compared with infantile haemangiomas (Figure 16.2). Aside from the distinctive clinical presentation of these congenital lesions, as well as differing clinical

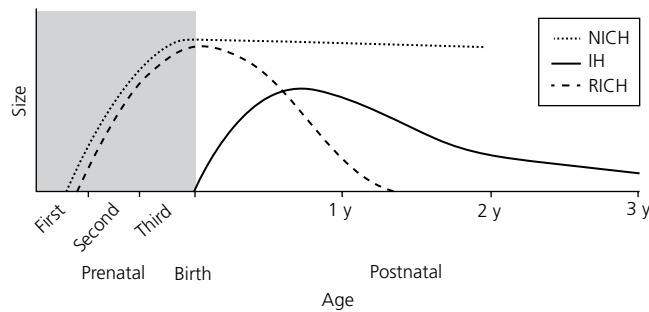


Figure 16.2 Growth curves for rapidly involuting congenital haemangiomas (RICH), non-involuting congenital haemangiomas (NICH) and infantile haemangioma (IH). Source: Mulliken and Enjolras.¹⁵ Reproduced with permission of Elsevier.

course, *negative GLUT1 immunohistochemical staining* is an important characteristic to distinguish congenital tumours from their infantile counterparts. There are no distinctive imaging features that reliably differentiate between a RICH and a NICH, with both types appearing as well-defined, hypervascular, relatively homogeneous masses on ultrasound.

For some RICH, involution may begin within the first week of life. In most infants with RICH, regression is complete within the first 7–14 months of life, with anetodermic cutaneous excess remaining. Interestingly, in a small proportion of patients with RICH, involution may be incomplete, resulting in a residuum clinically indistinguishable from NICH. A typical NICH forms a flat violaceous vascular plaque with coarse surface telangiectasia bordered by a blue-white rim. Unlike their infantile counterparts, congenital haemangiomas rarely require intervention in infancy. Given the clinical course of RICH, observation is the mainstay of treatment. Occasionally, very large RICH may incite congestive heart failure due to their hypervascularity, in which case embolization is considered to dampen the lesion's arterial supply until regression has commenced. The latter is particularly common with hepatic RICH.

Aside from their clinical behaviour, hepatic RICH are typically unifocal, and are therefore readily distinguished from hepatic infantile haemangiomas, which are multifocal and usually clinically silent. Large RICH may also result in localized deformity requiring surgical intervention in later childhood. More specifically, these lesions may cause *fat atrophy* resulting in contour deformity (in contradistinction to infantile haemangiomas, which often leave fibro-fatty residuum) or excess anetodermic skin requiring excision. Moreover, regressed lesions can leave an unsightly bed of large, dysplastic veins, which respond well to sclerotherapy.

Some lesions may be difficult to differentiate from other tumours, such as congenital fibrosarcoma or rhabdomyosarcoma, and as such may warrant early biopsy and/or surgical excision. NICH may necessitate excision for aesthetic concerns as these lesions never involute.

Kaposiform haemangioendotheliomas are locally aggressive solitary vascular tumours, which are usually present at or soon



Figure 16.3 Clinical image of an infant with a large violaceous oedematous lesion typical of a kaposiform haemangioendothelioma.

after birth. This tumour often presents as a large violaceous oedematous lesion (Figure 16.3). Diagnosis can be confirmed with MRI, which shows a characteristic stranded, T2 hyperintensity, poorly defined margins and invasion of adjacent tissues. By 2 years of age, these tumours have usually undergone partial involution but complete involution does not occur. Kaposiform haemangioendothelioma is associated with *Kasabach–Merritt phenomenon*, characterized by thrombocytopenia $<25 \times 10^9/L$, petechiae and bleeding. The other tumour related to this phenomenon is *tufted angioma*, which has distinct clinical and histological features that distinguish it from kaposiform haemangioendothelioma. The primary treatment for kaposiform haemangioendothelioma is vincristine.¹⁶ Other therapeutic options include rapamycin and sirolimus.¹⁷ Platelet transfusion is contraindicated unless there is active bleeding or surgical intervention is planned. Although the primary indication for therapeutic treatment is Kasabach–Merritt phenomenon, drug therapy is also considered for large asymptomatic tumours to minimize fibrosis and consequent pain and stiffness.

Pyogenic granuloma is a small, solitary pedunculated red papule that commonly occurs in childhood. The primary complications associated with this lesion are bleeding and ulceration. These tumours have been treated with laser, curettage, electrocautery and shave excision, with a recurrence rate in excess of 40%. The latter is in view of the fact that these lesions extend into the reticular dermis and therefore effective management requires full-thickness resection.

Vascular malformations

Vascular malformations arise from abnormal development of vascular elements during embryogenesis. Although present at birth, vascular malformations typically only become evident in

childhood or occasionally, in adult life, do not undergo involution and grow commensurate with the child. Malformations are grouped into fast flow versus slow flow, with the former containing an arterial component. Genetic aberrations may give rise to vascular malformations, but the majority are non-syndromic and non-familial. Arteriovenous malformations may be caused by mutations in endoglin (hereditary haemorrhagic telangiectasia type 1, *HHT1*), activin receptor-like kinase 1 (*HHT* type 2), *RASA1* (capillary malformation–arteriovenous malformation) or *PTEN* (PTEN-associated vascular anomaly). Venous malformations can result from mutations in the *TIE2* (sporadic venous malformations, cutaneomucosal venous malformation), glomulin (glomovenous malformation), *CCM1/KRIT1*, *CCM2* or *CCM3* (cerebral cavernous malformations) genes. Mutations in *VEGFR3*, *FOXC2*, *SOX18* or *CCBE1* genes are responsible for primary lymphoedema.² Unlike infantile haemangiomas, vascular malformations tend to demonstrate an equal sex distribution.

Capillary malformations commonly present at birth as red cutaneous discolorations. Over time, the colour often deepens and the skin thickens to eventually adopt a raised ‘cobblestone’ appearance. A proportion of lesions are associated with contiguous soft tissue and bony hypertrophy. Traditionally, facial capillary malformations have been described as occurring in one of the three trigeminal nerve branch distributions (45%), with a greater proportion (55%) involving more than one dermatome or occurring bilaterally. However, there does not appear to be any pathophysiologic relationship with the trigeminal nerve. With capillary malformations in the so-called first trigeminal nerve distribution, *Sturge–Weber syndrome* should be considered.¹⁸ *Sturge–Weber syndrome* is a sporadic disorder, with an incidence of 1 in 50 000. The capillary malformation involves the first trigeminal nerve distribution, but may extend into the second and third (Figure 16.4). The two other cardinal features of this syndrome are ocular anomalies (choroidal vascular abnormalities, glaucoma) and leptomeningeal involvement (capillary, venous or arteriovenous malformations). Ophthalmology consultation and intracranial MRI examination are mandatory in these patients.

Midline capillary malformations are occasionally associated with CNS anomalies, such as encephalocele, occult spinal dysraphism, tethered spinal cord or arteriovenous malformations of the spinal cord (Cobb syndrome). Capillary malformations, particularly facial malformations, can result in significant psychological morbidity secondary to the disfigurement often associated with these lesions. Pulsed dye laser is now routinely offered as early as 1 year of age as a therapeutic modality. Laser treatment is highly beneficial in attenuating pigmentation and lessening the likelihood of developing secondary complications including darkening, dermal thickening and pyogenic granulomas. In later childhood, adolescence and into adulthood, surgical debulking may be required to correct any anatomic deformity.

Lymphatic malformations, often referred to as ‘cystic hygromas’ or ‘lymphangiomas’, result from aberrant development of lymphatic channels and are grouped into macrocystic, microcystic



Figure 16.4 Capillary malformation involving the first, second and third trigeminal nerve distributions bilaterally in a patient with *Sturge–Weber syndrome*.

or combined lesions, depending on the size of the malformed channels, with microcystic usually defined as containing cysts less than 1–2 cm in diameter.¹⁹ Macrocystic lesions are the most common subtype of lymphatic malformation and typically arise in the neck, although they occur in any anatomic region including mesentery, orbits and bladder. Lymphatic malformations can be apparent at birth, presenting as a large, soft, fluctuant swelling with absence of overlying skin discoloration (Figure 16.5). These lesions transilluminate and are usually non-tender. Some present later in life following an acute intralesional bleed, with firm, tender clot-filled cysts (Figure 16.6). Lymphatic malformations are also prone to sudden expansion with concurrent, usually viral, illness. Most lesions are easily diagnosed on ultrasound alone; however, MRI may be required to further define deep extension in complex lesions (Figure 16.7).

Microcystic lesions are firmer on palpation relative to their macrocystic counterparts. Moreover, the malformation may involve the contiguous skin and/or mucosa, presenting as vesicles, which may seep serous fluid or bleed. Microcystic



(a)



(b)

Figure 16.5 (a, b) Clinical images of a child with a large mixed macro- and microcystic lymphatic malformation involving the chest, axilla and upper extremity.



Figure 16.6 Clinical image of a child with a microcystic lymphatic malformation of the right upper lip. This lesion was complicated by an episode of intralesional bleeding.

lesions often have a large connective tissue component and on MRI demonstrate localized tissue overgrowth with a high T2 signal and a diffuse mild enhancement pattern. Lymphatic malformations are occasionally combined with cutaneous capillary malformations and with venous anomalies, most commonly as a *Klippel-Trénaunay* type association in a lower limb. Differentiation between lymphatic and venous malformations can be facilitated with focused physical examination. Although both types of malformations are soft and compressible, unlike venous malformations, lymphatic lesions do not expand with Valsalva manoeuvre and the overlying skin is often normal or displays only a subtle bluish hue. Moreover, venous malformations are more diffuse and ill defined when compared with lymphatic lesions. Lymphatic malformations may cause significant morbidity secondary to mass effect, chylous effusions, recurrent infection (sepsis), bleeding, pain and deformity.

The first-line therapy for macrocystic or combined (macro-/microcystic) lymphatic malformations is usually percutaneous sclerotherapy, which involves the injection of a sclerosing agent into the cysts to collapse their walls and prevent their re-expansion (Figure 16.8). Sclerotherapy is also utilized to control recurrent

infection and intralesional bleeding. Over the last three decades, multiple sclerosant agents including OK-432, sodium tetradecyl sulfate (STS), ethanol, bleomycin and doxycycline have been used in the treatment of lymphatic malformations. In general, doxycycline, STS and bleomycin are currently the preferred sclerosants. Although microcystic lymphatic malformations tend to be less amenable to sclerotherapy, largely in view of the more solid nature of these lesions, some series have shown a good response rate with bleomycin.

The most common complications associated with sclerotherapy are infection, cutaneous ulceration and nerve injury; with these potential risks varying according to the agent used. Lymphatic malformations that fail to respond to sclerotherapy, can be considered for surgical resection. Seroma formation is the most common complication following surgical extirpation, and can be minimized by careful application of compressive dressings. Complex and extensive lesions are associated with more significant morbidity, including infection, delayed healing, peripheral nerve injury and deformity. Resection carries an important rate of recurrence, especially for lesions that involve multiple tissue planes where excision is usually incomplete (Figure 16.9).

Venous malformations may present earlier than lymphatic malformations due to the associated discoloration of the overlying skin. However, many are deep-seated and are not problematic until they grow with puberty.²⁰ Venous malformations exhibit variable morphology, from focal round lesions (often mistaken for haemangiomas) to diffuse and ill-defined lesions involving multiple tissue planes, or clusters of dysplastic veins involving the entire limb. Most commonly, venous malformations are restricted to subcutaneous tissue and muscle (Figure 16.10), though lesions may extend to deeper structures, with bone and joint involvement seen on imaging. In fact, venous malformations frequently occur around joints, particularly the knee, and as such cause symptoms with movement. Lesions can be associated with pain, swelling, bleeding and deformity (Figure 16.11). Moreover, extensive venous malformations can be

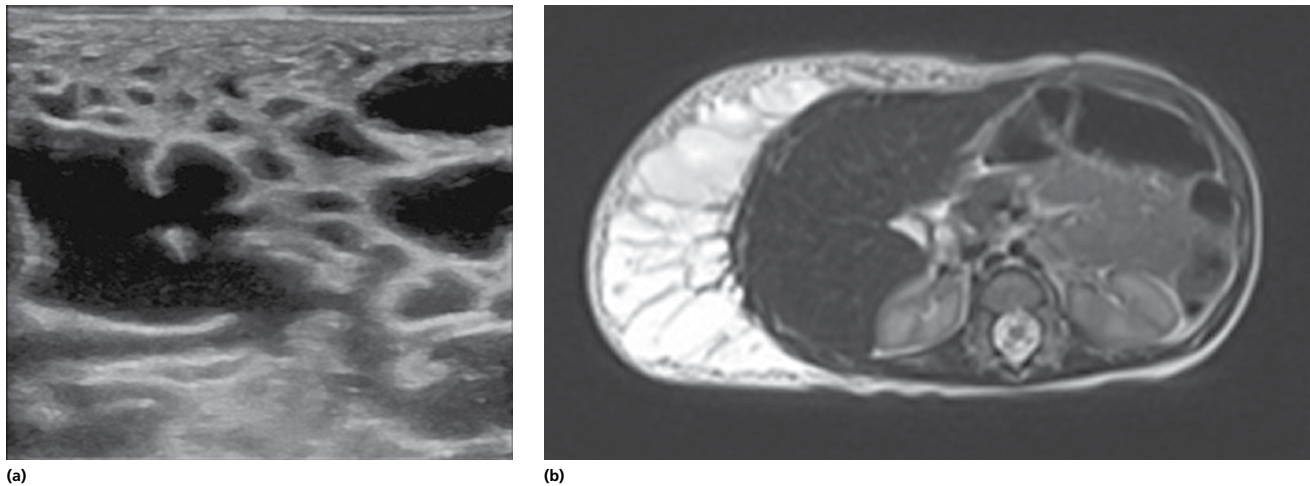


Figure 16.7 (a) Ultrasound image of the lymphatic malformation of the chest wall in the child shown in Figure 16.5. Cysts of varying sizes can be seen, separated by thin septae. (b) T2-weighted axial MRI image of the same lymphatic malformation. The majority of the macrocysts lie deep to the microcysts.

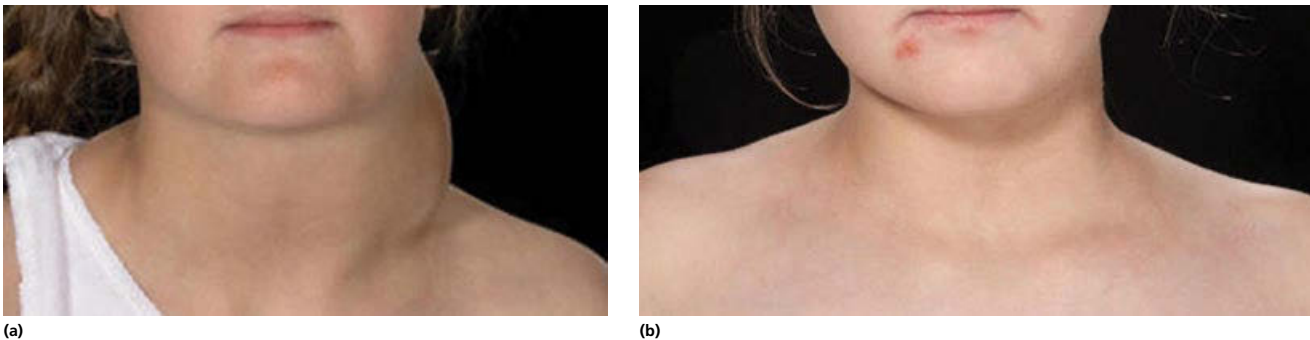


Figure 16.8 Clinical images of a macrocystic lymphatic malformation of the neck (a) before and (b) after a single sclerotherapy treatment.

associated with localized intravascular coagulopathy and phlebothrombosis. On physical examination, these lesions are found to enlarge with dependent positioning and Valsalva manoeuvre. Calcified phleboliths can often be palpated within the mass, and are pathognomonic on imaging, appearing as rounded signal voids. Ultrasound is useful to identify the slow flow nature of the lesion. On MRI, these malformations are hyperintense on T2-weighted imaging and, contrary to lymphatic malformations, enhance with contrast (often with a patchy distribution of enhancement due to the slow flow of contrast through the malformation) (Figure 16.12). Most malformations cause pain secondary to venous congestion and thrombus formation.

For venous malformations of the extremity, use of compression garments are recommended to reduce venous stasis, expansion, and localized intravascular coagulopathy. Like lymphatic malformations, sclerotherapy is usually the therapy of choice for treatment of venous malformations, with resection reserved for well-localized lesions that can be removed completely or malformations that remain symptomatic or cause important residual deformity despite sclerotherapy. The therapeutic mechanism of sclerotherapy for venous malformations is the same as for lymphatic malformations,

wherein the sclerosant induces fibrosis within the malformation, thereby reducing venous stasis and mass effect. For these lesions, STS and ethanol are the preferred sclerosants.

The complication rates are higher for venous sclerotherapy than for lymphatic lesions, given the more aggressive nature of the agents used for treatment of venous malformations. Both STS and ethanol are associated with a potential for tissue necrosis. When surgical resection is considered, caution must be exercised to avoid aggravating the existing deformity, and thus conservative resection is recommended especially in view of the fact that complete removal is often unattainable and recurrence is high. Some centres advocate the use of prophylactic aspirin for recurrent pain secondary to phlebothrombosis and low-molecular-weight heparin in patients with significant localized intravascular coagulation.

Two syndromes commonly associated with venous malformations are *Maffucci syndrome* and *blue rubber bleb naevus syndrome*. The former is characterized by cutaneous venous malformations, with enchondromas and/or bony exostoses. The latter includes multiple, small (<2 cm) venous malformations localized to skin, subcutaneous tissue and

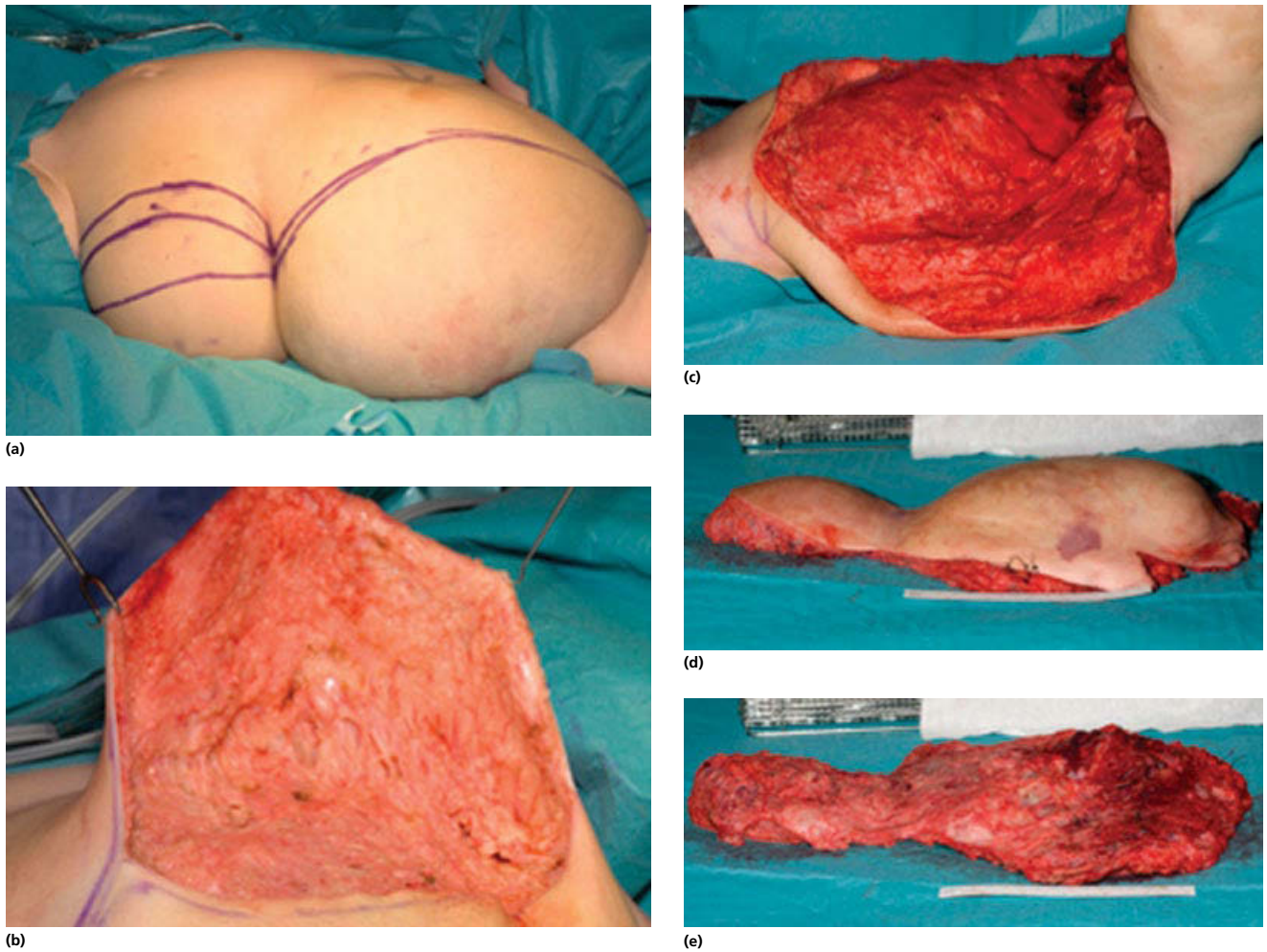


Figure 16.9 The lymphatic malformation of the child in Figure 16.5 was initially treated with sclerotherapy, following which surgical extirpation was undertaken in view of the extent of the lesion and associated disability it engendered. Intraoperative photographs, including (a) markings of the malformation, (b) the malformation *in situ*, (c) chest wall and axilla following excision of the lesion, and (d, e) the excised malformation.



Figure 16.10 Clinical images of a child with a venous malformation of the left cheek.



Figure 16.11 Clinical image of a venous malformation of the hand. The significant anatomic deformity caused considerable functional impairment.

most particularly the gastrointestinal tract. The recently proposed treatment of blue rubber bleb naevus syndrome is rapamycin (sirolimus).²¹

Klippel-Trénaunay syndrome is a capillary-lymphatic-venous malformation of an extremity (lower extremity in 95% of cases) with considerable soft tissue and bony overgrowth, often to disfiguring proportions (Figure 16.13). The malformation may extend to involve the pelvis. A large embryonic vein in the subcutaneous tissue of the lateral aspect of the leg and thigh (*marginal vein of Servelle*) is present. Management includes measures outlined to address the capillary, lymphatic and venous components of the malformation and requires a multidisciplinary approach.

Arteriovenous malformations are the rarest form of vascular malformation, although clinically they usually attract the most attention.²² These lesions arise from an error in vascular development during embryogenesis, where abnormal connections are created between arterial and venous systems without intervening capillaries. The latter developmental aberration results in vascular shunting, tissue overgrowth and arterial steal. Although some arteriovenous malformations remain small and inconsequential throughout life, others can demonstrate insidious growth, causing significant morbidity secondary to pain, ulceration, bleeding, tissue destruction and cardiac failure. The majority are located within the musculoskeletal tissues of the trunk and extremities. Lesions localized to the head and neck often have complex deep connections (Figure 16.14).

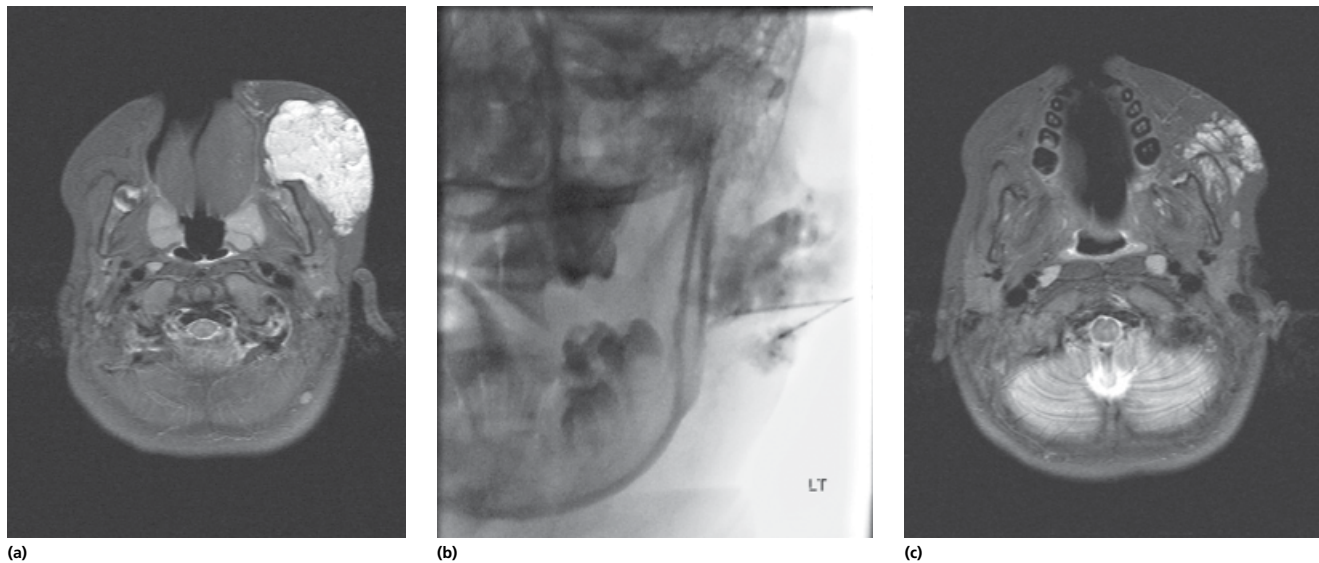


Figure 16.12 (a) T2-weighted axial MRI image of a venous malformation of the left cheek in a child. The malformation is visualized as a lobulated, well-defined mass within the subcutaneous tissue. (b) Intraoperative fluoroscopic image of sclerotherapy of the lesion. A two-needle technique was used to instill opacified sclerosant into the malformation. (c) T2-weighted axial MRI image of the same venous malformation following four sclerotherapy treatments. The malformation was considerably reduced in size and the distortion of the adjacent soft tissues of the cheek was lessened.



Figure 16.13 Clinical images of a child with a capillary-lymphatic-venous malformation of the lower extremity, demonstrating considerable overgrowth and distortion of the limb, characteristic of Klippel-Trénaunay syndrome.



Figure 16.14 Clinical image of an arteriovenous malformation of the ear.

Most malformations can be diagnosed on clinical examination, as a localized thrill and/or bruit is present and the soft tissues are typically warm and diaphoretic. Ultrasound can be used to confirm the diagnosis in difficult cases but there is generally no role for cross-sectional imaging. Angiography should be reserved for those cases where an interventional radiologist is planning to treat the lesion definitively. Schobinger proposed a classification system for arteriovenous malformations based on clinical characteristics. In stage I (quiescence), where the lesion is warm, pink and shunting is seen on colour Doppler examination. In stage II (expansion), there is enlargement of the

lesion, and a thrill or bruit may be noted. Stage III (destruction) is characterized by dystrophic skin changes, ulceration, bleeding and pain. Finally, in stage IV (decompensation) congestive heart failure is present.

Arteriovenous malformations remain the most challenging lesions to treat effectively. Management is complicated by the fact that these anomalies progress over time, complete extirpation is rarely possible, recurrence after treatment is high, and resection may be associated with significant morbidity and may result in more considerable deformity than the malformation per se. Liu *et al.*²³ recommend resection for Schobinger stage I and II arteriovenous malformations in non-anatomically sensitive areas, where progression to a higher stage will render extirpation more difficult or if there is an aggravation of symptoms or deformity, for stages I and II lesions respectively. Intervention for stage III and IV lesions is dictated by the degree of associated symptoms. Embolization should be considered as a first-line treatment for most lesions, as it is minimally invasive and can be directed to the point of shunting, traditionally called the *nidus* (Figure 16.15). The latter can also be implemented prior to surgical extirpation to reduce the size of the malformation preoperatively and intraoperative blood loss. Incomplete embolization or excision often stimulates the malformation to enlarge and become problematic. Thus, management of these complex lesions should remain the remit of an experienced multidisciplinary team.

Parkes-Weber syndrome is characterized by small, diffuse multifocal arteriovenous malformations with associated extremity overgrowth and an overlying pale capillary malformation. The *capillary malformation-arteriovenous malformation* is an autosomal dominant condition associated with *RASA1* gene mutation. Patients characteristically present with atypical capillary malformations that are small, multifocal pink lesions surrounded by a pale halo, and can be associated with internal arteriovenous malformations.

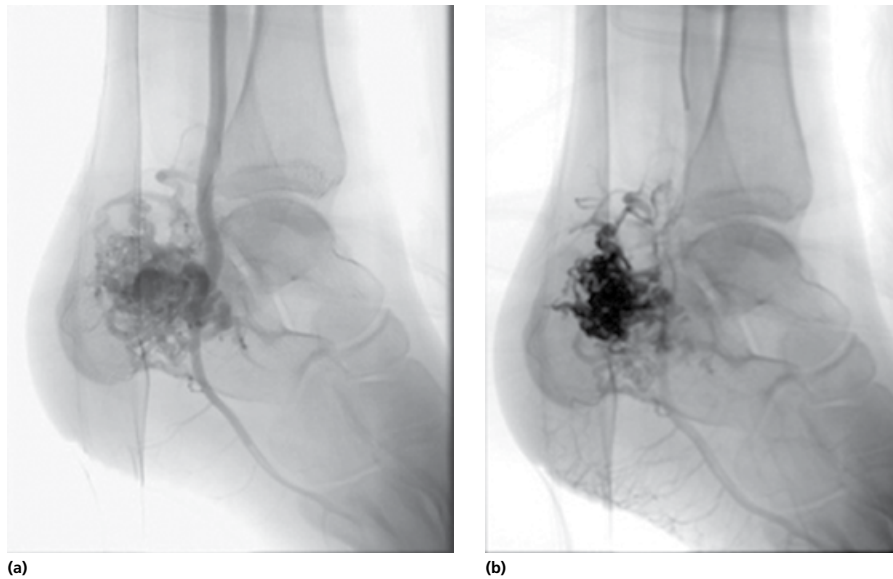


Figure 16.15 (a) Angiography image demonstrating a focal high flow arteriovenous malformation of the left ankle, which is supplied by the posterior tibial artery. (b) Angiographic image following embolization, demonstrating dense onyx material filling nearly the entire malformation while preserving the posterior tibial artery.

Conclusion

Management of vascular anomalies necessitates interdisciplinary care by a dedicated team with expertise in plastic surgery, dermatology, interventional/diagnostic radiology, general paediatric surgery, orthopaedic surgery, haematology/oncology, pathology and vascular surgery. Moreover, allied health professionals such as psychologists, occupational therapists and physiotherapists are also integral members of the team. In the majority of cases, the correct diagnosis can be made by thorough history and physical examination. Appropriate imaging and biopsy for histological analysis can assist where diagnostic ambiguity remains. Treatment depends on the nature, size and location of the lesion, associated symptoms and anatomic deformity. Ultimately, management demands a holistic approach, which renders this field of medicine such a challenging and rewarding speciality.

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CHAPTER 17

Cleft lip

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Embryology

Between the fourth and seventh weeks of gestation, the two medial nasal prominences of the frontonasal process fuse to form the intermaxillary segment (Figure 17.1), consisting of the premaxilla and prolabium. In addition, the two maxillary prominences merge, from which the lateral lip and maxillary arch elements arise. By week 7 of gestation, mesenchymal confluence follows apoptosis of the epithelial seams between the medial nasal and maxillary prominences, resulting in the formation of a complete upper lip. Failure of fusion of the medial nasal prominence with the ipsilateral maxillary prominence results in a unilateral cleft lip and, if replicated bilaterally, a bilateral cleft lip results. The ratio of unilateral left, unilateral right and bilateral cleft lip deformity is 6:3:1, respectively. The lateral nasal prominences form the alae of the nose and do not contribute to the upper lip. At the molecular level, several major signalling pathways, including *BMP*, *FGF*, *Shh* and *Wnt*, are critical for midfacial morphogenesis, and subtle abnormalities in any one of these processes may lead to oro-facial clefting.

Cleft lip is defined as a congenital abnormality of the primary palate. It may be complete, forme fruste or incomplete, unilateral or bilateral, and may be associated with a palatal cleft. The *forme fruste* cleft is qualified by a notch in the vermilion, from which a visible band or furrow may extend to the mid-nostril floor, and is associated with irregularity of the cleft-side alae. A *Simonart* band occurs in approximately 30% of patients with a unilateral cleft lip and palate, and describes the small soft tissue bridge, most often devoid of orbicularis oris muscle, that spans the lateral and medial elements of a cleft lip.^{1,2} The integrity of the underlying alveolus is probably of greater clinical relevance than the magnitude of the soft tissue band.

In the context of sporadic clefting, the risk of unaffected parents with one child with cleft lip $\pm$ palate having a second affected child is approximately 4%, whereas with two affected children, the risk increases to 9%.^{3,4} If one parent has a cleft lip $\pm$ palate, the risk of having an affected child is similarly around 4%, which increases to 17% for a second affected child.

The anatomical severity of the cleft has a direct influence on the recurrence risk in first-degree relatives, with the type of cleft being predictive of the type of recurrence.⁴

First reported in 1969, most obstetric units in the developed world now routinely offer a mid-trimester fetal anomaly ultrasound scan, with facial examination being advocated since 1984.^{5,6} Using two-dimensional antenatal ultrasound, a Norwegian study detected up to 57% of cases of cleft lip $\pm$ palate, of which 43% had an associated anomaly; no cases of isolated cleft palate were diagnosed antenatally.⁷ Diagnostic accuracy has improved with the advent of three-dimensional sonography, which has led to a sensitivity of 95% and a specificity of 92.3%.⁸ In high-risk populations (i.e. those with a positive family history of oro-facial clefting), detection rates of 100% have been achieved.⁹ Antenatal MRI offers no particular advantage over ultrasound for the diagnosis of cleft lip, but has significant potential in improving the diagnosis of isolated cleft palate.¹⁰

Clinical anatomy

The orbicularis oris is the highly complex sphincteric muscle of the lip which facilitates feeding, speech and oral continence.¹¹ It consists of four substantially independent quadrants – upper, lower, left and right – each of which contains a smaller pars marginalis and a larger pars peripheralis. The surface marking of the junction between the marginalis and peripheralis fibres is the border between the vermilion and skin. The pars peripheralis fibres fan out obliquely from the modiolus to interdigitate with the other muscles of facial expression (including zygomaticus major superiorly and depressor anguli oris inferiorly) and terminate within the dermis. Their role is primarily one of articulation and facial expression.

The midline philtral dimple of the non-cleft upper lip is flanked by symmetrical philtral columns that arise from each peak of the Cupid's bow and curve gently cephalad to the base of the columella. The ridges of the philtral column are created by dermal insertions of the pars peripheralis fibres as they interdigitate in the

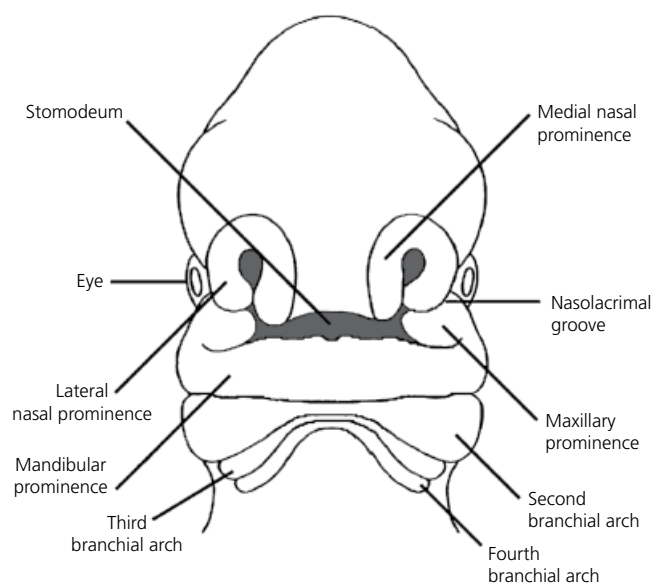


Figure 17.1 Embryology of the primary palate. Failure of fusion of the medial nasal prominence and the ipsilateral maxillary prominence results in a unilateral cleft lip.

midline, each crossing approximately 5 mm into the contralateral half of the upper lip. The Cupid's bow peaks are created by the pull of the levator labii superioris fibres inserting into the base of the ipsilateral philtral column.¹² The cutaneous roll (alternatively, the 'white roll' of Gillies or the 'white skin roll' of Millard) is the mucocutaneous junction between the vermillion and skin. The cutaneous roll runs from commissure to commissure, but is most prominent centrally at the Cupid's bow and gradually attenuates laterally. It contains four to five rows of progressively superficially positioned pilosebaceous units containing tiny unpigmented silky vellus hairs, which, by reflecting ambient light, highlight the contour of the Cupid's bow. The vermillion has an associated midline tubercle caused by the eversion of opposing pars marginalis fibres. The vermillion-mucosal junction ('red line' of Noordhoff) represents an abrupt transition between the keratinized vermillion and thick non-keratinized squamous epithelium of the oral mucosa.¹³

In the unilateral cleft lip, the Cupid's bow is preserved in the medial lip element. The lip height, as measured from the cleft-side peak of Cupid's bow to the base of the columella, is characteristically deficient and the bow is sloped. The cutaneous roll of the entire length of the bow is well preserved. However, there is vermillion height deficiency below the cleft-side half of Cupid's bow.¹³ The proposed peak of Cupid's bow on the lateral lip element has traditionally been less well defined. Noordhoff advocated preserving the proposed base of the philtral column as it is an anatomically determined landmark. He defined it as being located at the point where the cutaneous roll and the vermillion-mucosal junction begin to converge medially (Figure 17.2).¹⁴ At 'Noordhoff's point' there is adequate vermillion height (usually matching the height of the vermillion below the non-cleft side peak of Cupid's bow) and good-quality

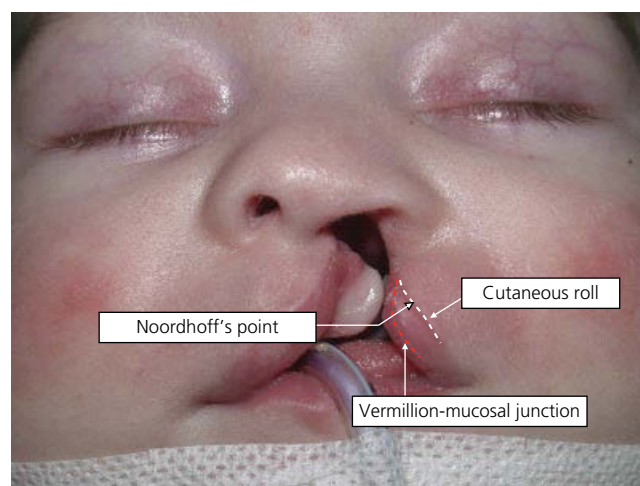


Figure 17.2 Infant with a complete left cleft lip and palate. The cardinal position of Noordhoff's point is marked.

cutaneous roll. Medial to Noordhoff's point, the quality of these features diminishes.

The height of the lateral lip element (as measured from the subalare to Noordhoff's point) is frequently different from the height of the non-cleft side (subalare to Cupid's bow peak). The latter is often excessive in incomplete clefts and usually shorter in complete clefts.^{15,16} The transverse length of the lateral lip element (Noordhoff's point to the commissure) is almost always shorter than that of the non-cleft side (Cupid's bow peak to the commissure). Lateral lip element hypoplasia, defined as combined height and transverse length deficiency, is present in almost two thirds of patients with a unilateral cleft.¹⁶

The orbicularis oris muscle is misdirected by the cleft (Figure 17.3).^{17,18} On the cleft side, the pars superficialis changes direction as it approaches the cleft to run almost vertically to find insertion to the cleft-side nostril and periosteum of the piriform aperture. The pars peripheralis inserts into the lateral aspect of the alar and the nasolabial fold; contraction of the lateral lip element orbicularis produces a noticeable bulge in the lateral lip. On the non-cleft side, the orbicularis fibres insert into the base of the columella and nasal septum. Activation of orbicularis exacerbates the nasal deformity by splaying the alar cartilages, broadening the nasal tip, shortening the columella, and deviating the nasal septum to the non-cleft side. Successful correction of the unilateral lip deformity must address these aberrant muscular attachments.

In complete bilateral cleft lip and cleft palate, the prolabium and premaxilla are entirely separated from the lateral lip and maxillary arch elements. The dimensions of the prolabium will vary considerably between affected individuals, but there are some consistent findings. The cutaneous portion of the philtrum is devoid of the dimple and ridges that, in the non-cleft lip, define the shape and dimensions of the philtrum. The cutaneous roll of the prolabium is typically poorly defined when compared with the lateral lip elements, although it is usually

better preserved in an incomplete bilateral cleft lip. The height of the prolabial vermillion is inadequate with the superior labial sulcus being shallow or, in some patients, entirely absent. Orbicularis muscle does not enter the prolabium, and therefore lies passively upon the premaxilla, which is protruded and may, in extreme cases, adopt a horizontal posture. The lateral lip elements in the bilateral cleft are similar to the lateral lip element of a unilateral cleft.

The inferior and superior labial arteries (which arise from the facial artery at the modiolus) are the primary blood supply to the lips (Figure 17.4).¹⁹ They course close to the free border of the upper and lower lips, lying deep to the orbicularis muscle, juxtaposed to the mucosa, to anastomose with its fellow from the opposite side. The facial artery continues cranially along the

line of the nasolabial fold, and after giving off the lateral nasal branch, becomes the angular artery, which then anastomoses with the dorsal nasal artery. The ascending septal branches of the superior labial artery nourish the columella and anastomose with the terminal branches of the anterior ethmoid artery and the lateral nasal artery.

In the unilateral cleft, the superior labial artery on the cleft side is unable to pass horizontally through the lip and therefore anastomoses with the ipsilateral lateral nasal artery. The resultant interruption of the upper lip arterial arcade is of little surgical importance in unilateral cases due to the density of collateral blood supply. However, in complete bilateral clefts, the prolabium receives no input from either the superior labial arteries or, indirectly, via the greater palatine arteries, and is therefore entirely

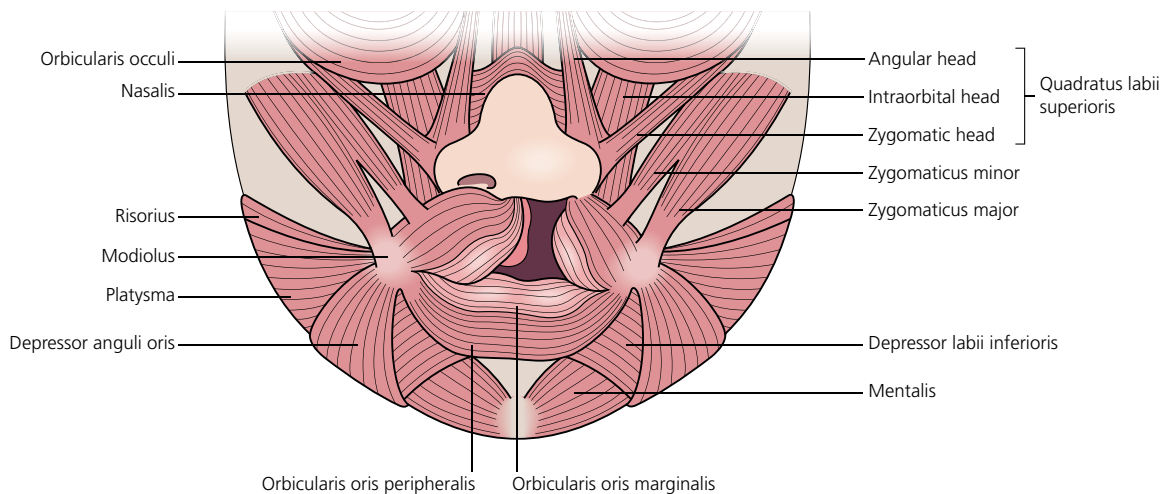


Figure 17.3 Orientation of the orbicularis oris fibres in a patient with a complete left unilateral cleft lip. Source: Millard.²⁶ Reproduced with permission of Lippincott, Williams & Wilkins.

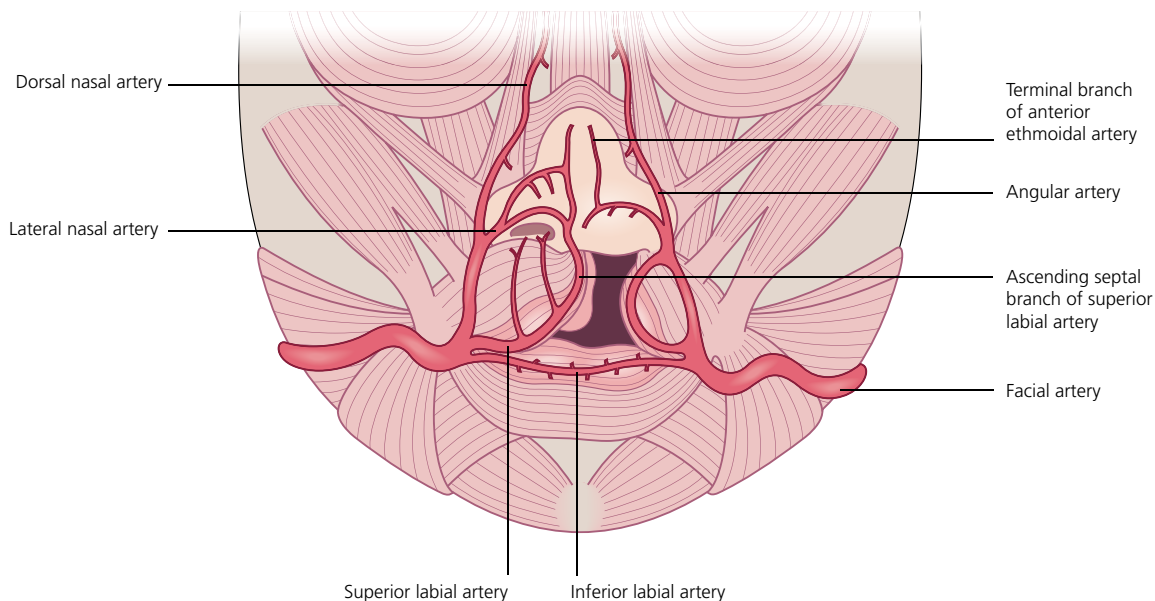


Figure 17.4 Blood supply to the nasolabial complex in a patient with a complete left unilateral cleft lip. Source: Millard.²⁶ Reproduced with permission of Lippincott, Williams & Wilkins.

reliant upon the terminal branches of the posterior septal artery (arising from the sphenopalatine artery), the anterior ethmoid artery and the lateral nasal artery.

The characteristics of the nasal deformity in the complete unilateral cleft were first described in 1931, and consists of a depressed cleft-side nasal dome and splayed alar cartilages, with eversion of the alar rim and exposure of the nasal lining.²⁰ The cleft medial and lateral crura are not believed to be hypoplastic based on both cadaveric dissections and intraoperative observations.^{21–23} The cleft alar base is depressed due to the underlying maxillary hypoplasia. Further, anomalous insertion of orbicularis oris into the alar base retracts it both laterally and inferiorly. The nasal spine, caudal nasal septum and columella, together with the premaxilla, are deviated to the non-cleft side due to the unopposed action of the aberrant insertion of orbicularis oris. Nasal obstruction may result from septal deviation, turbinate hypertrophy, or obstruction of the external nasal valve by nasal webbing. In the bilateral cleft nose, the columella is typically short or nearly absent due to deficient soft tissue and the septum may be excessively broad.

Preoperative assessment

It is essential to ensure that the infant is optimized prior to surgery; significant comorbidities are common in syndromic children and will inevitably require specialist paediatric input. Early liaison with the cleft anaesthesia team is recommended, particularly if there are potential airway concerns. Failure to thrive is seen in approximately 40% of children with a palatal cleft and 9% of those with an isolated cleft lip, thus all cleft patients must be closely monitored preoperatively to ensure that they are gaining weight appropriately.²⁴ Close cooperation between the family and the cleft unit (via the clinical nurse specialist, dietician and feeding specialist) is essential at this stage.

Although probably less controversial than the timing of palatal closure, currently no global consensus exists for the optimal timing for cheiloplasty. In 1966, Wilhelmsen and Musgrave proposed the 'rule of tens' as a criterion for lip surgery. Namely, weight greater than 10 pounds (4.5 kg), haemoglobin over 10 g/dL and age over 10 weeks (which replaced a white cell count of less than 10×10^3 per mm³ in their original iteration).²⁵ Millard observed that lip closure could occur at any time from birth to old age, and he advocated waiting until the child was three months of age as this achieved the best results in his opinion.²⁶ Indeed, the majority of cleft centres defer surgery until the age of 3–6 months, since it allows the optimization of underlying medical issues, minimizes the anaesthetic risks, and allows time for presurgical orthopaedic treatment if indicated.

Straith performed neonatal lip repair under local anaesthesia in the 1940s²⁷ and many cleft centres continue to perform neonatal cleft lip repair, sometimes as early as 48 h of age.²⁸ It has been argued that with modern specialist neonatal care and anaesthetic support, the perioperative risks are minimized, and

neonatal repair potentially facilitates improved integration of the child into the family network. There is little evidence to suggest that operating on neonatal cleft lips is unsafe.^{29–31} Although parents may express preference for earlier surgical intervention, there is no evidence of significant psychological morbidity in mothers when delaying repair until the age of three months.^{32,33}

The concept of an intrauterine cleft lip repair has garnered much interest due to the potential for scarless wound healing prior to 24 weeks gestation.³⁴ Furthermore, fetal repair might reasonably be expected to reduce the magnitude of the secondary 'downstream' dentoalveolar and midfacial deformities.³⁵ However the technical challenges are vast, and although feasible in both small (murine and lapine) and large (ovine) animal models, albeit with guarded aesthetic and functional results, the perceived benefits of fetal repair do not currently outweigh the significant risks of such early intervention.^{36–39}

The role of preoperative microbiological analysis in an attempt to predict the nasal and oropharyngeal flora at the time of cleft surgery is unhelpful, and culture results appear to have no influence on postoperative outcome, regardless of whether antibiotics were used.⁴⁰ If children with an unrepaired cleft lip and palate undergo preoperative microbiological analysis (with swabs taken from the nasopharynx and oropharynx), approximately 30% will have positive cultures, with the predominant pathogen being *Staphylococcus aureus*; beta-haemolytic streptococci were isolated in a small proportion of patients.⁴¹ The clinical significance of these results is unclear and debate continues as to whether preoperative microbiology analysis is justified. The evidence for cancelling an elective cleft procedure if a child has a concurrent upper respiratory tract infection remains ambiguous, although most cleft surgeons are cognizant of the increased postoperative risks, and would therefore be reluctant to proceed, favouring an honourable deferral for four weeks until the child has fully recovered.⁴²

A typical treatment pathway, from antenatal diagnosis to adulthood, for a child with a unilateral complete cleft lip and palate, as practised at The Hospital for Sick Children in Toronto, Canada, is outlined in Table 17.1. Although a child with an isolated incomplete cleft lip may require just the single operation, the burden of care in the case of a complete bilateral cleft of the lip and palate is far more substantial.

Presurgical orthopaedics

Historically, the first record of facial binding in order to realign the premaxilla in a complete bilateral cleft was in 1686.²⁶ In the context of a complete unilateral cleft lip, presurgical orthopaedics (PSO) is defined as the use of a device to improve the alignment of the maxillary arch segments in order to facilitate lip repair, and was first reported by McNeil in 1950 by utilizing a removable intraoral plate, and subsequently popularized by Burston.^{43,44} The principles have also been applied to the complete bilateral cleft lip in order to correct the protruded premaxilla.

Table 17.1 Typical surgical treatment pathway for a child with a unilateral complete cleft lip and palate

Age	Treatment
Prenatal diagnosis	Genetic counselling (if requested by family) Parental consultation with cleft surgeon
2 weeks	Presurgical orthopaedics (if indicated)
3–6 months	Cleft lip repair and primary nasal correction
10–14 months	Palate repair ± grommets
≥3 years as required	Lip revision (if indicated)
≥4 years as required	Surgical correction of velopharyngeal incompetence (if indicated)
≥7 years as required	Lobule rhinoplasty (if indicated)
8–10 years (mixed dentition)	Alveolar bone graft
≥7 years as required	Definitive septorhinoplasty (if indicated)
≥16–18 years (skeletal maturity) as required	Orthognathic surgery (if indicated)

**Figure 17.5** Nasoalveolar moulding plate *in situ* with left nasal stent evident in a three-month old infant with a complete unilateral cleft lip and palate.

PSO devices may be passive, such as a dental plate or facial strapping, or active, such as Latham's appliance, which is pinned to the child's maxilla, and is adjusted by turning an integral coaxial screw, that gradually aligns the alveolar segments.⁴⁵ In 1999, Grayson *et al.* introduced the concept of nasoalveolar moulding (NAM), whereby the oral plate has nasal prong extensions that are able to reposition the deformed nasal cartilages and lengthen the columella (Figure 17.5).⁴⁶ The resultant approximation of the alveolar segments allows the surgeon to perform a primary gingivoperiosteoplasty at the time of the lip repair, which is said to limit the need for secondary alveolar bone grafting in up to 60% of patients.^{47,48} In those institutions who do not practise PSO/NAM, alternative strategies can be utilized to reduce alveolar cleft width, such as single-layer vomerine flap closure of the hard palate at the time of lip repair, with subsequent secondary bone grafting of the alveolus.^{49–51}

In spite of considerable enthusiasm for the deployment of PSO/NAM by its proponents, its adoption is by no means universal, with some units reserving its use for more severe deformities such as the wide unilateral cleft or asymmetrical bilateral cleft, while many units do not offer it as a treatment option. This may, in part, reflect concerns regarding the additional burden of care placed on families undergoing PSO/NAM treatment with the associated healthcare costs incurred. Moreover, robust evidence in support of the long-term positive effects of PSO or NAM is lacking, although, as any surgeon who has struggled with a difficult primary lip repair may testify, the short-term benefits cannot be completely ignored. A recent systematic review of the literature failed to demonstrate a long-term benefit to the use of PSO,⁵² and a systematic review examining the evidence for NAM reported that although there is a trend towards a positive effect, the results are inconsistent regarding long-term changes in nasal symmetry.⁵³ In 2013 Sommerlad presented the interim results of a randomized controlled study comparing PSO with controls in complete unilateral cleft lip and palate patients; no difference in aesthetic or functional outcomes could be discerned at their 5-year and 10-year audit assessments.^{54,55} The issue of PSO/NAM has a tendency to polarize opinion in the worldwide cleft community; a position that is unlikely to change until randomized controlled trial evidence with long-term follow-up becomes available for scrutiny.

Unilateral cleft lip repair

The first recorded cheiloplasty was performed in China in AD 390.⁵⁶ In renaissance Europe, Paré was extremely influential, with contemporaneous texts describing the scarification ('paring') of the cleft margins and approximation of the raw edges using needles and sutures.⁵⁷ Husson first recognized that by creating curved incisions, a degree of lengthening of the lip repair could be achieved on wound apposition; a technique later refined by both Rose and Thompson.^{58,59} In 1844 Mirault identified that straight-line scars resulted in unsightly lip shortening, with the potential for notching, and introduced the concept of a triangular flap from the lateral lip element to both lengthen the lip and disrupt the scar.⁶⁰ In 1949, LeMesurier recognized the importance of reconstructing the Cupid's bow using a quadrilateral flap from the lateral lip element, although it was Tennison who preserved the anatomy of the bow, and Randall who reduced the size of the triangular flap.^{61–63} Millard introduced his rotation-advancement technique in 1957, which has subsequently undergone numerous modifications.^{64,65} In 2005 Fisher published his anatomical subunit approximation technique, which utilizes the wound-lengthening principles of Rose and Thompson with the anatomic subunit approach of Berget and Mennick.^{14,66,67}

Principles of surgical repair

Regardless of the surgical technique employed, the principal goals of unilateral cleft lip repair remain constant. These include approximation of the medial and lateral lip elements at all levels

(i.e. nostril sill, cutaneous roll, vermillion–cutaneous junction and vermillion–mucosal junction) without loss of natural landmarks. Balance should be achieved by providing length where tissue is short and excision where height is excessive; poor-quality tissue at the cleft margins should ideally be discarded. The scar of union should be placed along natural lines, with appreciation of the anatomical subunits of the nasolabial complex. The nostril margins should be of equal circumference and the alar bases should be symmetric from the anterior view, with careful attention paid to reconstruction of a natural ‘nostril sill roll’.⁶⁸ Complete release of the aberrant attachments of the orbicularis oris muscle must be performed before undertaking a functional muscle reconstruction.

The height of the non-cleft side (i.e. the philtral column length from the lip–columellar crease to the Cupid’s bow peak) provides the dimension that must be created by the cleft-side reconstruction. The medial cleft-side height (i.e. the philtral column length from the lip–columellar crease to the projected Cupid’s bow peak) is short and will necessitate lengthening from the lateral cleft-side lip height. The entire length of the Cupid’s bow should be preserved in the medial lip element. In the lateral lip element, Noordhoff’s point should be conserved and used to form the base of the philtral column incision. The cutaneous roll of the medial and lateral lip elements should be approximated in a side-to-side fashion (creating a ‘butt joint’). Vermillion height deficiency below the cleft-side half of Cupid’s bow should be augmented, with attention paid to recreating a level vermillion–mucosal junction.

Operative techniques

Tennison–Randall inferior triangle repair

The inferior triangle repair is a Z-plasty where the middle limb of the Z is shared by the incised medial and lateral cleft margins (Figure 17.6). Whereas Tennison used a bent wire to stencil

equal limbs of the Z-plasty scar, Randall refined the technique using calipers and geometric planning in order to minimize the size of the inferior triangle.^{62,63} The total lip height of the non-cleft side is measured from a reference point in the nostril sill to the peak of the ipsilateral Cupid’s bow. The greater lip height is measured on the medial cleft side from the proposed medial point of closure at the height of the lip in the nostril sill (symmetric with the reference point in the non-cleft-side nostril sill) to the peak of the cleft-side Cupid’s bow. The difference between the total lip height and the greater lip height (i.e. the lesser lip height) stipulates the base width of the inferior triangle required to level Cupid’s bow.

There are a number of advantages of the inferior triangle repair. First, nostril sill closure is accomplished by simple side-to-side approximation of the medial and lateral nostril sill elements, with resultant scarring at the base of the nose being kept to a minimum. Second, adequate lengthening of the medial lip height can be achieved even when the medial lip height is very short. Furthermore, the position of Noordhoff’s point need not be compromised when the lateral lip height is short, due to the lengthening effect of the Z-plasty. The repair may be criticized due to the non-anatomical alignment of the Z-plasty scar and the fact that secondary revisions may be hampered by the presence of the sizeable triangular flap.

Millard rotation–advancement repair

The rotation–advancement technique is reported to account for the majority of lip repairs performed worldwide.⁶⁹ The ‘rotation flap’ is created by a curvilinear incision directed along the medial lip element, such that it mirrors the non-cleft-side philtral column in its lower half (Figure 17.7).⁶⁴ The incision then gently arcs into the territory of the philtrum and approaches (but should never cross) the non-cleft-side philtral column. This permits caudal rotation of the cleft-side peak of Cupid’s bow, and the resultant defect is resurfaced with a large triangular ‘advancement’ flap from the lateral lip element. The triangular portion of prolabial skin lateral to the ‘rotation flap’ forms the ‘columella base flap’, or ‘c-flap’, which rotates cranially to reconstruct the cleft-side nostril sill. Millard noted that the rotation of Cupid’s bow was often inadequate and subsequently introduced a ‘back-cut’,⁶⁵ which extends from the most cranial point of the rotation incision down the philtrum just medial to the non-cleft-side philtral column. The resultant defect is now quadrilateral-shaped (rather than triangular) and can be filled by caudal rotation of the ‘c-flap’, however this risks lowering the transverse limb of the repair.

The main appeal of this repair is the ‘cut-as-you-go’ approach and the preservation of the Cupid’s bow and philtral dimple. In the lower portion of the lip, the scar mirrors the non-cleft-side philtral column, however the aesthetic result is compromised in the upper half of the lip as the scar fails to emulate a normal philtral column. Furthermore, excessive scarring is created at the base of the nose, and if the ‘c-flap’ is utilized to backfill the secondary defect created by the back-cut, it can no longer

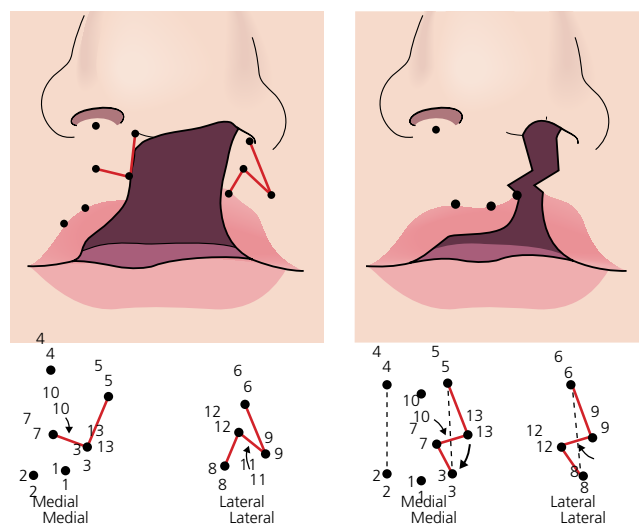


Figure 17.6 The inferior triangle repair of Tennison as modified by Randall. Source: Randall, 1959.⁶³ Reproduced with permission of Lippincott Williams & Wilkins.

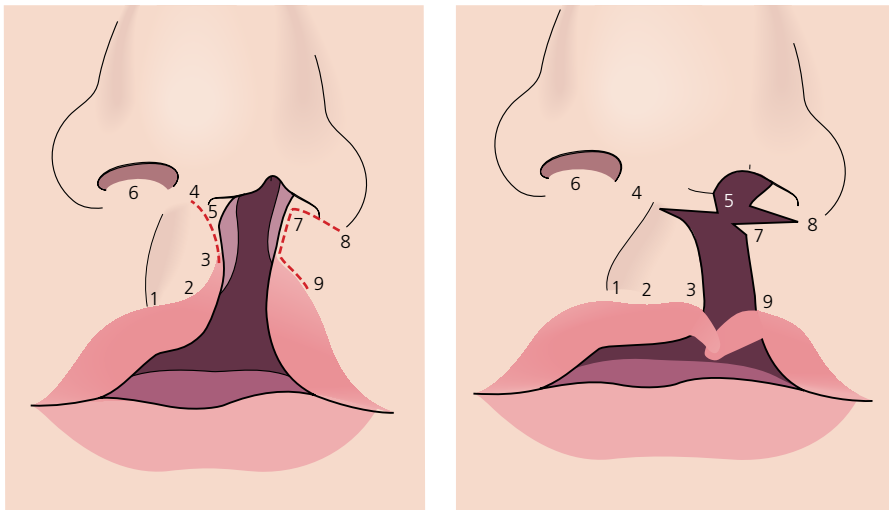


Figure 17.7 Millard's original 'rotation-advancement' technique of unilateral lip repair. Source: Millard, 1960.⁶⁴ Reproduced with permission of Lippincott Williams & Wilkins.

contribute to closure of the nostril sill, with the resultant risk of potential nostril stenosis. The cleft-side alar base incision in Millard's original description produces a noticeable scar and is therefore best avoided; indeed many surgeons believe that such an aggressive alar base incision is unnecessary. The length of the marginal incision at the lateral lip must be of sufficient length to marry up with the full length of the corresponding rotation incision. In the case of a complete cleft with a vertically short lateral lip element, it may be necessary to extend the incision beyond Noordhoff's point in order to achieve adequate vertical height, which will further compromise the transverse length of the lateral lip. A criticism of the Millard technique, particularly in the wide cleft, is that it tends to produce a 'short' lip, which can be 'vertically short' if related to an inadequate rotation incision or back-cut, or 'transversely short' if Noordhoff's point is violated.⁷⁰

In 1987, Mohler published a modification to the standard rotation-advancement repair in an attempt to produce a scar that is more symmetric with the non-cleft philtral column in the upper half of the lip (Figure 17.8).⁷¹ The curve of the rotation incision is straightened such that it extends into the columella base rather than towards the non-cleft philtral column. A 90° back-cut is created to the level of the labio-columella crease, which allows the columella tissue to descend by transposing a Z-plasty into the lip such that the 'advancement flap' is not required to encroach as medially, thus improving the lie of the scar. A relative contraindication to this approach is therefore a narrow columella.¹⁴ The resultant transverse limb of the repair is elevated such as to lie inconspicuously within the lip-nose junction. In common with all rotation-advancement repairs, the Mohler modification is prone to compromise of the transverse lateral lip length when the lateral lip is vertically short.

Noordhoff also modified Millard's original repair.¹⁴ In the 'Chang Gung repair', if rotation of Cupid's bow is inadequate, a small opening back-cut is created just above the cutaneous roll at the cleft-side peak of Cupid's bow, into which is incorporated a small triangular flap from the lateral lip element. As well as providing additional height, the triangular flap also

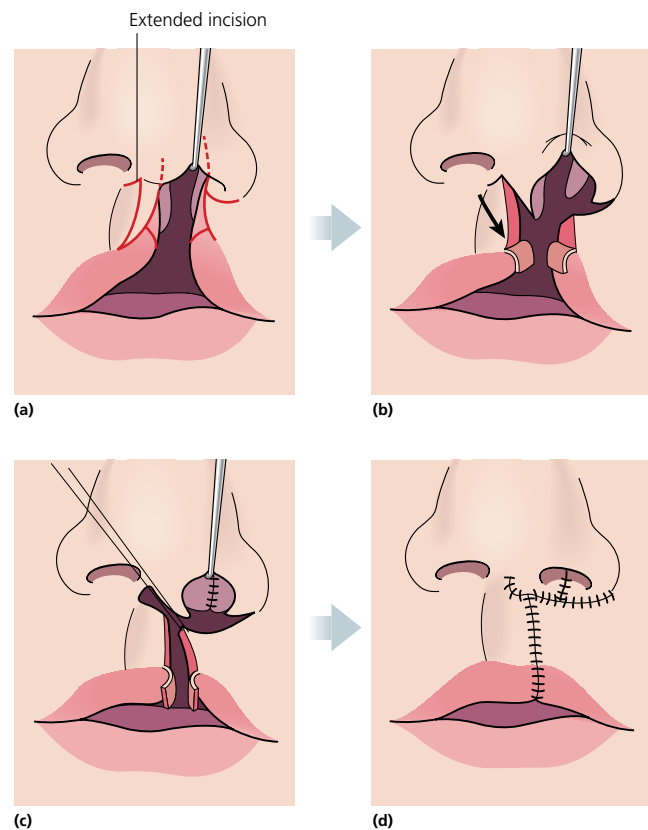


Figure 17.8 Mohler's modification of the 'rotation-advancement' repair with extension of the rotation incision into the columella and a right-angled back-cut to allow transposition of a Z-plasty into the lip. Source: Mohler, 1987.⁷¹ Reproduced with permission of Lippincott Williams & Wilkins.

helps to accentuate the pout of the lip. Noordhoff also augmented the deficient vermilion below the cleft-side Cupid's bow by means of a laterally based vermilion flap. Numerous additional modifications have been made to Millard's original 'rotation-advancement' approach, with Stal *et al.* providing a comprehensive account of the evolution of the technique.⁷²

Fisher anatomical subunit approximation repair

The anatomical subunit approximation technique aims to produce a cutaneous scar along the 'ideal line of repair' (Figure 17.9).⁷³ The incision on the medial cleft side ascends from a point above the cutaneous roll such that it mirrors the ridge of the non-cleft philtral column. It then curves superolaterally along the lip–columellar crease to the medial point of closure in the nostril sill. Lip length is achieved by two mechanisms: the previously described Rose–Thompson principle (which accounts for approximately 1 mm of lengthening) and, if required, a small inferior triangle from the lateral lip element, which is positioned just superior to the cutaneous roll (a modification of the Tennison–Randall repair). The lengthening achieved by the Rose–Thompson principle significantly reduces the size of the inferior triangle that would otherwise be required had a traditional inferior triangle repair been performed. The technique also utilizes a Noordhoff's lateral vermilion triangle flap to augment the deficient vermilion height of the medial lip element on the cleft side.

This technique has a number of advantages over the inferior triangle and rotation–advancement repairs. In variance with the Millard technique, there is minimal scar at the base of the nose. The lip scar is positioned along the seams of anatomical subunits with the exception of, when required, a small triangle above the cutaneous roll. Tension is ideally positioned above the roll. Continuity of the cutaneous roll is achieved by side-to-side approximation of the roll elements. In common with the inferior triangle repair, lateral lip transverse length need not be compromised to achieve vertical height.

A summary of the skin markings for the anatomical subunit approximation technique is highlighted in Figure 17.9. The Cupid's bow and philtral column heights are marked in the standard fashion, with the cleft-side bow peak and philtral column heights being equidistant relative to the midline as the corresponding non-cleft-side markings. Note that the non-cleft-side Cupid's bow peak is not marked at the highest point of the curve of the lip, but rather where the straight portion of the

non-cleft-side half bow meets the convexity of the lateral lip element, as may be appreciated in Figure 17.9. No such convexity exists on the cleft side of the medial lip element. Therefore, the curve of the bow on the cleft side will come from the lateral lip element lateral to Noordhoff's point.

The point of origin of the opening incision for the medial lip back-cut is then located just above the cutaneous roll (the 'supra-white roll point') at a point in line with the cleft-side bow peak and perpendicular to the vermilion–cutaneous junction. Caudally, from this point, the incision line will continue across the free border of the lip into the mucosa. The medial lip incision (equivalent to the 'greater lip height') will ascend from the supra-white roll point to the height of the cleft side philtral column (within the lip–columellar crease). The incision line then traverses superolaterally to the medial point of closure in the nostril sill (equivalent to the 'third height').

The medial and lateral points of closure within the nostril sill are not distinct anatomical points and are arguably the hardest points to define in this repair. The medial point of closure in the nostril sill is positioned lateral to the curve of the lip–columellar crease. It will be positioned more medially in complete cases and more laterally in incomplete cases. The lateral point of closure in the nostril sill is then chosen on the lateral lip element, relative to the medial point of closure in the nostril sill, such that when these two points are approximated, two goals are accomplished: nares of equal circumference and symmetry of the alar bases from the anterior view. Gentle manipulation of the medial and lateral lip elements should confirm acceptable positioning of these points. In complete clefts where the lateral lip is tethered such that the medial and lateral lip elements cannot be brought together, it is advisable to release the lateral lip element first.

Calipers are used to measure the 'total lip height' and the 'greater lip height'. The 'total lip height' is measured with the lip at rest and is defined as the distance from the height of the non-cleft philtral column (at the lip–columellar crease) to the non-cleft-side supra-white roll point (i.e. the non-cleft side equivalent to the point of origin of the opening incision for the medial lip back-cut). The 'greater lip height' is measured with gentle downward traction on the lip to unfurl the medial lip and is defined as the distance from the height of the cleft-side philtral column (at the lip–columellar crease) to the point of origin of the opening incision for the medial lip back-cut. The 'lesser lip height' (equivalent to the base width of the small inferior triangle) is equal to the 'total lip height' minus the 'greater lip height' and less 1 mm (Figure 17.10). The 1 mm accounts for the approximate length achieved by the Rose–Thompson principle. If an inferior triangle is required, the back-cut for the opening incision is drawn perpendicular to the 'greater lip height' incision, and of a length equal to the 'lesser lip height' (the inferior triangle being equilateral). The back-cut rotates the cleft-side peak of Cupid's bow caudally. The 'lesser lip height' is usually between 1 and 1.5 mm and never exceeds 2 mm. For incomplete clefts, an inferior triangle is often not necessary.

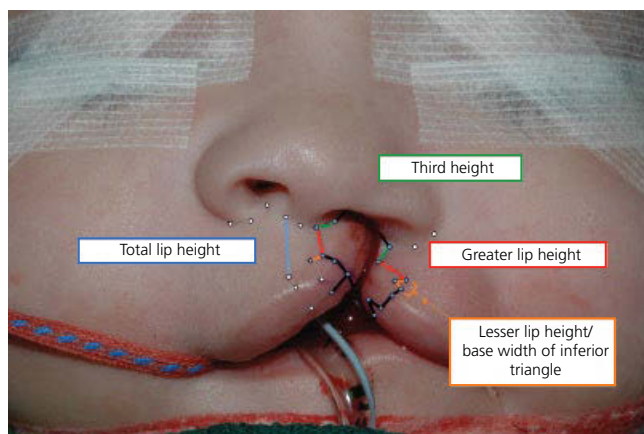


Figure 17.9 The anatomical subunit approximation technique as described by Fisher. Key markings are shown, including the total lip height, greater lip height, lesser lip height and the third height.

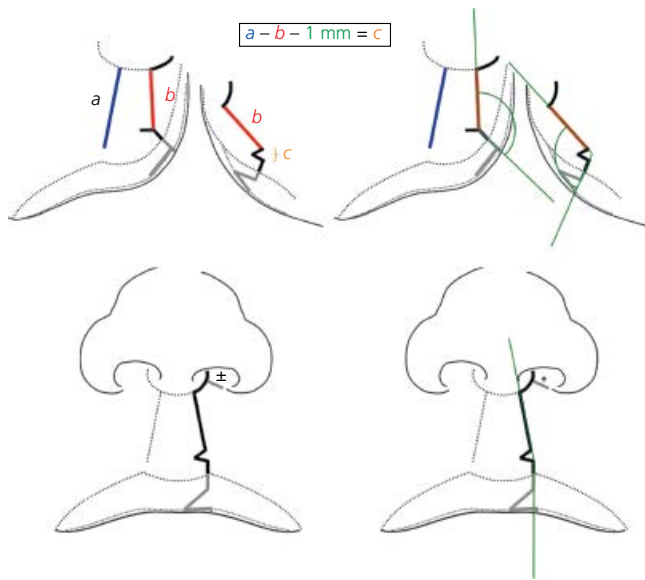


Figure 17.10 The total lip height (*a*) and greater lip height (*b*) are measured from points just above the cutaneous roll (the supra-white roll point) at the bow peaks to the height of the ipsilateral philtral column in the lip–columellar crease. The base width of the inferior triangle (*c*) can then be calculated. Approximately 1 mm of lengthening occurs by the Rose–Thompson principle, thus reducing the size of the inferior triangle.

With respect to the lateral lip, Noordhoff's point is marked along the vermillion–cutaneous junction at the point where the cutaneous roll and red line begin to converge medially, as previously described (Figure 17.2). The natural convexity of the cutaneous roll lateral to Noordhoff's point is ideally suited for reconstruction of the cleft side curve of the bow. A supra-white roll point is marked above the cutaneous roll both in line with Noordhoff's point and perpendicular to the vermillion–cutaneous junction. Between the lateral point of closure in the nostril sill and the supra-white roll point on the lateral lip, three lengths must be accommodated: the greater lip height, the lesser lip height and the 'third height' (corresponding in length to the line connecting the height of the cleft-side philtral column and the medial point of closure in the nostril sill). This is readily achieved by placing the greater lip height caliper on the lateral lip element such that its lower tip lies the lesser lip height distance away from the supra-white roll point and its upper tip lies the third height distance away from lateral point of closure in the nostril sill.

In the scenario of a complete cleft with a short vertical height to the lateral lip element, the angle of the greater lip height incision will be more horizontal and, in extreme cases, may be confluent with the upper limb incision of the inferior triangle (Figure 17.11a). The contrary position is seen in an incomplete cleft with a relative excess of vertical height; the third height may be lengthened by lateralizing the medial and lateral points of closure within the nostril (Figure 17.11b). If vertical height excess persists in spite of this manoeuvre, it may be necessary to perform a medially based wedge excision of the upper lip below the lateral point of closure in the nostril sill (Figure 17.11c). The

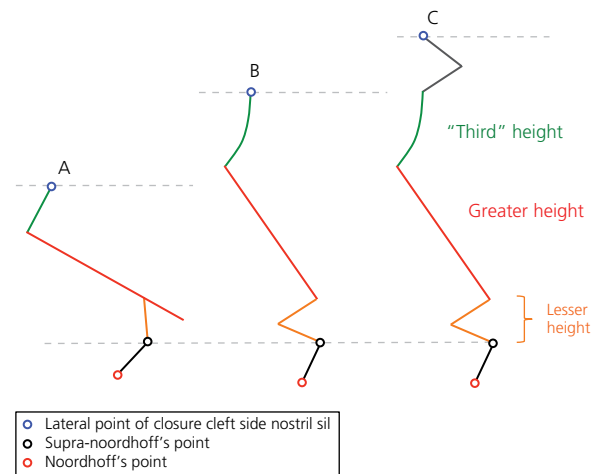


Figure 17.11 Lateral lip markings vary depending on the vertical height of the lateral lip element. Note that the position of 'Noordhoff's point' is not compromised regardless of the lateral lip height.

lateral apex of this wedge excision should never extend laterally beyond subalare as this is unnecessary and leaves an unsightly scar. Care should be taken not to alter the vertical height of the lateral point of closure in the nostril sill as this will compromise the final vertical height of the alar insertion.

The vermillion deficiency below the cleft-side half of Cupid's bow requires augmentation. An opening incision is created along the vermillion–mucosal junction below the cleft-side half of Cupid's bow. This back-cut will receive a laterally based vermillion triangular flap from the lateral lip element as described by Noordhoff.

In incomplete clefts a wedge excision of the nostril sill above the medial and lateral points of nostril sill closure is performed. The excision should be sufficient to balance the circumference of the nares, with great care taken not to over-resect as the resultant nostril stenosis presents an unenviable reconstructive challenge. The mucosa of the cleft margins is removed by wedge excision; a short lateral upper buccal sulcus advancement incision may be helpful on occasions.

In complete clefts, the mucosal incisions on the medial and lateral lip elements extend cranially to the points of attachment of each lip element to the greater and lesser alveolar segments, respectively. An upper buccal sulcus incision extending laterally from the point of attachment of the lateral lip element is made. From the medial point of closure in the nostril sill, the incision continues intranasally along the caudal margin of the septum for a distance of up to 12 mm to facilitate access for septal repositioning. The lateral free edge of the septal flap will receive the medial free edge of the lateral vestibular flap. The lateral vestibular flap is fashioned by incising from the lateral point of closure in the nostril sill to the attachment point of the lateral lip element to the alveolus and then along the piriform margin to allow supra-periosteal mobilization of the alar base. An anteriorly based inferior turbinate flap may be required to augment nasal floor closure in clefts with a significant anteroposterior distance between the greater and lesser maxillary segments.

Bilateral cleft lip repair

A fundamental advantage in bilateral cleft lip deformity is that of symmetry, however the philosophical approach to managing the premaxilla has evolved significantly over time.²⁶ In 1556, Franco erroneously advocated primary excision of a protruding premaxilla – a practice now universally condemned. Fortunately others subsequently recognized its importance and attempted to preserve it, often using quite elaborate devices to provide compression or traction in an attempt to correct its alignment.⁷⁴ As the significance of the reconstructed orbicularis muscle in moulding the underlying premaxilla became recognized, the benefits of soft tissue closure over surgical setback on maxillary growth became evident.^{75,76} The concept of a staged reconstruction by utilizing a temporizing lip adhesion has gained widespread acceptance.² The destiny of the prolabium has provoked much debate, as techniques were devised to relocate it within the columella, the lip, or both. With a small prolabium, the importance of maintaining length of the frontonasal component was the motivation for using lateral lip skin flaps to augment below the prolalial component.⁷⁷ In 1965 Manchester described a single-stage closure in which the prolabium is left attached to the premaxilla, the mucosa unrolled and the lateral lip elements sutured to the sides of the prolalial component, without a muscle repair.⁷⁸ This technique therefore *preserved* the prolalial cutaneous roll and vermillion. Conversely, Millard's fundamental contribution was to *discard* the prolalial cutaneous roll and vermillion and introduce the lateral lip elements in order to achieve a single-stage bilateral lip closure. He also promulgated the concept of forked flaps to lengthen the columella.^{79,80} Talmant favoured an unscarred Cupid's bow and, in a variant of the Manchester technique, preserved a large triangular mucosal flap from the prolabium to provide enhanced volume to the central tubercle.⁸¹ Mulliken, in an adaptation of the Millard technique, also discards the prolalial cutaneous roll and vermillion.⁸² He utilizes presurgical orthopaedics by means of the Latham appliance and creates a narrow biconcave prolalial flap, establishes a median tubercle from the lateral labial elements, and aims to anatomically realign the alar cartilages.

The principles of bilateral cleft lip repair were outlined by Mulliken and include: the maintenance of symmetry (i.e. simultaneous bilateral lip repair), primary muscle reconstruction, adequate reduction of the prolabium, reconstruction of the median tubercle and Cupid's bow from the lateral lip elements, and primary rhinoplasty.⁸² PSO/NAM is commonly employed to control of the protruded premaxilla and to reduce the risk of the lateral alveolar segments collapsing medially thus creating a 'locked out' premaxilla.⁶⁸ A bilateral lip adhesion may be considered if PSO/NAM is either unavailable, not tolerated by the patient/family, or has been unsuccessful in correcting the premaxilla. The aim is to restore continuity to the upper lip such that it provides a physical constraint to the growth of the premaxilla. The incisions are designed so as to preserve the landmarks necessary for a subsequent definitive lip repair.² Premaxillary setback is no longer recommended as a primary procedure due to the deleterious effects on midfacial growth.⁸³

Mulliken initially advocated a two-staged repair, however he subsequently abandoned the use of forked prolalial flaps in favour of a single-stage repair (Figure 17.12).⁸⁴ He found that sufficient primary columellar lengthening and nasal tip projection could be achieved in a single procedure by manipulation of the alar cartilages (which are splayed and rotated caudally). He ceased creating an additional vertical nasal tip incision and accessed the alar cartilages via bilateral rim incisions alone. As the prolalial cutaneous roll and vermillion is of poor quality and should be discarded, Mulliken recommended a biconcave prolalial flap that is 2–2.5 mm wide at the lip–columellar angle and 3.5–4 mm wide between the bow peaks. The upper labial sulcus is invariably deficient in complete bilateral cleft lips and is usually augmented by a caudally based prolalial mucosal flap that is draped over the premaxillary periosteum in order to reconstruct the posterior wall of the upper labial sulcus.

Fisher's method of bilateral lip repair utilizes a tie-shaped prolalial shield flap, although this may be modified depending on the ethnicity of the patient and the parental appearance.⁸⁵ The dimensions of the prolalial flap in a 4- to 6-month-old child are typically 2.5–3 mm wide at the lip–columellar junction, 3.5–

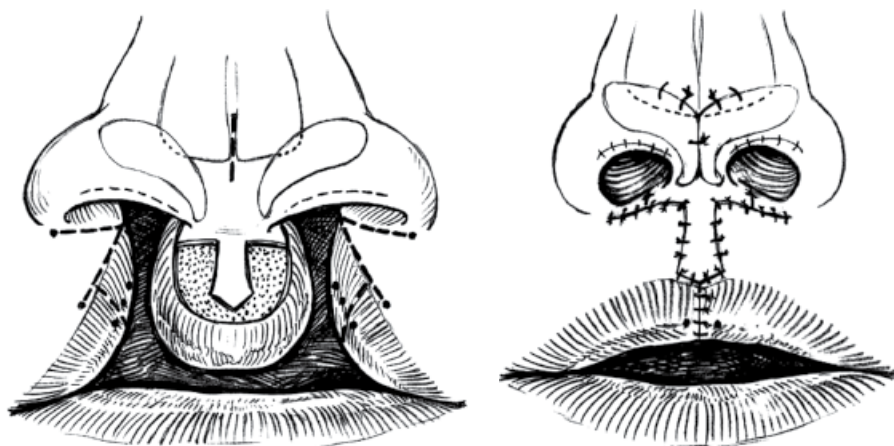


Figure 17.12 Mulliken's method of single-stage bilateral complete cleft lip repair. Source: Kohout *et al.*, 1998.¹⁰³ Reproduced with permission of Lippincott Williams and Wilkins.

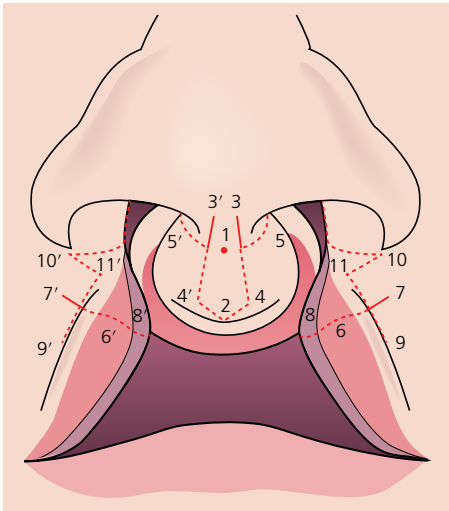


Figure 17.13 Fisher's surgical markings for repair of a complete bilateral cleft lip. Points on the patient's left side are described, with the corresponding right-sided points assigned the prime (') symbol. The corners of the prolabial skin are marked 3 and 4; 1 and 2 denote its height. Point 5 is just lateral to the lip–columellar crease. Point 6 denotes Noordhoff's point. Point 7 is the supra-white roll point, with point 8 being below Noordhoff's point on the vermilion–mucosal junction. Point 9 is made just above the cutaneous roll lateral to point 7. The distance between points 7 and 9 should equal the distance between points 2 and 4. Point 10 is placed medial to the alar insertion and meets point 5 at the height of the lip repair at the anterior nostril sill. Point 11 is positioned near the cleft margin at a distance from point 9 that is equal to the distance between points 3 and 4, and at a distance from point 10 that is equal to the distance between points 3 and 5. Source: Panossian & Fisher, 2008.⁸⁵ Reproduced with permission of Taylor & Francis.

4.5 mm wide between the bow peaks, and 6–8 mm in height (Figure 17.13). From the base of the prolabial flap at the lip–columella angle, the incisions pass superolaterally in a curvilinear fashion into each nostril sill. Noordhoff's point is marked on each lateral lip segment at the vermilion–cutaneous junction; in perpendicular orientation with the vermilion–cutaneous junction, and in alignment with Nordhoff's point, a supra-white roll point and a vermilion–mucosal junction point are marked. The incision along these three points is convex medially across the vermilion in order to replicate the median tubercle. A lateral back-cut is created from the supra-white roll point parallel with the vermilion–cutaneous junction, thus creating a cutaneous roll–vermillion flap whose geometry precisely corresponds to the caudal dimensions of the prolabial flap. Release of the lateral lip elements is performed via an upper lateral buccal sulcus incision in order to minimize the tension of the repair, with the soft tissues being freed from the underlying maxilla in a supra-periosteal plane such that the alar bases are completely mobilized. Aberrant insertions of the orbicularis oris muscle should be released from the lateral lip skin and the alar bases to allow descent of the cutaneous roll–vermillion flaps. The upper labial sulcus is recreated in a similar manner utilizing a prolabial mucosal flap. The inter-alar base width is reduced by means of an alar base cinch suture. A conservative primary rhinoplasty is advocated with the alar base

dissection extending cephalad in order to release the attachments of the alar–accessory cartilage complex from the lateral piriform margin. The nasal tip is not routinely dissected and internal nasal valve plication sutures are employed.

Primary nasal correction

The timing of nasal correction may be considered to be primary (concurrent with cheiloplasty), intermediate (during the school years) or secondary (once facial growth is complete). The concept of primary repositioning of the alar cartilages was advocated by McComb, who disproved the belief that manipulation of the nasal cartilages might adversely affect growth.^{86,87} The Tajima technique utilizes a reverse-U incision along the alar margin and redistributes the overhanging skin of the alar margin to augment deficient lining.⁸⁸ Open approaches, such as that described by Trott and Mohan, afford excellent visualization of the alar domes, but may compromise prolabial blood supply if adopted in complete bilateral cases, and carry a risk of nostril stenosis.⁸⁹ The goals of primary rhinoplasty include improving nasal contour, symmetry and nasal tip projection, with relocation of the caudal nasal septum. Primary rhinoplasty may reduce the complexity of a definitive secondary rhinoplasty procedure.⁹⁰ The potential risks of a primary rhinoplasty include iatrogenic deformity to the skin envelope and the introduction of scarring which may complicate subsequent interventions.

Nasal splints, such as the commercially available silicone Koken conformers, are frequently used after primary rhinoplasty as they are believed to maintain the nostril contour postoperatively, and may reduce the risk of relapse.^{91,92} Evidence for their efficacy is largely anecdotal. The optimal duration of treatment is much debated, ranging from 5 days to many months or more. Moreover, patient/parental compliance is essential and is typically the rate-limiting step for their continued use.

Postoperative care

The necessity for elbow restraints is much debated.⁹³ Our practice is for them to be worn for two weeks postoperatively. Children may resume oral feeding immediately in the recovery room following surgery. Most units discharge patients the following day, although day-case repairs are becoming increasingly common in selected patients.⁹⁴ If non-absorbable sutures are used, removal is undertaken on the 5th–7th postoperative day, often under sedation or general anaesthesia. Concerns regarding repeated anaesthetics on the neurocognitive development of children, a desire to minimize the burden of care in this patient cohort, and to rationalize healthcare resources, has resulted in the use of absorbable skin sutures becoming increasingly widespread.⁹⁵ Absorbable sutures may be associated with increased complications, including abscess formation or permanent suture marks, however, to date, the two closure methods have not been scientifically evaluated in the

setting of a randomized controlled trial. Significant variance exists with regard to the need for a postoperative dressing. The preference at The Hospital for Sick Children in Toronto is for topical bacitracin-polymyxin B ointment, which allows adequate wound toilet to prevent excessive crusting. The use of a Logan's bow to minimize tension across the lip repair is still practised in some centres.⁹⁶

Complications and revision surgery

Airway obstruction and anaesthetic complications are rare in otherwise healthy infants. Rare potential complications include bleeding, infection and wound dehiscence. A degree of scar contraction is inevitable, which will lead to a shortened lip at the three-month postoperative check. However, it invariably responds favourably to regular scar massage. Hypertrophic scars are less common, with keloid scars being extremely rare, even in darker skin types. Late inclusion dermoid cysts are occasionally seen.

Lip repairs performed with adherence to the defined principles are capable of producing reconstructions of excellent quality with minimal secondary deformity. Minor revisions, such as mucosal redundancy on the cleft side, can usually be deferred at least until the time of alveolar bone grafting. More significant deformities invariably warrant a complete lip revision. Timing is critical from the perspective of the child's psychosocial development; indeed the deformity may need addressing prior to commencing school. A secondary septorhinoplasty is commonplace in patients with a complete unilateral cleft lip and palate, and should be deferred until facial growth is complete.

Outcomes

Determining objective standardized outcome measures for cleft patients has been notoriously difficult to achieve. Indeed no international unanimity exists.⁹⁷ The 1992 Eurocleft study was the first international multicentre audit to compare treatment results in children with a unilateral cleft lip and palate.⁹⁸ Nasolabial aesthetics were assessed by a panel using cropped two-dimensional photographs, with significant differences in outcome noted between centres.⁹⁹ Limitations exist with still photographs, with video analysis essential for the assessment of dynamic facial appearance and the quality of orbicularis oris reconstruction. Three-dimensional imaging is an exciting adjunctive tool in aesthetic assessment. Quantitative measurement of nasolabial symmetry from two-dimensional photographs is now possible using SymNose software.¹⁰⁰ A universally agreed objective method of facial aesthetic assessment is vital if robust comparisons are to be made between centres.

The primary aim of cleft lip repair is to achieve a functional and aesthetically acceptable nasolabial appearance by minimizing the visible stigmata of the cleft-related deformity. However, the psychological status of cleft patients, in particular their socio-emo-

tional functioning, is critical for their ability to fully integrate into society and lead content and fulfilling lives. Cleft patients achieve a similar level of educational and vocational attainment as controls, although economic achievement may be less marked.¹⁰¹ Patients with cleft lip and palate have impaired psychological wellbeing with increased levels of anxiety and depression.¹⁰² Many avoid positions that expose them to direct public contact; cleft patients are less likely to marry, particularly those with a bilateral deformity. These issues highlight the importance of the clinical psychologist as an integral member of the cleft service.

Conclusion

In 1937 Kilner stated 'there is no branch of surgery that offers greater fascination than that which concerns itself with the repair of congenital clefts of the lip and palate, and none in which successful results give greater satisfaction.'⁹⁶ He acknowledged that surgeons shoulder a tremendous responsibility for treating these children, although to achieve results of the highest standard requires the collective effort of every member of the multidisciplinary team and a sound relationship with the child and their family.

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CHAPTER 18

Cleft palate and velopharyngeal dysfunction

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Embryology

The primary and secondary palates arise from the frontonasal process and palatal processes of the maxilla, respectively, separated by the incisive foramen. From the incisive foramen, fusion of these processes occurs, progressing anteriorly during weeks 5–6 for the primary palate and posteriorly in weeks 7–8 for the secondary palate. Aberration of this process results in clefts between these embryologically distinct regions. More specifically, a cleft of the primary palate occurs between the premaxilla and ipsilateral palatal process of the maxilla, whereas a cleft of the secondary palate occurs between the two palatal shelves, with each shelf united to the premaxilla.

Cleft palate can occur in isolation or in combination with cleft lip, unilaterally or bilaterally. A cleft of the lip involving the primary palate terminates at the incisive foramen, in keeping with the embryologic origin of these structures as previously described. When a cleft of the lip and primary palate occurs in combination with a cleft of the secondary palate, the latter two malformations may be in continuity, resulting in the classic unilateral or bilateral cleft lip and palate, or discontinuous (for example, cleft lip and primary palate with cleft of the soft palate or incomplete cleft lip and complete cleft of the secondary palate) (Figure 18.1). An isolated cleft palate is subclassified as complete or incomplete, with the latter involving the soft palate with or without a portion of the posterior hard palate. In isolated cleft palate, the vomer is usually midline, vertically oriented and attached to the palatal shelves as far back as the anterior extent of the cleft. The vomer also maintains a vertical orientation in complete bilateral cleft lip and palate; however, may be laterally deviated contingent on the orientation of the premaxilla, from which the vomer projects. This is in contradistinction to the morphology of the vomer in unilateral cleft lip and palate, where the cranial portion of the vomer is almost vertical with angulation (often at 90°) of the caudal segment towards the

palatal shelf to which it is attached on the non-cleft side. The point of attachment between vomer and palatal shelf can be discerned by the colour variance of nasal and oral mucosa, respectively.

Isolated cleft palate is epidemiologically and aetiologically distinct from cleft lip and palate. The incidence of cleft lip and palate varies by race, with approximately 2 in 1000 in East Asian populations, 1 in 1000 in white populations and 0.4 in 1000 in black populations. Moreover, males are twice as likely to be affected than females. In contrast, isolated cleft palate occurs in 1 in 500 live births across all ethnic backgrounds and more commonly in females, with a 2:1 predominance over males. Of all orofacial clefts, approximately 20% are isolated cleft lips, 35% isolated cleft palates, and 45% are clefts involving lip and palate. The aetiology of clefting is multifactorial, including genetic factors, environmental factors and gene–environment interactions. Environmental risk factors include maternal obesity, diabetes mellitus, exposure to alcohol, smoke, diet, viral infections, drugs and various teratogenic agents, as well as physical and/or emotional stress. A strong association between the *interferon regulatory factor 6* (*IRF6*) gene and non-syndromic cleft lip and palate has been demonstrated. It has been reported that aberrations of *IRF6* comprise 12% of the genetic contribution to cleft lip and palate and increase the risk of recurrence three-fold in families with one affected child.¹ The familial association for cleft palate is commonly cited as follows:²

- If normal parents have a child with cleft palate, the frequency of cleft palate in the next child is estimated at 2%.
- If normal parents with a family history of cleft palate have a child with cleft palate, the risk for the next child is 7%.
- If normal parents have two children with cleft palate, the risk for the next child is 1%.
- If one parent has a cleft palate and no previously affected children, the risk for the next child is approximately 6%.
- If one parent has a cleft palate and a child with cleft palate, the risk for the next child is 15%.



(a)



(b)



(c)



(d)



(e)

Figure 18.1 Clinical images of permutations of the clefting process, given the distinct embryologic the origin of lip and primary palate and of the secondary palate. (a) Complete bilateral cleft lip and primary palate. (b) Complete/incomplete bilateral cleft lip and primary palate and complete cleft of secondary palate. (c) Complete bilateral cleft lip, primary and secondary palates. (d) Incomplete unilateral cleft lip and primary palate. (e) Incomplete cleft of secondary palate.

Anatomy

The hard palate is composed of palatine processes of the maxilla and the palatine bone. The palate is supplied by the greater palatine vessels and nerves, which emerge through the greater palatine foramina in the postero-lateral regions of the hard palate. The nasal mucosa of the hard palate is in continuity with the mucosa of the lateral nasal walls, and medially with the vomerine mucosa. The soft palate is the mobile palatal segment, with muscles that act synergistically to effect velopharyngeal closure. The sensory supply of the soft palate is derived primarily from the lesser palatine nerves, which are comprised of several branches that extend from the lesser palatine foramina in the palatal processes of the sphenoid through the palatal aponeurosis.

From a functional perspective, the *levator veli palatini* is considered the most important muscle of the soft palate. This muscle originates from the petrous temporal bone and the junction of bony and cartilaginous portions of the Eustachian tube. It then courses inferiorly, anteriorly and medially to enter the velum and join the levator from the opposite side, forming the *levator sling*. The levator is found in the middle third (40%) of the velum,³ between the two heads of the palatopharyngeus⁴

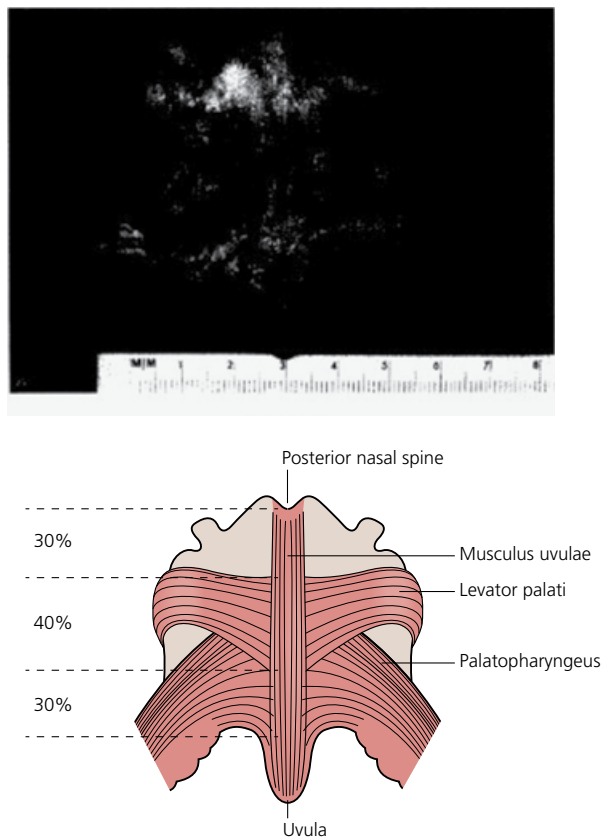


Figure 18.2 A nasal view of dissected soft palate musculature, demonstrating the position of the levator veli palatini, which occupies the intermediate 40% of the soft palatal length. Source: Boorman & Sommerlad, 1985.³ Reproduced with permission of Elsevier.

(Figure 18.2). The levator is innervated by the pharyngeal plexus (motor branches of the ninth, tenth and eleventh cranial nerves). Moreover, it has been suggested that the muscle also receives innervation from the seventh cranial nerve. This is supported by findings of hemi-palatal palsy in patients with hemifacial microsomia.⁵ The cumulative action of the levator is to pull the velum superiorly and posteriorly. In the cleft palate, the levator inserts into the cleft margin in the anterior half of the velum and *not directly into the posterior edge of the hard palate*, as is commonly cited.⁶

The palatopharyngeus originates from the palatine aponeurosis, and then divides into vertical and transverse fibres. The vertical fibres, palatopharyngeus, form the posterior faucial (tonsillar) pillars, course in the lateral pharyngeal walls and insert into the greater horns of the thyroid cartilage of the larynx. The transverse component, palatopharyngeus proper, courses posteriorly along the lateral pharyngeal walls and intermingles with the superior pharyngeal constrictor muscle. The palatopharyngeus is also innervated by the pharyngeal plexus. Although most of the literature supports the notion that the palatopharyngeus is more active during swallowing than speech, the palatopharyngeus fibres are in an anatomical position to act as an antagonist to the elevating action of the levator muscle but as a synergist in stretching the velum backwards. The palatopharyngeus proper assists in the medial displacement of the lateral pharyngeal walls during velopharyngeal closure and contributes to the formation of *Passavant's ridge*. Further, muscular contraction of the palatopharyngeus stretches the velum inferiorly and laterally to increase the available velar surface area and optimize velar shape for contact with the posterior pharyngeal wall. In the midline of the cleft palate, the levator and palatopharyngeus fuse to form the 'cleft muscle' as described by Veau.⁷ However, in contradistinction to the levator veli palatini, the palatopharyngeus also inserts into the posterior border of the hard palate.

The palatoglossus is the most superficial (oral) muscle of the soft palate, traverses through the anterior tonsillar pillars and inserts into the tongue. As such, contraction results in tongue elevation, palatal depression and narrowing of the pharyngo-oral isthmus. The muscle does not have an ascribed role in velopharyngeal closure per se. The levator, palatopharyngeus and palatoglossus diverge anteriorly to insert into the margins of the cleft, aberrant palatal aponeurosis, as well as the oral and nasal mucosa.

The superior pharyngeal constrictor is a quadrangular muscle, which originates from the medial pterygoid plates and pterygomandibular raphe. The muscle courses around the pharynx, forming the lateral and posterior pharyngeal walls, enclosing the nasopharynx and upper oropharynx. The muscle fibres diverge superiorly and inferiorly as they approach the posterior midline to insert into the pharyngeal ligament. The space between the superior margin of the muscle and cranial base is occupied in the lateral nasopharyngeal wall by levator and Eustachian tube and in the posterior pharyngeal wall by the pharyngobasilar fascia. The muscle, innervated by the tenth cranial nerve, causes medial excursion of the lateral pharyngeal walls and, along with palatopharyngeus, anterior displacement of the posterior pharyngeal wall. The latter accounts

for the formation of *Passavant's ridge*. Although historically considered a compensatory mechanism in patients with velopharyngeal insufficiency (VPI),⁸ much evidence exists to challenge this assertion.⁹ For instance, the ridge is often noted to be below and slower than palatal excursion.

The tensor veli palatini does not contribute to movement of the velum. The tensor originates from the scaphoid fossa of the greater wing of the sphenoid and membranous portion of the Eustachian tube. The muscle courses medially and inferiorly parallel to the levator and forms a tendon. The tendon hooks around the hamulus of the pterygoid plate (to which it is partially attached), assumes a horizontal orientation and inserts along the posterior border of the hard palate as the palatal aponeurosis, occupying the anterior third of the velum. In the cleft palate, the tensor palatini reaches the hamulus and appears to diverge into two components – a triangular tendinous portion, which inserts into the lateral posterior edge of the hard palate (does not form the palatal aponeurosis), in close proximity to the nasal mucosa, and a less substantial component, which passes towards the oral surface behind the greater palatine neurovascular bundle. Some authorities recognize a separate but closely related muscle, the dilator tubae, which rounds the middle third of the pterygoid hamulus without an insertion. There has been much debate as to whether the tensor (and/or dilator tubae), have a significant action on the velum and regarding the relative importance of the tensor (and/or dilator tubae) and levator in Eustachian tube function.

The musculus uvulae, thought to be supplied by the lesser palatine nerve, is a paired muscle located above the levator, attached to the palatal aponeurosis anteriorly and passing into the uvula posteriorly. The muscle is covered nasally and laterally by mucous glands. Contraction of the muscle may assist in velopharyngeal closure by adding to the bulk of the midline dorsal ridge over the levator eminence and also possibly by lifting the free border of the palate into deeper contact with the posterior

pharyngeal wall.¹⁰ Others argue that this muscle may not be of importance in velopharyngeal function.¹¹ In the cleft palate, the presence of the musculus uvulae is controversial and rarely seen.

Finally, the salpingopharyngeus is a small muscle that originates from the torus tubarius and courses vertically along the lateral pharyngeal walls. Although this muscle does not contribute to velopharyngeal closure, it may draw the lateral pharyngeal walls superiorly.

Submucous cleft palate

Submucous cleft palate is a form of cleft palate wherein the mucosa is intact but the underlying musculature is similarly abnormal to that of an overt cleft palate. In 1954, Calnan¹² qualified the overt submucous cleft palate with a *classic triad*, including bifid uvula, palpable notch in the posterior hard palate and zona pellucidum. The zona pellucidum is a midline translucent zone that represents diastasis of the velar musculature (Figure 18.3). Kaplan¹³ described the *occult* submucous cleft palate, where velar malfunction is observed, but the aforementioned or typical features are not apparent. Given evidence of velopharyngeal dysfunction (VPD), the diagnosis of occult submucous cleft palate is often made following evaluation with nasopharyngoscopy, where a midline groove or concavity is seen on the nasal surface of the palate. The latter is thought to represent hypoplasia or agenesis of the musculus uvulae, as well as velar musculature diastasis.¹⁴ Sommerlad *et al.*¹⁵ argued that there is a spectrum of severity in submucous clefting and described a scoring system based on the severity of each feature of the triad. In effect, Kaplan¹³ postulated that isolated clefts of the secondary palate, overt and occult submucous cleft palates are variations in expression of the same embryonic process. Diagnosis of submucous cleft palate is often delayed until speech development allows appreciation of VPD.



(a)



(b)

Figure 18.3 Clinical images of an overt submucous cleft palate, demonstrating the zona pellucidum and bifid uvula.

Associated syndromes

Although a syndrome occurs in approximately 10% of patients with cleft palate in association with cleft lip, associated syndromes are described in up to 50% of cases with isolated cleft palate.

Pierre Robin sequence

The most commonly associated anomaly is Pierre Robin sequence, which occurs in approximately 1 in 8500–14000 births. The condition typically occurs sporadically, but may be familial, with autosomal dominant mode of inheritance. The sequence includes micrognathia/retrognathia, glossoptosis and respiratory distress.¹⁶ Although not a qualification of the sequence, a cleft palate occurs in approximately 60–90% of cases. Indeed, in his original description of 1923, Pierre Robin, the French stomatologist credited for the identification of these

infants, failed to include cleft palate as an associated feature, but later added cleft palate as an aggravating factor.¹⁷ The cleft palate in the context of Robin sequence is typically U-shaped, as opposed to the classic V-shaped cleft palate, and implies a wider palatal gap. The theory for the occurrence of cleft palate in Robin sequence is that the posterior displacement of the mandible results in elevation and dorsiflexion of the tongue at approximately 9 weeks of gestation, preventing migration and fusion of the palatal shelves. The majority of cases (83%) present as a component of a named syndrome, including Stickler syndrome, velocardiofacial syndrome, Treacher Collins syndrome, fetal alcohol syndrome, Nager syndrome, oculo-auriculo-vertebral spectrum, Apert syndrome, ectrodactyly-ectodermal dysplasia clefting syndrome, Opitz-G/BBB syndrome and orofacial digital syndrome. Thus, with the Robin sequence, the presence of other anomalous features should be considered (Figure 18.4).

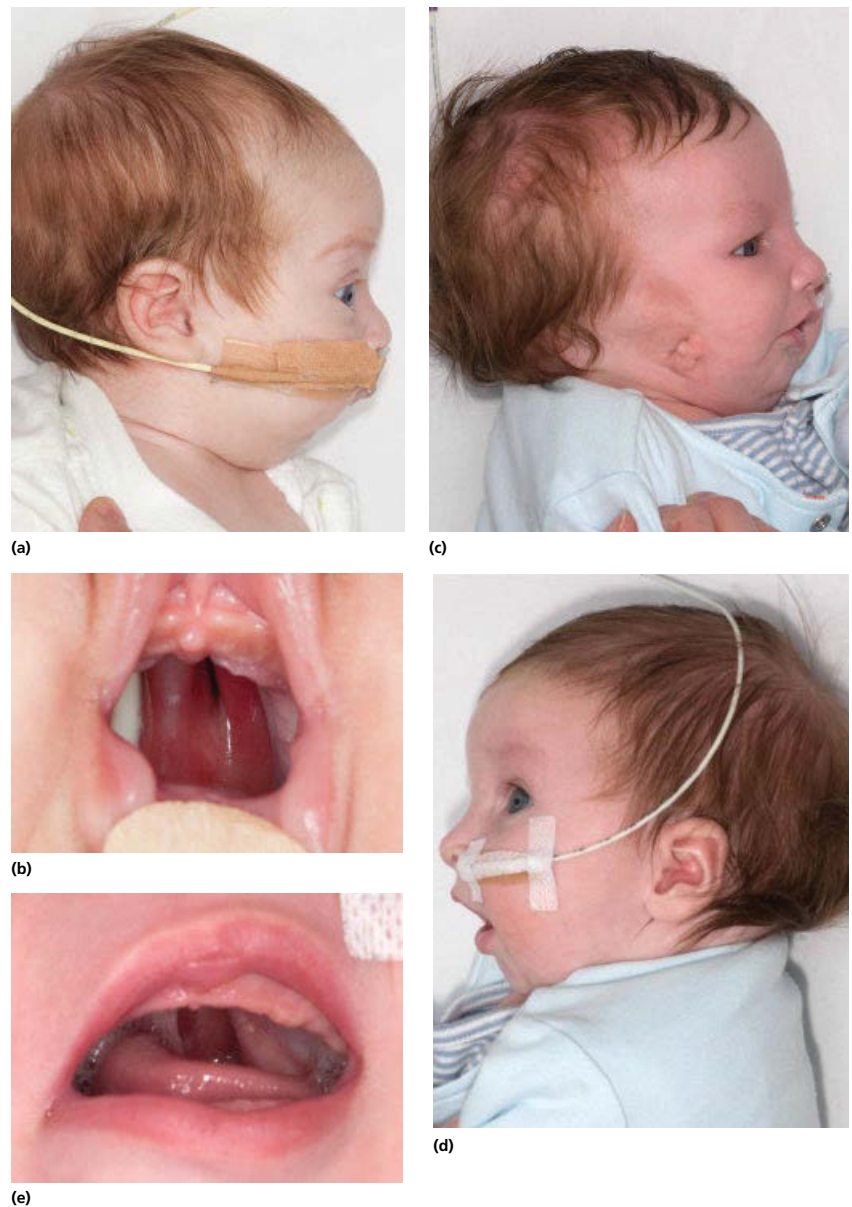


Figure 18.4 Lateral photographs of infants with Pierre Robin sequence. (a) Non-syndromic infant with severe micrognathia, nasopharyngeal airway and gavage tube in place. (b) Typical U-shaped cleft palate. (c, d) Infant with Robin sequence and hemifacial microsomia. (e) In this case, there is a cleft of the soft palate only.

The glossoptosis in Robin sequence initiates a sequence of events including airway obstruction, increased energy expenditure and impaired caloric intake from poor feeding. Thus, in the neonatal period, the primary objectives are to relieve airway obstruction and avert failure to thrive. However, glossoptosis is not the only contributor to upper airway obstruction in children presenting with Robin sequence. Acute angulation of the basicranium, pharyngeal, laryngeal and/or lingual anomalies, subglottic anomalies, hypotonia and CNS disorders may all cause or contribute to airway obstruction.¹⁸ Thus, microlaryngobronchoscopy is essential to determine the mechanism(s) of airway collapse and if obstruction can be relieved completely with treatment strategies that address tongue positioning alone.

In effect, Sher *et al.*,¹⁹ based on endoscopic observations, demonstrated four patterns of pharyngeal collapse in patients with craniofacial anomalies, any of which can be found in Robin sequence.²⁰ In types 1 and 2, the retro-positioned tongue is the primary aetiological factor responsible for obstruction, whereas in types 3 and 4 airway compromise is secondary to lateral pharyngeal wall collapse and pharyngeal stenosis, respectively. Thus, no single treatment will resolve respiratory obstruction in all patients, and management must be tailored to the specific underlying aetiology. Airway compromise is assessed with oxygen saturation recordings and polysomnographic studies, the need for oxygen supplementation and appropriate weight gain (with or without gavage feeding). Routine laboratory investigations and additional studies include serial blood gas analysis for elevated levels of carbon dioxide and serum bicarbonate, and pH recording for gastroesophageal reflux. As primary measures, management of airway compromise secondary to *glossoptosis* include prone or semi-prone positioning and placement of a nasopharyngeal airway with or without continuous positive airway pressure. If the latter conservative treatment modalities fail to relieve airway

obstruction, surgical intervention is warranted. Douglas²¹ popularized the technique of glossopexy, originally described by Shukowsky in 1911.²² This procedure was later modified by Routledge²³ and various modifications were subsequently developed. The latter concept is based on the premise that airway obstruction is secondary to glossoptosis and surgical adhesion between tongue and lip can effectively mitigate the retro-displacement of the tongue. In 1983 a French stomatologist, Viviane Epou, ²⁴ asserted that the imbalance of the muscular insertion of the tongue on the mandible was responsible for the micrognathia, tongue elevation and glossoptosis. More specifically, that the musculature of the floor of the mouth is under increased tension and impels the tongue superiorly and posteriorly, resulting in respiratory obstruction. The latter is the premise for the subperiosteal release of the floor of the mouth musculature for refractory cases of respiratory obstruction in the Robin anomalad.^{25,26} Following release, the tongue can adopt a more anterior position. Mandibular distraction is now a well recognized and widely used surgical alternative in patients with obstruction refractory to conservative measures. With this method the tongue is indirectly pulled forward through muscular attachment to the lingual surface of the mandible.²⁷ In instances where the latter measures fail or in cases where other airway anomalies contribute to obstruction, tracheostomy is often required.

Stickler syndrome

Stickler syndrome is a hereditary progressive arthropathopathy with a estimated frequency of 1 in 10000. It is an autosomal dominant connective tissue dysplasia characterized by cleft palate, degenerative joint changes, ocular manifestations and progressive sensorineural hearing loss. Moreover, these patients often exhibit a characteristic flat facial appearance, secondary to hypoplasia of the mid-face and bridge of the nose (Figure 18.5).



Figure 18.5 Clinical images of infant with Stickler syndrome. Note the facial features characteristic of this syndrome, including prominent eyes, flat nasal bridge, anteverted nares and mid-face hypoplasia.

van der Woude syndrome

Although rare (1 in 100 000), van der Woude syndrome is an autosomal dominant syndrome with variable expressivity also associated with cleft palate. Van der Woude syndrome is associated with *IRF6* gene mutations on chromosome 1q. Moreover, it is unique in that it may also be expressed as either cleft lip with or without cleft palate. A distinguishing feature of the syndrome is bilateral para-median lower lip pits. *IRF6* mutations are also responsible for the popliteal pterygium syndrome, which is characterized by cleft lip and/or palate, lower lip pits and additionally qualified by the presence of bilateral popliteal webs, hypodontia, syndactyly, nail abnormalities, anomalies of the genitourinary system and ankyloblepharon. Interestingly, as previously mentioned, the *IRF6* gene has been recognized to have the strongest genetic association with non-syndromic cleft lip and palate. Indeed, some patients with van der Woude syndrome do not display the typical lower lip pits, and may be erroneously classified as non-syndromic. Thus, *IRF6* gene mutations should be considered in those with affected parent-child and especially segregating cleft palate and cleft lip and palate.²⁸

22q11.2 deletion (velocardiofacial) syndrome

Although clinically underrecognized, velocardiofacial syndrome (VCFS) is the most common multiple anomaly syndromes in humans, with an estimated frequency of 1 in 2000–4000 live births and equal sex distribution. Microdeletions occur in the q11.2 region of chromosome 22, identified by fluorescence *in situ* hybridization (FISH) test. In these patients, the *TBX1* gene is always deleted, with 90% of patients having a deletion of the typical deleted region (TDR; common 22q11.2 deletion, size 3 Mb), while 10% have a smaller deletion of only the proximal 1.5 Mb of the TDR (proximal deleted region, PDR). Both TDR and PDR include the *TBX1* gene area. A few individuals with VCFS have shown unique 22q11.2 deletion intervals or a chromosome

10p14 deletion. In the majority of patients (>90%) 22q11.2 deletions occur as spontaneous events, with both parents unaffected. However, in approximately 10% of individuals, deletion is identified in a parent and thus there is autosomal dominant transmission with variable expression.

There is phenotypic overlap between VCFS and DiGeorge, Sedlacková, conotruncal anomaly face and Cayler syndromes. Indeed, all these aforementioned syndromes are descriptions of the same entity, with over 180 separate anomalies or developmental sequences reported in association with VCFS covering nearly every organ and system. Notable among these are immune deficiency, hypocalcaemia, conotruncal cardiac anomalies, short stature, learning difficulties, behavioural problems, psychiatric illnesses and slender hands and digits. These patients also have a characteristic facial appearance, due to a combination of skeletal and soft tissue features. These include microcephaly, a long middle third of the face, flat zygomatic arches, mandibular retrognathia, broad nasal dorsum, narrow alar bases and bulbous nasal tip, hypotonic flaccid facies, narrow, down-slanting palpebral fissures, and external ear anomalies (Figure 18.6). Also reported in patients with VCFS are aberrant carotid and subclavian arteries. Indeed, it has been hypothesized that the large number of vascular anomalies in major vessels reported in patients with VCFS may result in a number of developmental sequences. The latter notion is supported by reports of Robin, DiGeorge, Potter and holoprosencephaly sequences in individuals with VCFS. The phenotypic expression in this syndrome is markedly variable with no clinical feature occurring in all cases and no reported case with all or even most of the associated clinical findings.²⁹

VCFS is now recognized as the most common syndrome associated with cleft palate and velopharyngeal insufficiency. The majority (approximately 75%) of children with VCFS have hypernasal speech and severe language impairment. Moreover,

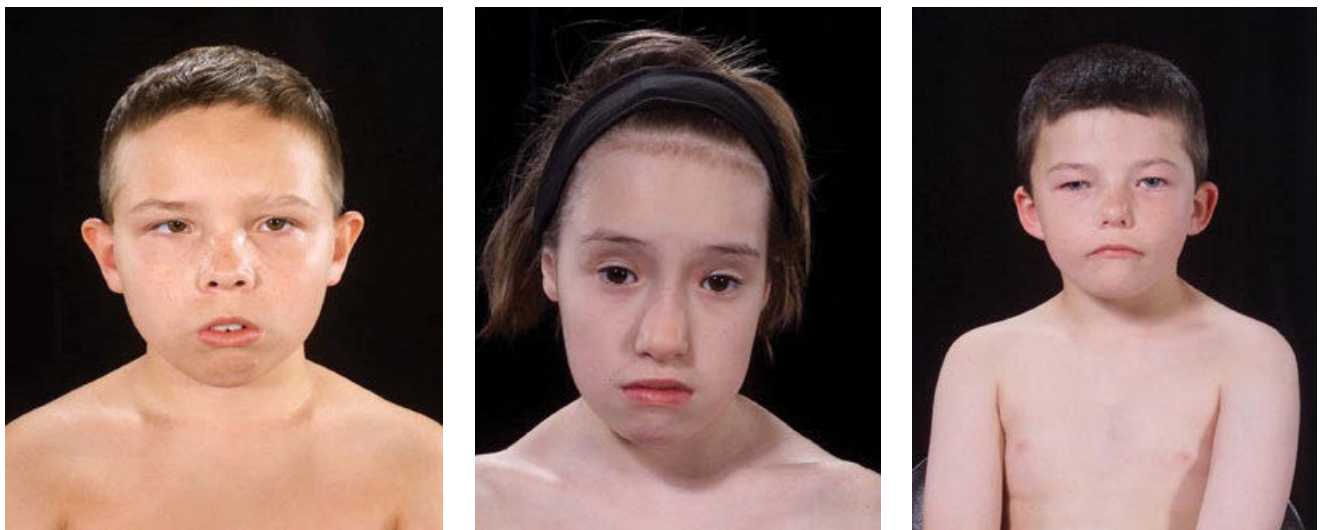


Figure 18.6 Clinical images of children with facies characteristic of velocardiofacial syndrome.

affected individuals commonly present with VPI, most often associated with overt or occult submucous cleft palate. While the frequency of VPI among individuals with non-syndromic submucous cleft palate is approximately <9%, VCFS patients show VPI associated with submucous cleft palate in >70% of cases. In addition to overt or occult submucous cleft palate, there are several other factors that contribute to the high frequency of VPI in VCFS, including platybasia, congenitally short velum, palatopharyngeal disproportion, abnormalities of pharyngeal muscles with hypodynamic velopharynx, lymphoid tissue (adenoid) hypoplasia, and tonsillar hypertrophy. The latter anatomic and physiologic anomalies often render management of VPD in patients with VCFS a greater therapeutic challenge than in their non-syndromic counterparts, often necessitating a combination of palatoplasty and pharyngeal surgery. Despite the more extensive surgical correction required, speech outcomes are universally inferior when compared with those for non-syndromic patients.

Palatoplasty

Pathologic sequelae of cleft palate include feeding and nutritional difficulties, serous otitis media (with potential hearing loss) and abnormal speech development. Thus, the goals of cleft palate repair include improved feeding and normal speech, while minimizing the deleterious impact on maxillary growth and dental arch relationship.

In the neonatal period, feeding and adequate weight gain are a priority. Due to reduced ability to suck, and particularly to draw the nipple into the mouth, infants with cleft palate have difficulty feeding, and breastfeeding can only be achieved with considerable effort and modification of technique. In developed countries, most infants are bottle-fed, which allows a steady stream of milk, usually by a modified teat and soft bottle.

With regards to hearing, all patients with cleft palate should undergo a neonatal hearing assessment. Some argue that ventilation tubes should be inserted if there is a persistent conductive hearing loss of >45 dB on distraction testing or >35 dB on pure tone audiometry. However, given there is evidence that correction of the muscle abnormality in the cleft palate improves Eustachian tube function, others contend that insertion of ventilation tubes should be deferred.

Children with clefts generally do not have difficulties with swallowing and aspiration unless there is a coexisting intrinsic neuromuscular or anatomic abnormality of the tongue or pharynx. Evidence of the latter usually warrants further investigation with videofluoroscopy and endoscopy.

Timing of repair

There is little debate that, in most situations, a cleft palate should be repaired. However, consensus is lacking on timing of repair. Restricted maxillary growth secondary to cleft palate repair has prompted some to promote two-stage palatoplasty, wherein the velum is repaired early in order to enable normal speech

acquisition and hard palate closure delayed to facilitate maxillary development.³⁰ Delayed closure of the hard palate may benefit facial growth but most reports suggest detrimental effects on speech.³¹ Irrespective, there is general agreement that soft palate repair should be effected before 12 months of age. The senior author aims to achieve complete palate closure by 6 months of age. There is evidence from a randomized controlled trial that speech outcome is superior following palatoplasty at 6 months as compared to 12 months.³² In this study, children with clefts repaired at 6 months of age did not develop compensatory articulations, while those with palate repair delayed to 12 months of age did display abnormal articulation patterns. Further, no difference in maxillary arch development, soft tissue profile or velopharyngeal dysfunction was noted between the two groups at 4 years from surgery. There is currently an international randomized controlled trial, the Timing of Primary Surgery for Cleft Palate (TOPS trial) comparing palate closure at 6 and 12 months of age.

For submucous cleft palate repair, timing is similar to overt cleft palate repair for infants with obvious evidence of a submucous cleft and persistent feeding difficulties. Where feeding is not a considerable issue, repair is otherwise indicated if an infant is not producing pressure consonants (/d/, /b/) by 15–18 months. In the older child with submucous cleft palate, surgical treatment is *indicated only in the presence of consistent VPI* or persistent hypernasality, as the presence of submucous cleft palate does not ensure VPD.

Preoperative assessment

Appropriate preoperative assessment is mandatory to ensure that there is no history of airway compromise or anaemia and there is adequate weight gain. The main contraindications to palate repair are associated medial co-morbidities, which may present an unacceptable increased anaesthetic risk and sub-optimal weight.

Controversy exists regarding the benefits of palate repair in older children and adults. Arguably, adults with unrepaired clefts have compensatory patterns of articulation that are so well embedded that palate repair is unlikely to make much difference to speech. However, there are reports of some improvement in speech even though normality is not achieved. An international task force has produced a report and recommendations on delayed palate repair.³³

History of palatoplasty

In 1859, Bernhard von Langenbeck described bilateral bi-pedicled mucoperiosteal flaps for palate repair.³⁴ Lateral relaxing incisions are made at the junction of gingival and oral palatal mucoperiosteum lateral to the greater palatine neurovascular bundle, in addition to cleft marginal incisions (Figure 18.7a). Further developments in technique by Victor Veau,³⁵ Thomas Kilner³⁶ and William Wardill³⁷ were introduced to increase palatal length in an effort to aid in velopharyngeal closure. The three- or four-flap Veau–Wardill–Kilner push-back palatoplasty

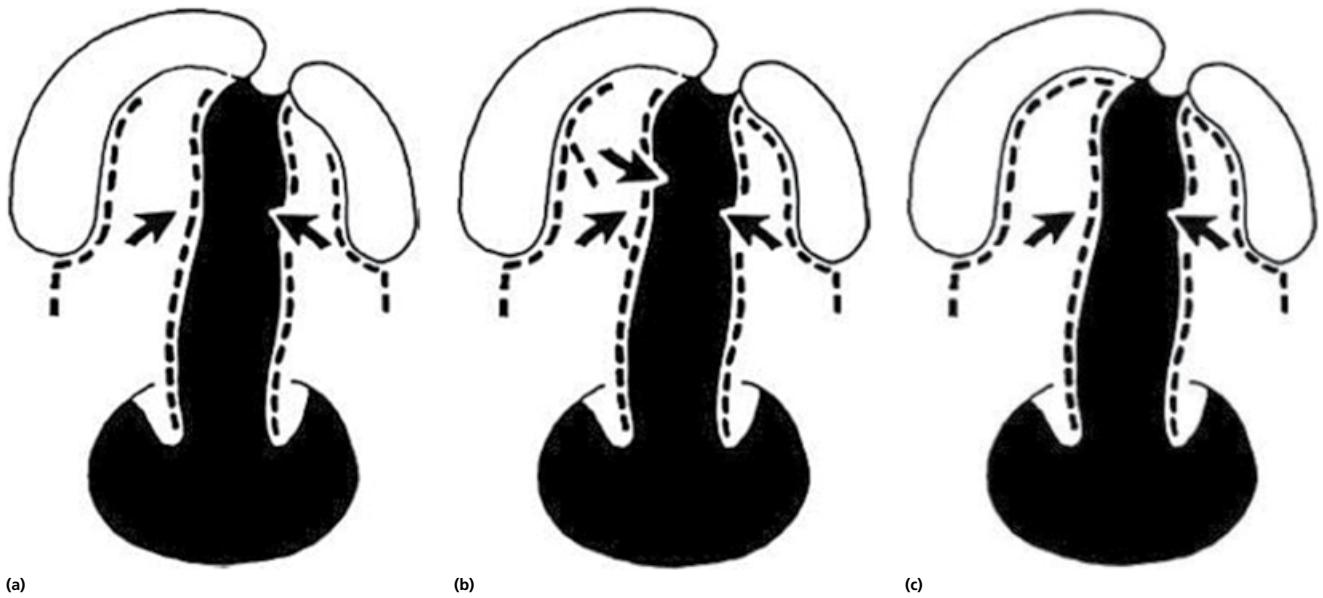


Figure 18.7 Illustration of the (a) Von Langenbeck technique, (b) three-flap Veau-Wardill-Kilner technique, (c) Bardach technique. Dashed lines indicate incisions used. Source: *Comprehensive cleft care*, 2009, Joseph E. Losee & Richard E. Kirschner. Reproduced with permission of McGraw-Hill Medical.

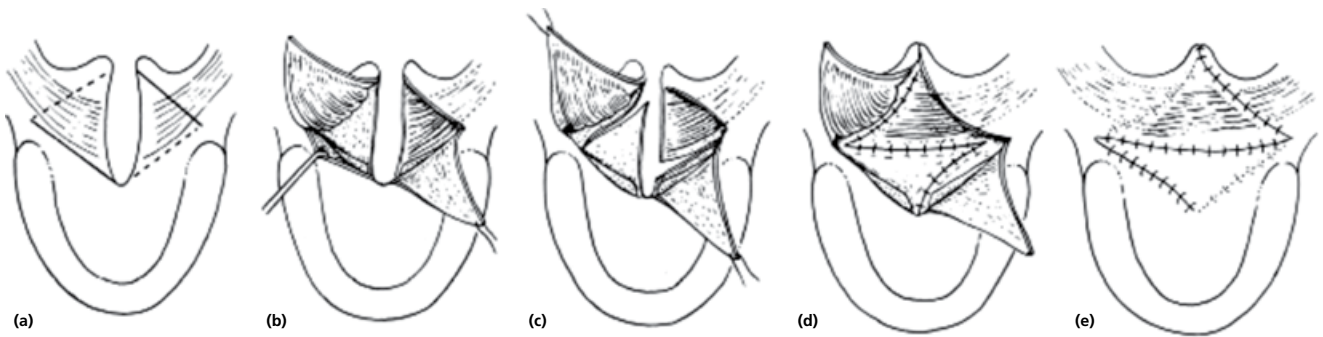


Figure 18.8 Diagrammatic illustration of the key steps in the operative technique for velar closure as originally described by Furlow. Source: Furlow, 1986.⁴²Reproduced with permission of Lippincott, Williams and Wilkins.

includes a V to Y incision and closure of the anterior hard palate (Figure 18.7b). The resulting flaps are based on the greater palatine arteries posteriorly. Inherent in the push-back operation is release of the muscular attachments from the posterior edge of the hard palate. Although the push-back palatoplasty lengthens the palate, theoretically retro-positioning the levator into a more favourable position, this has *not* been shown to be effective in improving speech. Moreover, by virtue of the fact that the bone is denuded anteriorly, this repair adversely affects maxillary growth and the single nasal mucosal layer anteriorly is associated with a higher rate of fistula. In effect, the latter procedure has been largely abandoned in modern era palatoplasty. The two-flap technique popularized by Janusz Bardach³⁸ does not aim to achieve push-back, but is believed by its proponents to improve exposure of the underlying velar musculature (Figure 18.7c). Many surgeons simply return the flaps to their original position to avoid leaving raw areas of bone. Arguably, this may not avoid impairment of maxillary growth.

In the early twentieth century, Victor Veau⁷ first described the anomalous cleft musculature and promoted the concept of suture approximation of the levator in a side-to-side fashion without any endeavour to address the aberrantly positioned muscle. Braithwaite³⁹ and Kriens⁴⁰ emphasized restoration of the *levator sling* by dissection of the abnormally positioned levator muscles prior to re-approximation. Edgerton and Dellon⁴¹ suggested that following dissection, the levator bundles should be rolled 180° and sutured to the musculus uvulae near the uvular base. The double opposing Z-plasty technique, described in 1978 and later published in 1986 by Leonard T. Furlow Jr,⁴² is often used for the primary repair of isolated clefts of the soft palate or submucous cleft palate, or as a secondary technique to lengthen the previously repaired palate. With this technique, 60° Z-plasties are outlined on both oral and nasal mucosa in opposite directions with the central limb placed along the cleft margin. The posteriorly based flap on each surface is composed of muscle (levator) and mucosa, and the anterior flap of mucosa

only (Figure 18.8). Possible advantages of this procedure include lengthening, increased vascularization of the muscle and technical facility. Disadvantages are lateral tightening by Z-plasty closure, less anatomic reconstruction of the musculature, asymmetric velar excursion and difficult closure with wide clefts. Cutting⁴³ and Sommerlad⁴⁴ advocated more extensive dissection of the levator muscles and radical retro-positioning of the velar musculature in their respective techniques of intravelar veloplasty. In an effort to facilitate closure, von Langenbeck incisions are often combined with Furlow's palatoplasty or radical intravelar veloplasty for palate repair.

There are several different protocols and techniques for repair, with variable sequences for closure of the lip, alveolus, hard and soft palates. In the senior author's practice, the operating microscope has been used for palate repair since 1991.⁴⁵ In clefts of the lip and palate, since 1993, a single layer vomerine mucoperiosteal flap has been used for closure of the hard palate and nasal floor at the time of lip repair at three months of age. The vomer flap was first introduced by Pichler in 1926⁴⁶ and was originally described as an inferiorly based flap. This flap resulted in significant maxillary retrusion, presumably secondary to injury to the vomerine-premaxillary suture. However, long-term studies of the superiorly based vomerine flap have not demonstrated a similar deleterious effect on maxillary growth, probably due to the relatively more limited dissection.^{47,48} In the case of unilateral cleft lip and palate, an incision is made at the junction of palatal and vomerine mucosa on the non-cleft side, extending posteriorly down the crest of the vomer and anteriorly around the side of the premaxilla to become continuous with the medial lip incision. The oral mucoperiosteum is incised along the margin of the cleft and elevated to allow the vomerine flap to be inserted underneath. In bilateral cleft lip and palate, one vomerine flap is elevated as previously described. As complete elevation of bilateral vomerine flaps may jeopardize the blood supply of the premaxilla and prolabium, the flap on the other side is elevated only to the pre-vomerine suture. In the interim period between cleft lip and anterior palate repair and soft palate repair, the exposed periosteal surface of the vomerine flap epithelializes to create an intact hard palate.

Sommerlad technique for palate repair

A modified mouth gag is inserted followed by a gauze throat pack. The junction of the oral and nasal mucosa is marked with ink (Figure 18.9a). The nasal mucosa is usually on the oral side of the edge of the cleft and darker than the oral mucosa (Figure 18.9b). The palate is infiltrated with lidocaine (lignocaine) with adrenaline (epinephrine) and the palate incised along the margin of the cleft at the junction of oral and nasal mucosa. The incision then extends from the midline at the anterior limit of the cleft anteriorly onto the posterior border of the hard palate to enable subperiosteal elevation of the oral mucoperiosteal flaps to expose the bony palatal shelves (Figure 18.9c). Dimples are frequently seen on the oral side of the palate at a

point where oral mucous glands are attached to the posterior border of the hard palate and tendinous nasal component of tensor palatini. Care must be taken to elevate these mucous glands intact with the oral flap (Figure 18.9d). Dissection is facilitated with the use of a dental scaler or Freer's elevator.

In wide clefts, where more extensive mobilization of the oral layer is necessary, dissection extends laterally to free the greater palatine neurovascular bundle, with incision of its surrounding periosteal sleeve when necessary. Elevation of the oral layer is continued posteriorly into the soft palate and laterally to the pterygoid hamulus, separating the oral mucosa and attached mucous glands from the underlying musculature, the palatopharyngeus and palatoglossus. The nasal mucosa is mobilized from the nasal surface of the palatal shelves, if necessary, and sutured in the midline. For soft palate closure in patients with unilateral or bilateral cleft lip and palate, in whom a vomerine flap was used for closure of the hard palate at the time of unilateral or bilateral lip repair, a posteriorly based flap of the neomucosa from the epithelialized vomer may be raised and turned back to facilitate closure of the anterior nasal layer of the soft palate (Figure 18.9e,f). Following closure of the nasal layer, the anteriorly oriented muscle fibres are seen inserting into the posterior border of the hard palate (Figure 18.9g). For muscle dissection, an incision is started posteriorly approximately 4 mm from the midline and is extended anteriorly closer to the midline, just lateral to the nasal layer sutures, leaving the mucous glands in place centrally. The palatopharyngeus medially and the nasal component of the tensor tendon laterally are divided from the back of the hard palate and dissection proceeds in a plane between nasal mucosa and musculature. Vessels are seen on the nasal mucosa and are preserved where possible. The tensor tendon is then divided in an anteroposterior direction medial to the hamulus. The oral palatoglossus and palatopharyngeus fibres are then split to expose the levator, which is identified as a discrete cylindrical muscle within a defined fascial envelope (Figure 18.9h), and is paler than the other palatal muscles.

Dissection is continued until the muscle can be transposed to the posterior half of the velum. A stab incision is made in the nasal mucosa on each side, if a hole was not inadvertently created during muscle dissection. The latter permits draining in an effort to prevent haematoma formation. The levators are then united along with retro-displaced palatopharyngeus and tensor tendon, which imparts strength to the repair (Figure 18.9i). Finally, the oral layer is closed (Figure 18.9j,k). First, one or two sutures are placed in the midline anterior to the muscle repair between oral mucosa and the nasal mucous glands. The latter occludes the dead space and assists in maintaining the muscle retro-positioned. The oral layer is closed with a twisted loop mattress stitch.⁴⁹

For repair of submucous cleft palate, a similar technique is used as described above, leaving the nasal layer intact and leaving, if possible, some mucous glands on the nasal layer in the midline.

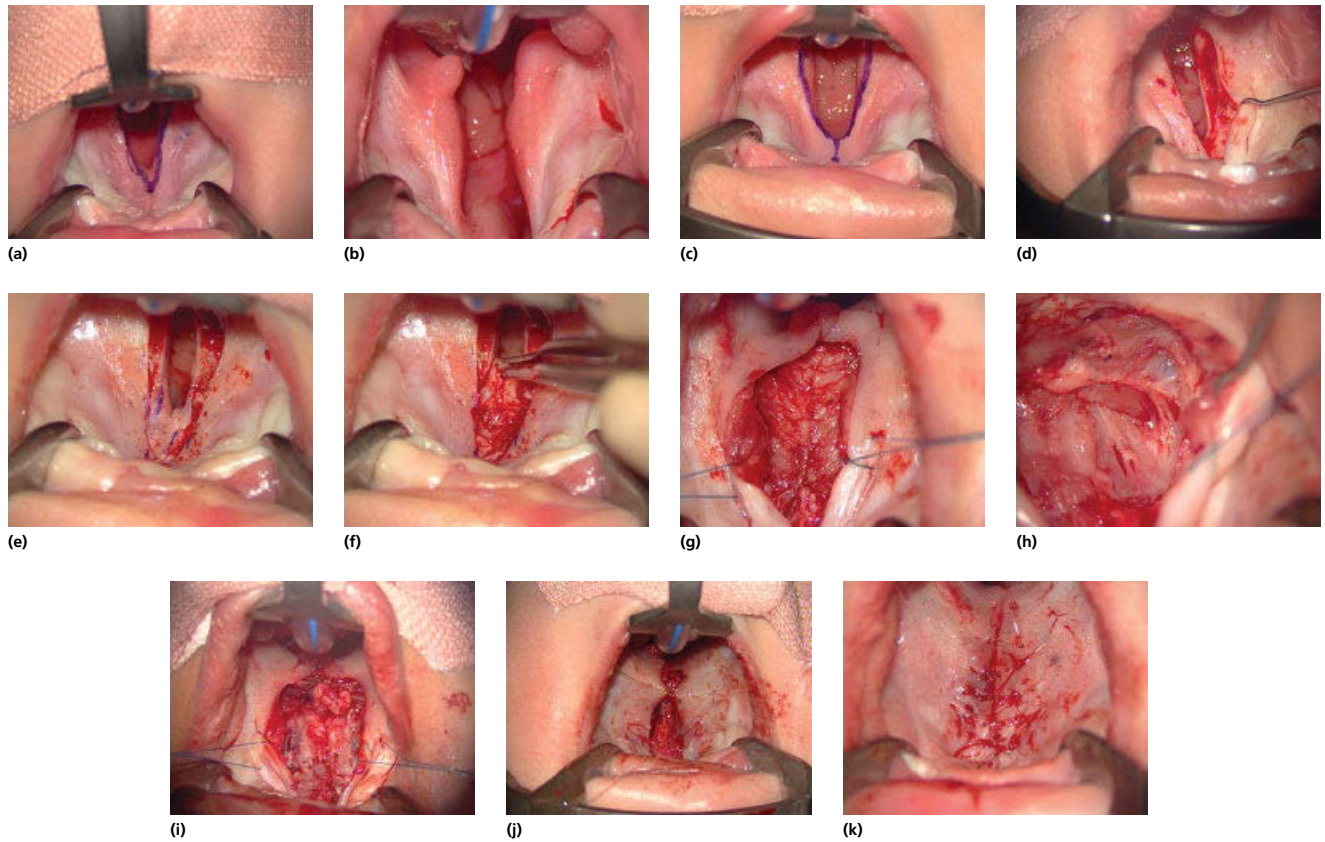


Figure 18.9 Clinical images of the key steps in the operative technique for velar closure by Sommerlad. (a) Marking of the incision at the junction of oral and nasal mucosa along cleft margin. (b) The junction of nasal and oral mucosa is distinguished by darker coloration of nasal mucosa. (c) Extension of the incision to allow elevation of oral mucoperiosteal flaps. (d) Sub-periosteal elevation of the oral mucoperiosteal flaps towards the posterior border of the hard palate. (e,f) Use of neo-mucosal vomerine flap for closure of the nasal layer of the soft palate. (g) Anteriorly oriented palatopharyngeus (and palatoglossus) fibres inserting into the posterior border of the hard palate. (h) Exposure and transposition of the levator muscle. (i) The levators united along with retro-displaced palatopharyngeus and tensor tendon. (j,k) Closure of the oral layer with twisted loop mattress sutures. Source: Croft et al., 1981.⁵⁴ Reproduced with permission of Wiley.

The senior author aims to achieve velar closure without lateral releasing incisions (von Langenbeck). The need for lateral releasing incisions depends on the type, extent and width of the cleft. Lateral releasing incisions are most often required in patients with Robin sequence with a wide U-shaped cleft, and anticipated if the width of the cleft at the back of the hard palate is in excess of 10 mm at an age of repair of six months. Hard palate closure without lateral releasing incisions depends on the 'drawbridge effect' (where the sloping high-arched oral mucoperiosteal flaps are brought into greater proximity when mobilized into horizontal position), unfolding of the infolded areas of the flaps and division of the oral component of the tensor palatini insertion and/or the periosteal sleeve of the greater palatine neurovascular bundle.

Repair of submucous cleft palate

In patients with submucous cleft palate, the extent of morphologic abnormality is not in direct proportion to the size of the velopharyngeal gap or severity of hypernasality. As previously mentioned, the latter is secondary to compensatory factors including prominent adenoid pad and/or significant excursion

of the pharyngeal musculature.⁵⁰ The authors' approach to the treatment of VPI in patients with submucous cleft palate is to optimize levator function with a radical dissection and retro-positioning of the abnormally positioned velar muscles. Upon palate exploration, if the levators are deemed to be in a satisfactory location, then management favours a Hynes pharyngoplasty to augment the posterior pharyngeal wall.⁵¹ Following reassessment at six months postoperative follow-up, the necessity for further surgery is determined. The latter may be either a modified Hynes pharyngoplasty or buccinator flap lengthening of the palate.

Complications of palatoplasty

The complications commonly associated with cleft palate repair include acute airway obstruction, bleeding, infection and partial or complete palatal dehiscence. The latter may result in fistula formation. Treatment of problematic fistulas depends on location and size. Associated symptomatology includes nasal regurgitation, as well as hypernasality and other manifestations of VPI. Corrective measures include repair with local mobilization of nasal and oral palatal tissue, with or without placement of

interpositional tissue or material, such as a segment of pedicled buccal fat pad, cartilage graft or acellular dermal matrix, or formal palate re-repair. Other options include regional flaps (buccal mucosal or musculomucosal flaps, facial artery myomucosal flap, tongue flap) and rarely, free tissue transfer. When circumstances preclude surgical correction, an obturator can be utilized. Potential long-term sequelae of palatoplasty include maxillary retrusion and, most notably, VPI.

Velopharyngeal dysfunction

The function of the velopharyngeal mechanism, which consists of the velum, lateral and posterior pharyngeal walls, is to separate the oro- and nasopharynx during speech and deglutition. Normal velopharyngeal closure is necessary for proper consonant production and nasal resonance. In the English language, only three sound /m/, /n/ and /ng/ do not require closure. The primary manifestations of ineffective closure of the velopharyngeal port are excessive nasal resonance or hypernasality and nasal air emission. Hypernasality results from abnormal coupling of the oral and nasal cavities during vowel production and nasal air emission is noted with consonant production.

Some authors have attempted to distinguish between *velopharyngeal insufficiency*, *incompetence* and *dysfunction*. Velopharyngeal *insufficiency* refers to hypernasality and nasal air emission during speech secondary to anatomic aberration of the velopharyngeal sphincter. In contrast, velopharyngeal *incompetence* is employed to describe dysfunction of an anatomically intact velopharyngeal mechanism, usually secondary to neuromuscular discoordination. Finally, velopharyngeal *dysfunction* is a term used to describe all conditions that affect velopharyngeal closure, such that the valve does not achieve consistent and/or complete closure during oral sound production.

Irrespective of the nature of the VPD, in an attempt to overcome the inability to achieve velopharyngeal closure, children develop compensatory patterns of articulation (e.g. pharyngeal or nasal fricatives and glottal stops) and other secondary speech patterns, including hoarseness and low speech volume, which can significantly compromise speech intelligibility. Moreover, children may display behavioural idiosyncrasies such as nasal or facial grimace in an attempt to decrease nasal airflow. Causes of VPI include repaired cleft palate, palatal fistula, submucous cleft palate, maxillary advancement, congenital large pharynx or palatopharyngeal disproportion, adenoid regression, irregular adenoid pad and/or adenoidectomy. The influence of the adenoids on velopharyngeal closure, merits more detailed description.⁵² Most children exhibit velo-adenoidal closure where the soft palate abuts the adenoid pad, which is positioned on the posterior pharyngeal wall in the area of usual velar contact. Although adenoid regression does not affect velopharyngeal closure in children with a normal velum, in those with a previously repaired cleft palate, overt or occult submucous cleft palate, evidence or deterioration of existent VPI may be noted. In like manner,

hypernasality or nasal air emission may occur following adenoidectomy in patients with no velar defect, but does not typically persist for more than six weeks postoperatively. Enduring VPI has been reported to occur in approximately 1 in 1500, more likely in patients with overt or occult submucous cleft palate, where removal of the space-occupying adenoids unmasks underlying palatal deficiency.

Finally, in some patients, the shape of the adenoid tissue may in fact be detrimental to closure, wherein irregular surface morphology prevents a tight velopharyngeal seal. Velopharyngeal incompetence can occur in patients with generalized hypotonia (e.g. VCFS), neurological conditions causing dysarthria (e.g. myotonic dystrophy, cerebral palsy, neurofibromatosis, Steinart's syndrome) or apraxia and cranial nerve defects (e.g. hemifacial microsomia).

Assessment of velopharyngeal function

Selection of the appropriate procedure for correction of VPD is based on speech evaluation by a trained speech and language therapist, direct physical examination of the palate, as well as instrumental assessment. On intra-oral examination, static and dynamic palatal evaluation is assessed. Inspection for features of submucous cleft palate, as well as presence of palatal fistula and tonsillar hypertrophy is completed. Normally during production of 'ah', presence of a simple midline palatal dimple is seen at the junction of the middle and posterior thirds of the soft palate. Presence of two lateral dimples, a single unilateral or complete absence of the dimple, as well as an anteriorly displaced dimple are indicative of abnormal levator muscle insertion and functional compromise.²

Nasoendoscopy allows direct visualization of the velopharyngeal mechanism from both anatomic and functional standpoints. With nasoendoscopy, the contribution of the velum, lateral and posterior pharyngeal walls (i.e. closure pattern) can be assessed, and in the presence of VPD, the size and location of the velopharyngeal gap as well as timing of velopharyngeal closure can be measured. Hypernasality is not necessarily related to the size of the velopharyngeal gap. Patients with a small velopharyngeal gap may present with severe hypernasality due to delayed time of velopharyngeal closure during speech. Velopharyngeal closure patterns, originally described by Skolnick *et al.*⁵³ and Croft *et al.*,⁵⁴ include coronal, sagittal, circular and circular with Passavant's ridge. The most common pattern is coronal, where the velum approximates the posterior pharyngeal wall, while the lateral pharyngeal walls move medially to abut the lateral edges of the velum. In sagittal closure pattern, velopharyngeal competence is primarily a function of the lateral pharyngeal walls, which move medially posterior to the velum with the velum approximating the anterior edge of the abutted lateral walls. Circular closure involves equal contributions from velum and lateral pharyngeal walls, while circular closure with Passavant's ridge also includes forward movement of the posterior pharyngeal wall resulting in a true sphincteric closure pattern (Figure 18.10).

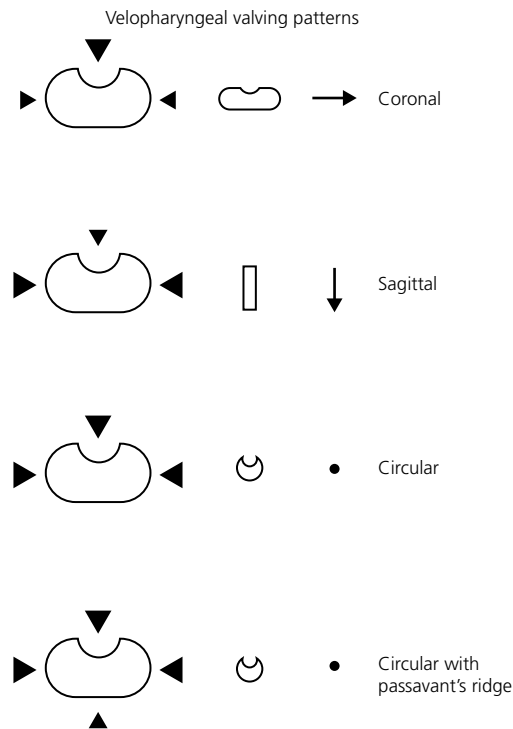


Figure 18.10 Patterns of velopharyngeal valving. Source: Croft *et al.*, 1981.⁵⁴

Although nasoendoscopy is principally utilized as an investigative tool for elucidation of the underlying aetiology of and treatment selection for VPD, some have implemented videonasopharyngoscopy as an instrument for visual biofeedback of the velopharyngeal sphincter during speech.⁵⁵ Compensatory articulations may result in aberrant pharyngeal movements and the latter method has been effectively used to alter velopharyngeal movements or valving pattern prior to surgery, improving prognosis for success. Others contend that correction of misarticulations with speech therapy alone can improve pharyngeal wall motion without the need for visual biofeedback. In general, nasoendoscopy cannot be performed in uncooperative children or those younger than age 4 years.

In contrast, multiview *videofluoroscopy*, with or without barium coating, also widely implemented to assess the above-mentioned parameters, can often be performed in children below 4 years of age. In the authors practice, *lateral view* videofluoroscopy only is performed, as this view is deemed to provide the most anatomic and diagnostic information with regards to *levator* function, and its functional relation to the posterior pharyngeal wall.⁵⁶ Effective velar length refers to the portion of the soft palate that contributes to velopharyngeal closure, located between the posterior edge of the hard palate and *levator knee* or eminence (Figure 18.11). The remaining portion of the velum extends below the contact region and therefore does not participate in closure. On lateral view videofluoroscopy, the effective velar length (*levator knee*), velar excursion and the level of attempted palatal contact (velopharyngeal closure) can be ascertained. Moreover, posterior pharyngeal wall and Passavant's ridge displacement and direction of projection, as well as abnormal speech behaviours such as timing abnormalities and

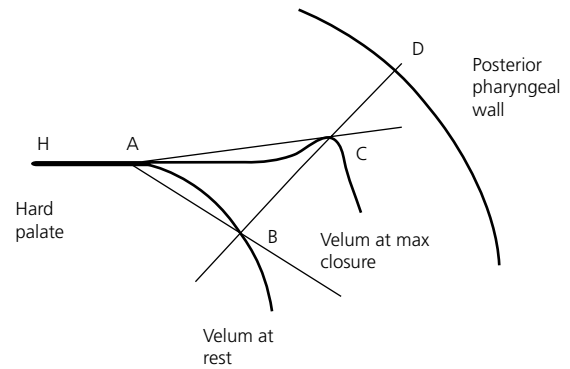


Figure 18.11 Analysis of velar function. Measurements are made with the velum at rest in the nasal breathing position and at the point of maximum closure during production of the sound /i/. Levator function is best assessed during production of the sound /i/. A denotes the posterior edge of hard palate. Point B on the velum forms the *levator knee*. C, velar knee at the point of maximum closure. The levator knee is the bend in the soft palate formed by levator contraction. D, projection of line BC onto the posterior pharyngeal wall, in the line of the levator action, to identify the point on the posterior pharyngeal wall at which closure/contact would occur. AB, velar length at rest; AC, velar length at maximum closure or the functional length of the velum (extended length); AC/AB, extensibility; BD, velopharyngeal gap size at rest; CD, velopharyngeal gap at maximum closure; BC, velar excursion, distance travelled by the velar knee from the resting position to the point of maximum closure; BC/BD, closure ratio (1 = complete closure).

'tongue-backing' (where lingual movement is used to passively elevate the palate) can be seen during speech production.

Both nasoendoscopy and videofluoroscopy also allow visualization of tonsillar and adenoid tissue, which may impede palatal excursion or present a limitation to appropriate placement of pharyngeal flaps, respectively. Of note, in the presence of a palatal fistula, velopharyngeal function should be assessed with the fistula covered, as velopharyngeal movements have been shown to improve or even normalize once an associated fistula is occluded.⁵⁷ A limitation of videofluoroscopy is difficulty distinguishing right and left palatal excursion. However, a 'double levator knee' is usually apparent with asymmetric palatal movement.

A more objective measure of nasal resonance can be obtained with *nasometry*, where two separate measures of sound output from the nasal and oral passages are recorded during speech. A ratio of sound pressure energy generates a nasalance score. A high nasal score reflects a high degree of nasal emission, which is useful in cases where nasal air escape is inaudible in speech evaluation.

Static MRI may offer useful information regarding the position of the palate muscles. Dynamic real-time MRI scanning is a very promising future investigative tool, with the potential of providing three-dimensional visualization of the velopharyngeal port.

Upper airway assessment, including presence or absence of mouth breathing, snoring, obstructive sleep apnoea or nasal airway obstruction, is essential prior to surgical management of VPI. Further, a Mueller test may be performed, in which lateral pharyngeal wall stability on deep inspiration against obstruction (with mouth and nose closed) is also examined. In the presence of symptomatology suggestive of obstructive sleep

apnoea on preoperative evaluation, a polysomnogram is requested. If indicated (i.e. structural impediment to flap harvest and/or inset), tonsillectomy and/or adenoidectomy is performed 3–6 months prior to surgery.

Surgical management of velopharyngeal dysfunction

One of the primary principles of treatment for VPD is that *speech therapy will not correct VPI*. Following restoration of the structural elements to allow for velopharyngeal closure and correct nasal resonance, speech therapy is likely to be required to eliminate compensatory misarticulations and behavioural abnormalities (e.g. nasal grimace), as well as provide auditory reinforcement to promote closure. However, speech therapy can be applied if VPI is *intermittent* and therefore, although inconsistent, sphincteric closure can be achieved. Management of VPD aims to achieve satisfactory nasal resonance without inducing airway or nasal obstruction. Surgical management can be divided into palatal procedures and pharyngeal surgery.

Modifications to the velum include Furlow palatoplasty or palate re-repair with intravelar veloplasty. The former is implemented to lengthen the palate and re-establish the levator sling. Additionally, by performing a Z-plasty, recruitment of lateral pharyngeal tissue also results in tightening of the superior pharyngeal constrictor and consequently the velopharyngeal port.⁵⁸ The premise for palate re-repair with intravelar veloplasty is to retro-position the palatal musculature in order to recruit the residual soft palate for velopharyngeal closure, thereby optimizing the position and function of the levator (i.e. increasing effective velar length).⁵⁹ Moreover, buccinator flaps can be interposed at the junction of hard and soft palates in a short velum with adequate motility and posteriorly positioned levator musculature in an effort to achieve palatal lengthening.⁶⁰ Pharyngeal procedures include pharyngeal flap, pharyngoplasty and pharyngeal wall augmentation with either autologous or allogenic material. Figure 18.12 outlines the senior author's algorithm for the surgical management of VPI.

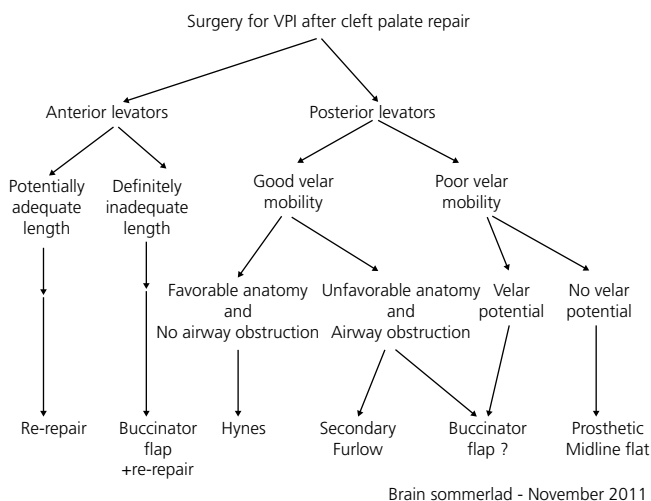


Figure 18.12 Algorithm for the surgical management of velopharyngeal insufficiency.

Pharyngeal flap

In 1865, Passavant described the original procedure for management of VPI in which adhesion between the soft palate and posterior pharyngeal wall was carried out, the forerunner of the pharyngeal flap.⁶¹ Later in 1875, Schoenborn introduced the inferiorly based pharyngeal flap,⁶² which he later modified to a superiorly based flap.⁶³ Rosenthal⁶⁴ reported on the use of a pharyngeal flap in primary cleft palate repair, where a pharyngeal flap was combined with a von Langenbeck palatoplasty. The posterior pharyngeal flap was popularized by Padgett⁶⁵ as a salvage procedure in patients with failed primary cleft palate repair. The pharyngeal flap is designed to create a passive central obturator in the velopharyngeal sphincter, leaving two lateral ports (Figure 18.13). Lateral gutters facilitate nasal breathing, nasal resonance and mucous drainage. Hogan⁶⁶ later refined pharyngeal flap design with the introduction of the concept of lateral port control, in which the size of the lateral ports are carefully calibrated at the time of surgery with the use of catheters placed through the two ports. In 1979, Shprintzen *et al.*⁶⁷ further advanced pharyngeal flap surgery with the advent of 'tailor-made' pharyngeal flaps, wherein the width of the flap is determined by the degree of lateral wall motion as seen on preoperative nasendoscopy and/or videofluoroscopy. Successful management of VPI with a pharyngeal flap necessitates satisfactory mesial movement of the lateral pharyngeal walls for lateral port occlusion,⁶⁸ and is therefore considered in patients with a sagittal closure pattern. If lateral wall motion is insufficient for lateral port closure (e.g. coronal closure pattern) or if the flap is too narrow (poor flap design) or attenuated (postoperative tubular contracture secondary to unlined flap under-surface), hypernasality may persist. In contrast, lateral port occlusion may result in airway compromise, hyponasality and mouth breathing.

Interestingly, Karling *et al.*⁶⁹ noted a correlation between flap width and adduction of the lateral pharyngeal walls. More specifically, patients with limited preoperative adduction demonstrated a greater increase in lateral wall excursion with a narrow flap than with a wide flap, whereas those with greater preoperative excursion showed a decrease in postoperative adduction in relation to flap width.⁷⁰ The latter 'learning phenomenon' provides one possible explanation for those patients that retain a certain degree of velopharyngeal competence following surgical flap division or dehiscence. Other potential reasons for persistent velopharyngeal function following flap resection include augmentation of the posterior pharyngeal wall with the residual flap remaining as a pad or possibly anatomic changes in the pharynx secondary to the initial surgery, such as narrowing of the velopharyngeal port due to scar contracture at the donor site.

The most common complications of pharyngeal flap surgery include bleeding and airway compromise. The most notable complication is the potential for obstructive sleep apnoea. There are a number of other factors that contribute to obstructive sleep apnoea following pharyngeal flap harvest, in addition to the mechanical barrier of the flap per se at the level of the

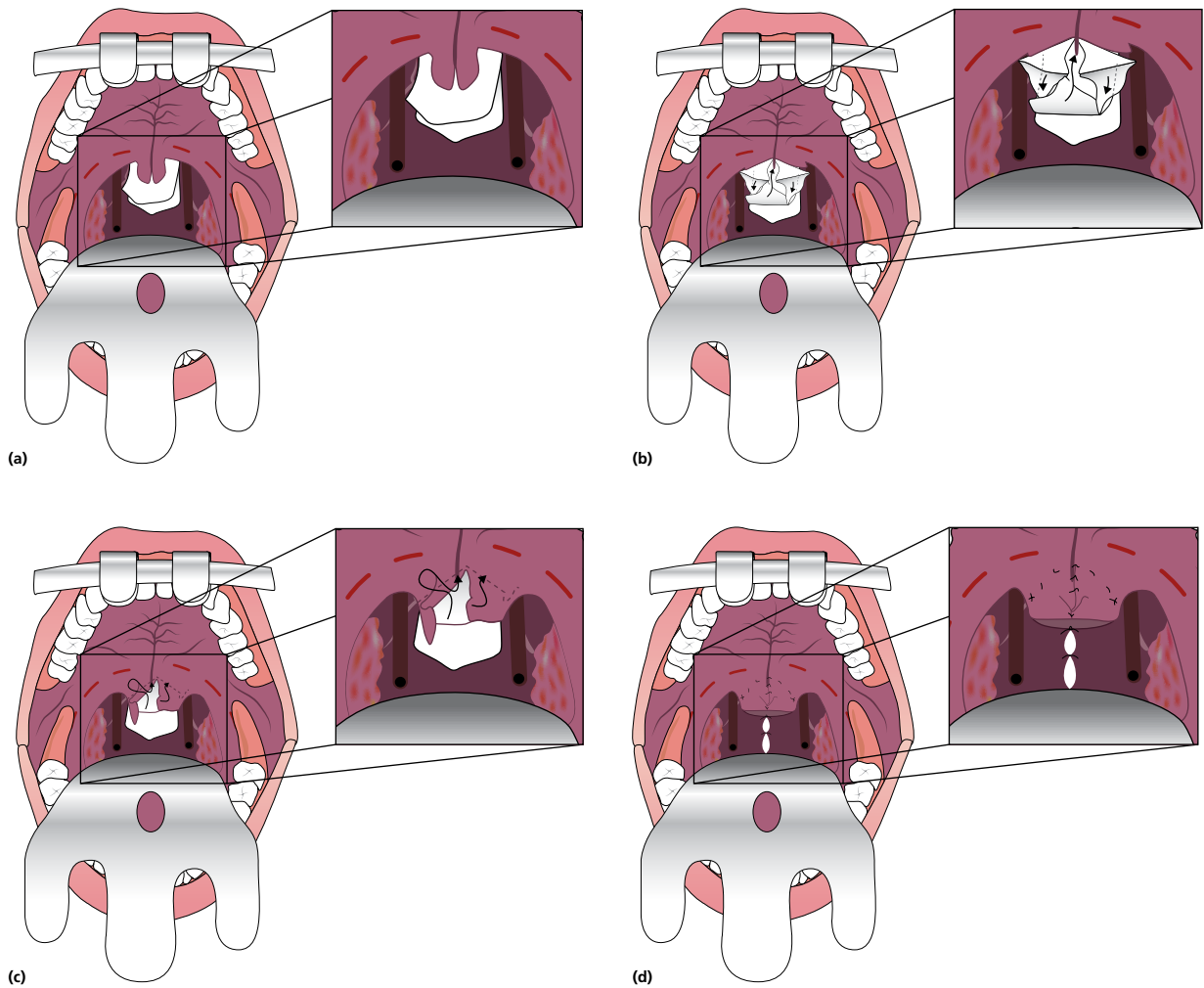


Figure 18.13 Diagrammatic illustration of key steps in the operative technique for superiorly based pharyngeal flap harvest and inset. (a) The superiorly based flap is incised in the posterior pharyngeal wall and the soft palate is divided in the midline. (b) Posteriorly based flaps of nasal lining of the velum are elevated. (c) The pharyngeal flap is inset onto the nasal surface of the soft palate and the previously raised flaps from the nasal surface of the palate are used to line the raw (oral) surface of the pharyngeal flap. (d) The soft palate is closed in the midline, along with the donor defect in the posterior pharyngeal wall.

nasopharynx. Moreover, syndrome-specific features may aggravate any mechanical obstruction. More specifically, obstruction is often secondary to lateral wall collapse against a wide pharyngeal flap. The latter is especially notable in patients with VCFS, in whom generalized muscular hypotonia presents a predisposition for pharyngeal wall collapse and obstruction. In addition, obstruction may occur as a result of flap harvest, which also narrows the lower portions of the pharynx, thus increasing resistance in the oro- and hypopharynx. The latter is secondary to the flap donor site that remains to heal by secondary intention thereby creating circular contraction.

Other factors include posteriorly displaced tonsils that extend within the lateral ports and occlude the nasopharyngeal airway. Tonsillar hypertrophy may also compromise the oropharyngeal airway, as circular contraction brings them in closer proximity. Obstructive sleep apnoea occasionally necessitates division of the flap, when not amenable to non-surgical management.

Caution should be exercised when selecting pharyngeal flap surgery in patients with VCFS, in view of the high prevalence of medial displacement of the internal carotid arteries. This poses significant risks in execution of this procedure given that the abnormal positioning of the vasculature in the pharynx is coincident with the region of pharyngeal flap harvest. Moreover, the danger of ectopic localization of the arteries is aggravated by the characteristically thin pharyngeal musculature. Many favour evaluation of the cervical vasculature with MRI angiography prior to consideration of surgical velopharyngeal correction. However, the results obtained with more sophisticated imaging modalities cannot be extrapolated to the patient at the time of operation, where the internal carotids adopt a less median position with neck hyperextension. Thus, routine vascular imaging, even when aberrant pulsations are noted on preoperative nasoendoscopy, does not appear crucial or reliable. According to Mehendale and Sommerlad,⁷¹ the most reliable measure is direct

observation and palpation of the pharynx at the time of surgery. If necessary, the position and width of the pharyngeal flap can be modified to minimize the risk of artery exposure.

Pharyngoplasty

Hynes⁷² described a technique for pharyngoplasty, where bilateral superiorly based musculomucosal flaps were elevated from the lateral pharyngeal walls and inset onto the posterior pharyngeal wall. Flaps included salpingopharyngeus and were later modified to contain the descending fibres of the palatopharyngeus and a portion of the superior constrictor.^{73,74} The elevated flaps, based at the level of the Eustachian cushions, are transposed 90° and sutured to the posterior pharyngeal wall, which is incised transversely from the superior end of the posterior limb of one lateral flap to the other, at the level of attempted velopharyngeal closure or atlas (first cervical vertebra) (Figure 18.14). The flaps are inset in overlapping fashion.^{73,74} In his original description, Hynes described division of the soft palate in order to gain access to the nasopharynx and facilitate flap inset. Pharyngoplasty

attenuates VPD by diminishing the central velopharyngeal space. This procedure is used in patients with a coronal or circular closure pattern and adequate palatal motion, or hypotonic velopharynx. In effect, these flaps are used to *augment the posterior pharyngeal wall*, thereby reducing the palatal movement necessary to achieve closure.

Orticochea^{75,76} described a pharyngoplasty that is quite distinct from the Hynes pharyngoplasty, despite the two procedures being often confused. In this operation, the flaps are raised from the posterior tonsillar pillars, based superiorly and containing palatopharyngeus muscle. The flaps are transposed and inset into a short but wide inferiorly based posterior pharyngeal flap *below* the level of attempted closure in the oropharynx and below that of the Hynes pharyngoplasty as previously described (Figure 18.15). Jackson and Silverton⁷⁷ modified this procedure by altering the inferiorly based flap to a superiorly based flap in an effort to bring the flaps closer to the level of attempted closure, but later eliminated the supplementary posterior pharyngeal flap. Orticochea proposed the notion of *dynamic*

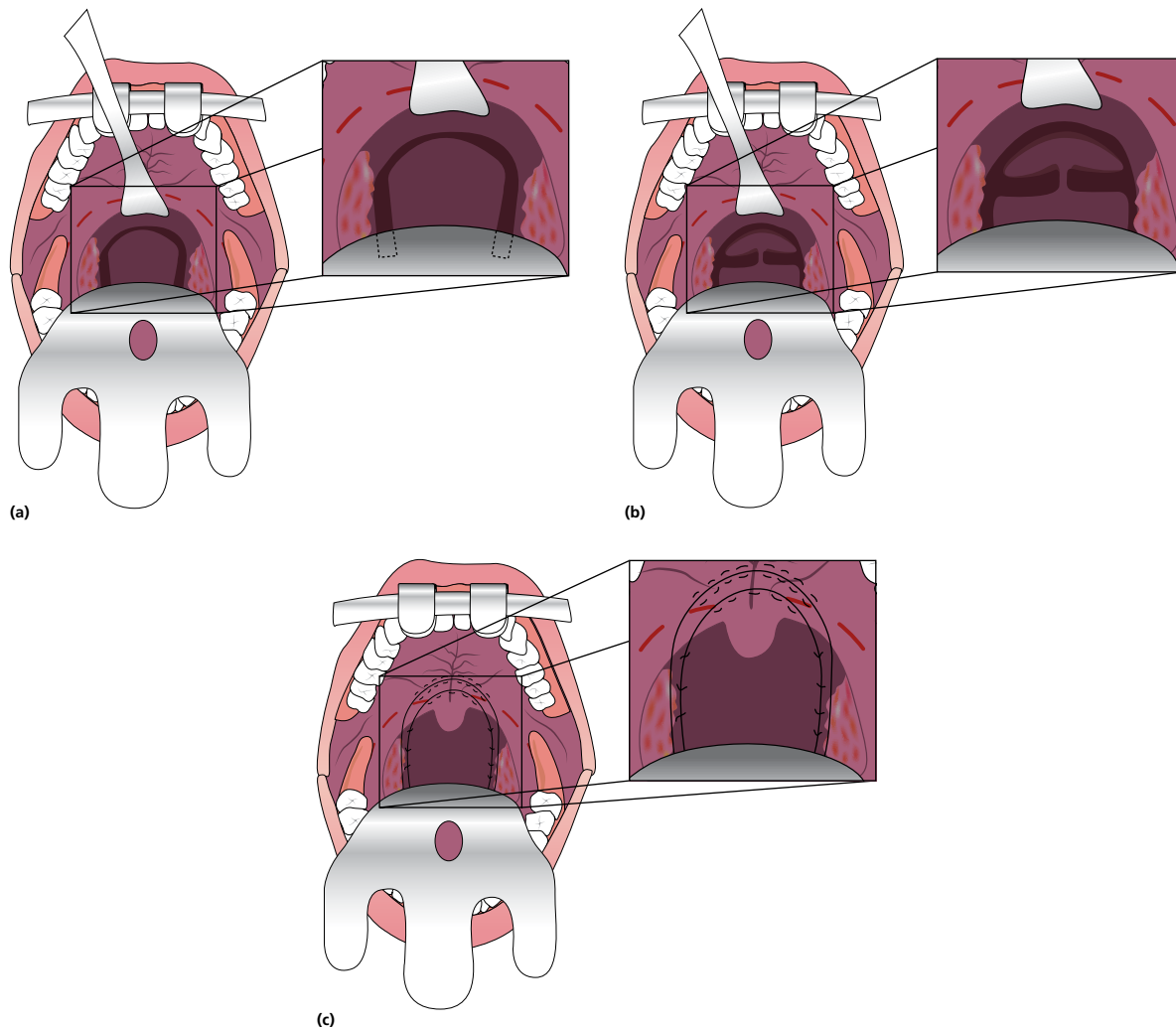


Figure 18.14 Diagrammatic illustration of the key steps in the operative technique for modified Hynes pharyngoplasty. (a) Bilateral palatopharyngeus muscle flaps are outlined. (b) The muscle flaps are elevated and transposed. (c) Flaps are sutured together end-to-end, as well as to the posterior pharyngeal wall.

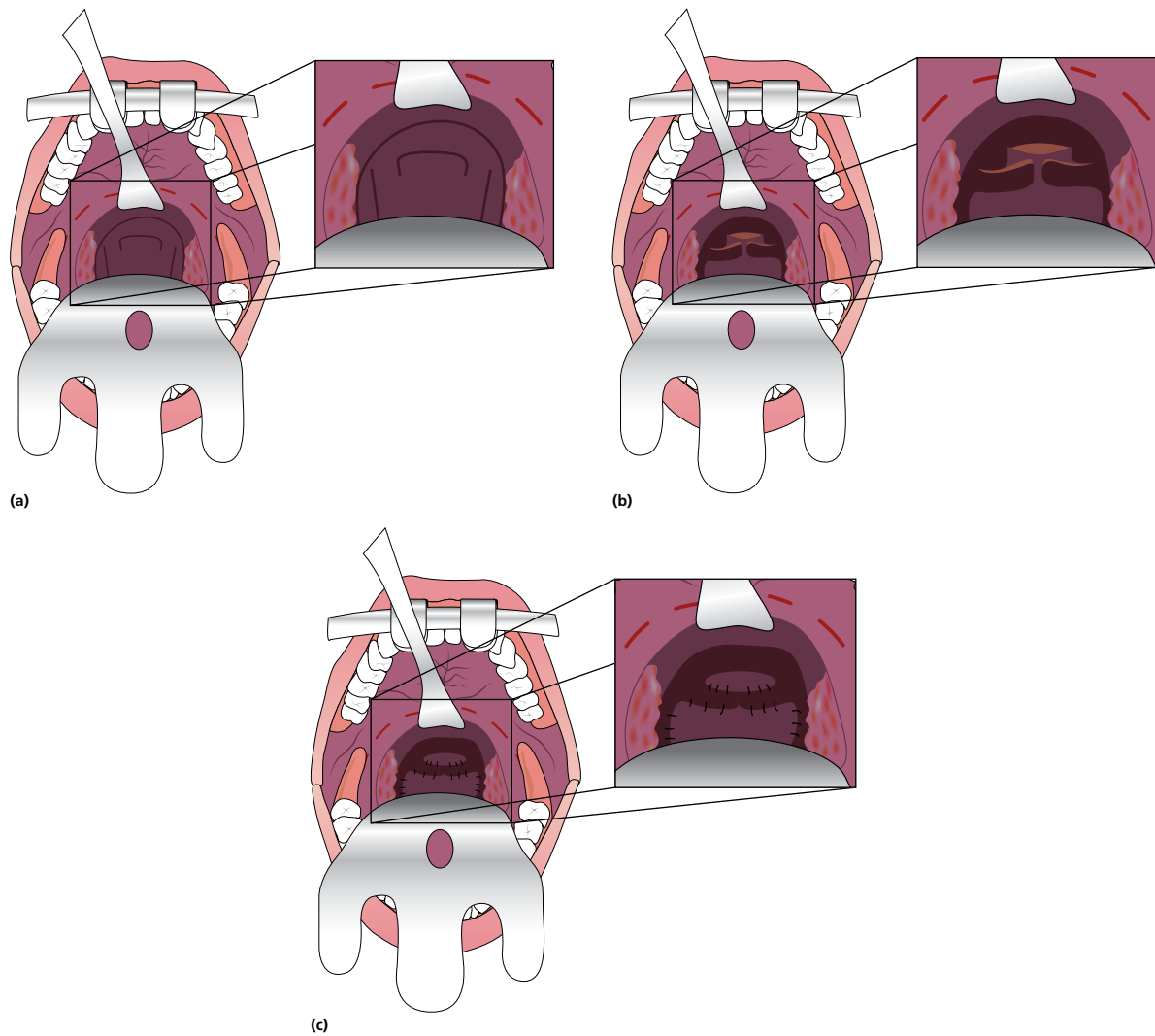


Figure 18.15 Diagrammatic illustration of the key steps in the operative technique for Orticochea pharyngoplasty. (a) Bilateral flaps from the posterior tonsillar pillars and an inferiorly based flap on the posterior pharyngeal wall are outlined. (b) The flaps are elevated and transposed. (c) The flaps are inset into the posterior pharyngeal flap.

pharyngoplasty, claiming that the transposed flaps of palatopharyngeus maintain intrinsic contractile function and therefore produce an active sphincter that assists in velopharyngeal closure (hence the term ‘sphincter pharyngoplasty’). However, many would argue that the dynamic nature of this procedure is questionable. Moreover, despite initial optimism that it would produce less airway compromise than a posterior pharyngeal flap, evidence suggests it is equally obstructive.

Prosthetic management

Non-surgical management is indicated in patients with a completely hypodynamic velopharyngeal mechanism (e.g. neuromuscular disorder), in those for whom surgical intervention is contraindicated or those with severe or persistent airway obstruction that is recalcitrant or not amenable to conservative management. Non-operative treatment consists of placement of a prosthetic device, either palatal lift and/or pharyngeal obtu-

rator. The palatal lift device is used to reposition the palate to a location where minimal excursion is required to effect closure, while the pharyngeal obturator acts to occlude the defect in the velopharyngeal sphincter.

Management of unilateral or asymmetric velopharyngeal insufficiency

Management of unilateral or asymmetric VPI includes pharyngeal bulb, skewed pharyngeal flap, unilateral pharyngoplasty and unilateral adhesion of the adynamic hemi-velum to the posterior pharyngeal wall. Mehendele and Sommerlad⁷⁸ described the use of a *unilateral* Moore pharyngoplasty flap to support the non-functioning or poorly functioning side of the velum, thereby assisting the active contralateral levator to elevate the entire velum. As originally described, the Moore pharyngoplasty involves bilateral postero-lateral pharyngeal flaps that are inset into a transverse incision at the junction of the middle and

posterior thirds of the nasal surface of the velum.⁷⁹ Goals of this procedure include obliteration of the lateral pharyngeal recesses, increase tissue bulk on the nasal surface of the velum at the point of potential contact of the velum with the posterior pharyngeal wall, and possibly velar lengthening. Moreover, the flaps are thought to augment the action of the levator by forming an active muscular sling. As previously mentioned, unilateral or asymmetric VPI is a common finding in patients with oculo-auriculo-vertebral spectrum.^{80,81}

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CHAPTER 19

Congenital ear anomalies

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Ear development

The intricate contours of the external ear are attributed to delicate elastic cartilage covered with a thin layer of subcutaneous tissue and skin. The ear develops around the first branchial cleft, which is the groove between the first (mandibular) and second (hyoid) arches. Auricular development begins during the fifth week of gestation. At the sixth week of embryogenesis six tubercles appear on the first and second branchial arches, the *auricular hillocks of His*. The three anterior hillocks of the mandibular arch give rise to the tragus, root of the helix and superior helix, respectively, and the three posterior hillocks of the hyoid arch form the remaining components of the auricle (Figure 19.1). The development of the ear is usually complete by the fourth month of gestation.¹ The auricles are initially positioned on the embryonic neck, and progressively migrate from this antero-caudal location postero-cranially as the mandible enlarges until they reach their final anatomic location by week 20. As a result, remnants of the developing ear may be found at any point along this route, often localized to the pre-auricular region, so-called *pre-auricular skin tags*. Rarely polyotia may be encountered, considered an extreme form of the rudimentary skin tag, distinguished by both size and morphology. Polyotia bears resemblance to the normal external ear and is often termed *mirror ear*, with a conjoined conchal bowl in place of the tragus (Figure 19.2). The middle ear structures are also derived from the first and second branchial arches, and external auditory meatus and canal from the first branchial groove. Thus, patients with microtia typically have atresia of the external auditory canal and tympanic membrane, as well as variable deformities of the middle ear ossicles. Before subtleties of congenital anomalies can be recognized, the topography of normal ear must be appreciated (illustrated in Figure 19.3).

Although the ear has its definitive form at birth, the auricular cartilage of the neonatal ear retains distinct malleability due to enduring high levels of circulating maternal oestrogen. Oestrogen upregulates the production of hyaluronic acid, which increases the pliability of cartilage. Oestrogen levels are highest

in the first 3 days after birth and decrease to a level comparable to older children at six weeks of age.² During this privileged period, *deformational* anomalies of the auricle can be corrected by non-operative means with external moulding devices. Authors have reported excellent results when correction is initiated within 3 days of birth.^{2–4} Thereafter, adjustment of deformational irregularities, such as auricular *malformations*, must be effected by surgical means. There is no consensus as to the upper age limit at which splinting therapy can be considered, nor reliable evidence on the length of time needed for auricular moulding.

There is marked variability in the phenotypic expression of congenital deformities of the ear, challenging classification of these anomalies. Marx⁵ published the first classification system, followed by Tanzer in 1978⁶ (Table 19.1). Congenital auricular anomalies can be categorized as either malformations or deformations. The malformed ear presents hypoplasia of cartilaginous and cutaneous constituents and gross morphological aberrations, whereas the deformed auricle has a normal chondrocutaneous component but irregular architecture. Further, although malformational anomalies arise from a combination of genetic and environmental factors, deformational abnormalities are deemed to result from external forces applied to the developing or neonatal ear and/or abnormal morphology or function of the three extrinsic and/or six intrinsic auricular muscles. The intrinsic musculature is mainly vestigial at birth but during auricular morphogenesis has an integral role in folding of the ear.

At birth, the normal auricle is approximately 66% of its adult size. The superior aspect of the helix is at the level of the tail of the eyebrow and the inferior aspect of the lobule corresponds to the base of the columella, with the long axis of the auricle inclined posteriorly at approximately 20° from vertical. At 5 years of age, the ear has grown to within 8 mm of its full adult vertical height (approx. 5.5–7 cm), and is therefore approximately 87% complete. Ear length reaches its mature size at age 13 in males and age 12 in females. In contrast, ear width at age 5 years has attained approximately 97% of its adult size.

Figure 19.1 Embryology of the external ear. (a) Hillock formation in an 11 mm human embryo. (b) Hillock configuration at six weeks gestation in a 15 mm embryo and (c) hillock derivations in the adult auricle. Source: Beahm & Walton, 2002.¹

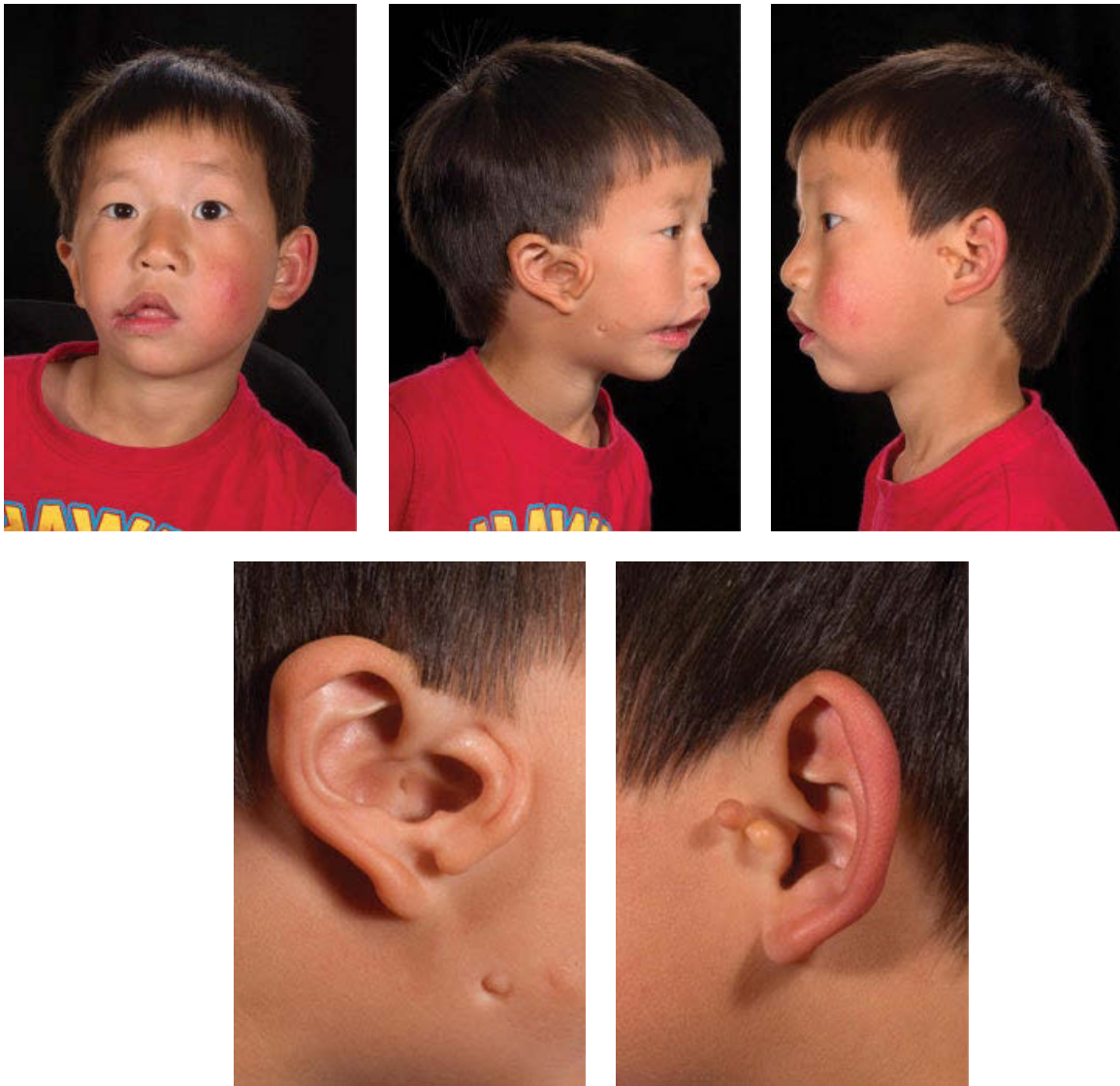
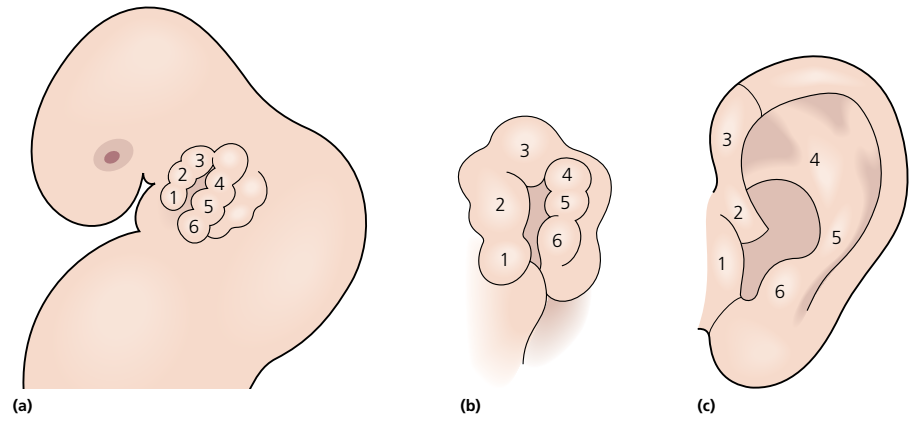


Figure 19.2 Patient with oculo-auriculo-vertebral spectrum who presented with simple pre-auricular skin tags on the left and classic mirror ear on the right.

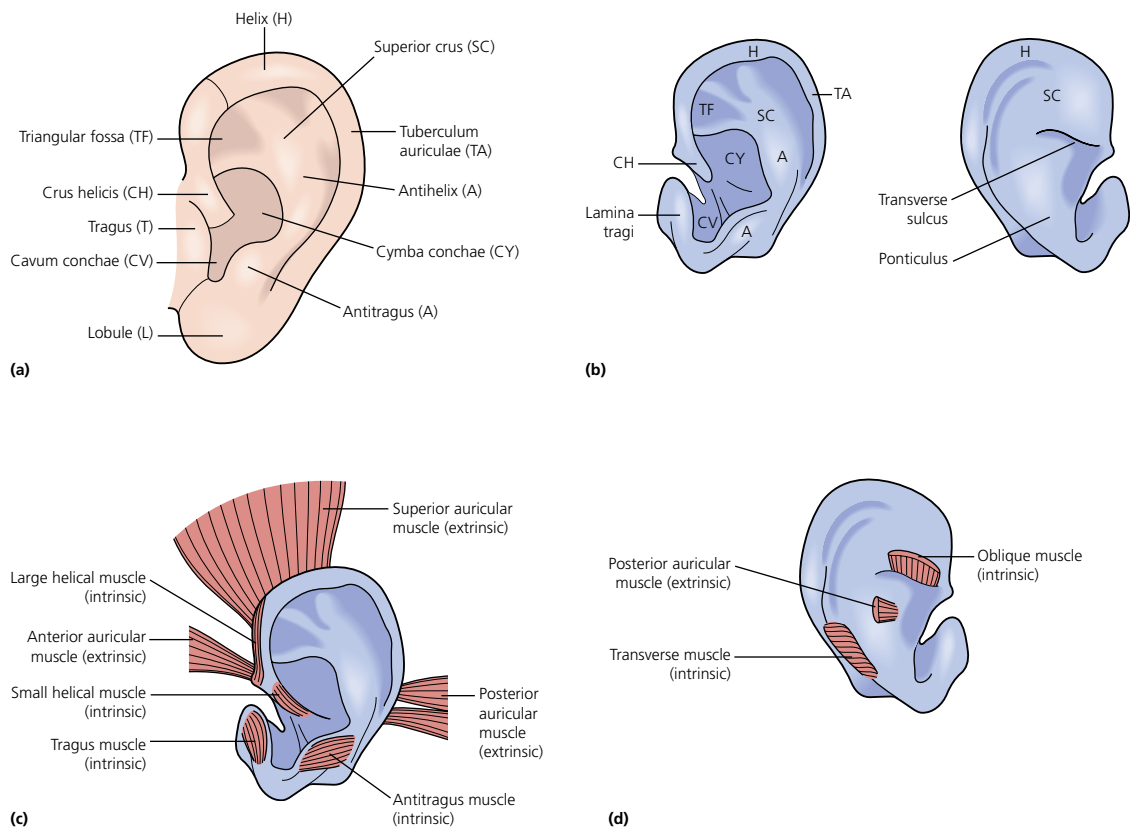


Figure 19.3 (a) External ear anatomy. (b) Anterior and (c) posterior views of ear cartilage anatomy. (d) Extrinsic and intrinsic auricular muscles. Source: Beahm & Walton, 2002.¹

Table 19.1 Comparison of the ear deformity classification systems of Marx and Tanzer

Marx ⁵	Tanzer ⁶
Grade I. Abnormal auricle with all identifiable landmarks	Type 1. Anotia
Grade II. Abnormal auricle without some identifiable landmarks	Type 2. Completely hypoplastic ear (microtia) a. With atresia of the external auditory canal b. Without atresia of the external auditory canal
Grade III. Very small auricular tag or anotia	Type 3. Hypoplasia of the middle third of the auricle Type 4. Hypoplasia of the superior third of the auricle a. Constricted (cup or lop) ear b. Cryptotia c. Hypoplasia of entire superior third Type 5. Prominent ear

The width of the ear continues to grow until age 7 and 6 in males and in females, respectively (width is approx. 55% of the length, 3–4.5 cm).⁷ Age at external ear maturity has important implications regarding timing of operative correction.

Prominauris

Ideal projection of the normal external ear is often cited as 1.5–2 cm from the scalp, and the projection of the helix from the mastoid as 10–12 mm in the superior portion, 16–18 mm and 20–22 mm in the middle and inferior portions, respectively.⁸ The auricle is considered as prominent if on frontal view it projects beyond these limits.

Prominent ears or prominauris is the most common congenital abnormality of the ear. It is associated with an autosomal dominant inheritance pattern with variable penetrance. This deformational anomaly is qualified by one or a combination of the following features:

- effacement or reduced definition of the antihelical fold;
- prominence of the concha (overdevelopment of the posterior wall of the concha and/or increased angle between mastoid and concha); and
- prominence of the lobule.

Other commonly associated features include excessive angulation of the helical root from the temple, absence of the helical roll (*shell ear* deformity) and macrotia. There is no consensus regarding the optimal age at which to perform correction (otoplasty), and surgery may be considered as early as age 4 years. Ideally, the authors' preference is to delay correction until at least age 6 years. At this age the ear has nearly attained its final

adult size and, although anecdotal, the increased rigidity of the auricle at age 6 appears to decrease the occurrence of inadvertent damage to the cartilage, which can result in irregularities in auricular form.

Numerous techniques have been described for correction of each of the three above-mentioned features of the prominent ear. With regards to re-creation of the antihelical fold, cartilage manipulation can be classified as cartilage suturing, cartilage scoring and cartilage breaking. Cartilage breaking involves placing a full-thickness cartilage incision or excision along the curve of the antihelix and superior crus, which permits the cartilage on either side of the incision to fold into position. As an alternative to this technique, initially proposed by Luckett in 1910,⁹ surgeons made a pair of incisions parallel to the desired antihelical fold and placed sutures between the two.¹⁰ The latter technique was modified by Converse *et al.*¹¹ and later Converse and Wood-Smith¹² to obviate the sharp fold of the antihelix that often results. Mustardé is credited for creation of the antihelical fold with suture fixation, wherein at least three full-thickness mattress sutures are placed to span the antihelix, with each suture purchase including cartilage and both perichondrial layers (Figure 19.4).¹³ Interestingly, Luckett in his original paper⁹ suggested that the antihelical fold could be created by appropriate suture placement, although he did not explore the concept.

Cartilage scoring techniques, as popularized by Stenström¹⁴ and Chongchet,¹⁵ are based on the principle of Gibson and Davis¹⁶ that cartilage tends to warp away from the abraded perichondrial surface. Some authorities contend that so-called conchal excess is in fact secondary to a lack of antihelical fold, and once the fold is restored any apparent 'excess' will be corrected. In

fact, Mustardé and Stenström address conchal protuberance by rolling back the posterior conchal wall, such that it is integrated into antihelix formation. Alternatively, conchal hypertrophy can be corrected with excision of a crescent-shaped cartilage segment (with or without overlying skin), through either an anterior or posterior approach, with care taken to avoid violation of the antihelix. Conchal-mastoid sutures, originally described by Owens and Delgado¹⁷ and later modified by Furnas,¹⁸ may be added to effect desired conchal setback, where the concha is sutured to the mastoid fascia and/or periosteum (Figure 19.5). Suture placement effectively rotates the concha forward and outward, thereby increasing the protrusion of the angular ledge that marks the junction of the concha with the external auditory canal. Thus, when this procedure is carried out one must be cautious to avoid narrowing of the external auditory meatus. Spira and Stal¹⁹ devised a laterally based conchal cartilage flap to appose to the mastoid periosteum, which decreased the tendency to obstruct the external auditory canal.

On the basis of cadaveric studies, Goulian and Conway²⁰ described the cartilage of the helix and concha as two discrete components, separated by a distinct fissure, and advocated relocation of the cauda helices (helical tail) onto the cavum conchae to reposition the prominent lobule. The latter is based on the commonly advanced premise that lobular protrusion is due to a displaced cauda helices.²¹ Many surgeons have subsequently adopted this approach and developed various technical modifications to retro-position the helical tail. Various other methods to address lobule prominence have been illustrated, such as fishtail excision of skin on the posterior surface of the lobule, wedge resection of the skin on the medial aspect of the lobule, and suture fixation of the lobule to the mastoid fascia.

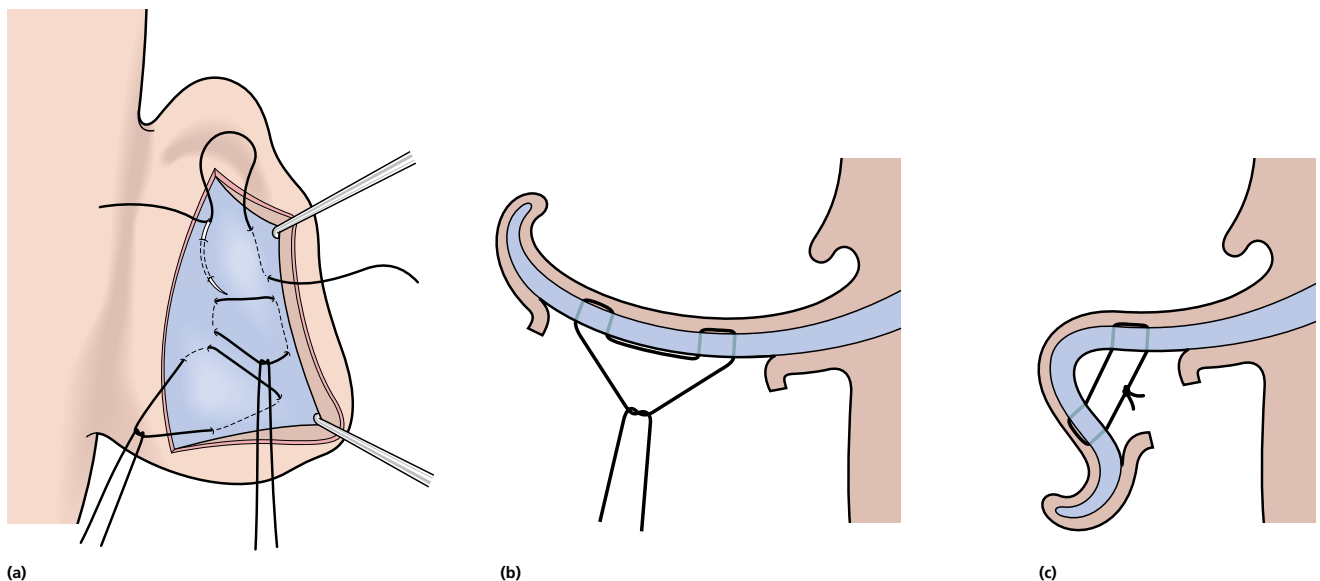


Figure 19.4 Diagrammatic illustration of Mustardé mattress suture placement for creation of an antihelical fold. (a) Posterior view of ear showing mattress sutures inserted from posterior aspect of cartilage at predetermined marks. (b) Coronal section with mattress suture inserted, to show 'bite' of cartilage and perichondrium. (c) With suture tightened to produce optimum folding. Source: Mustardé, 1963.¹³ Reproduced with permission of Elsevier.

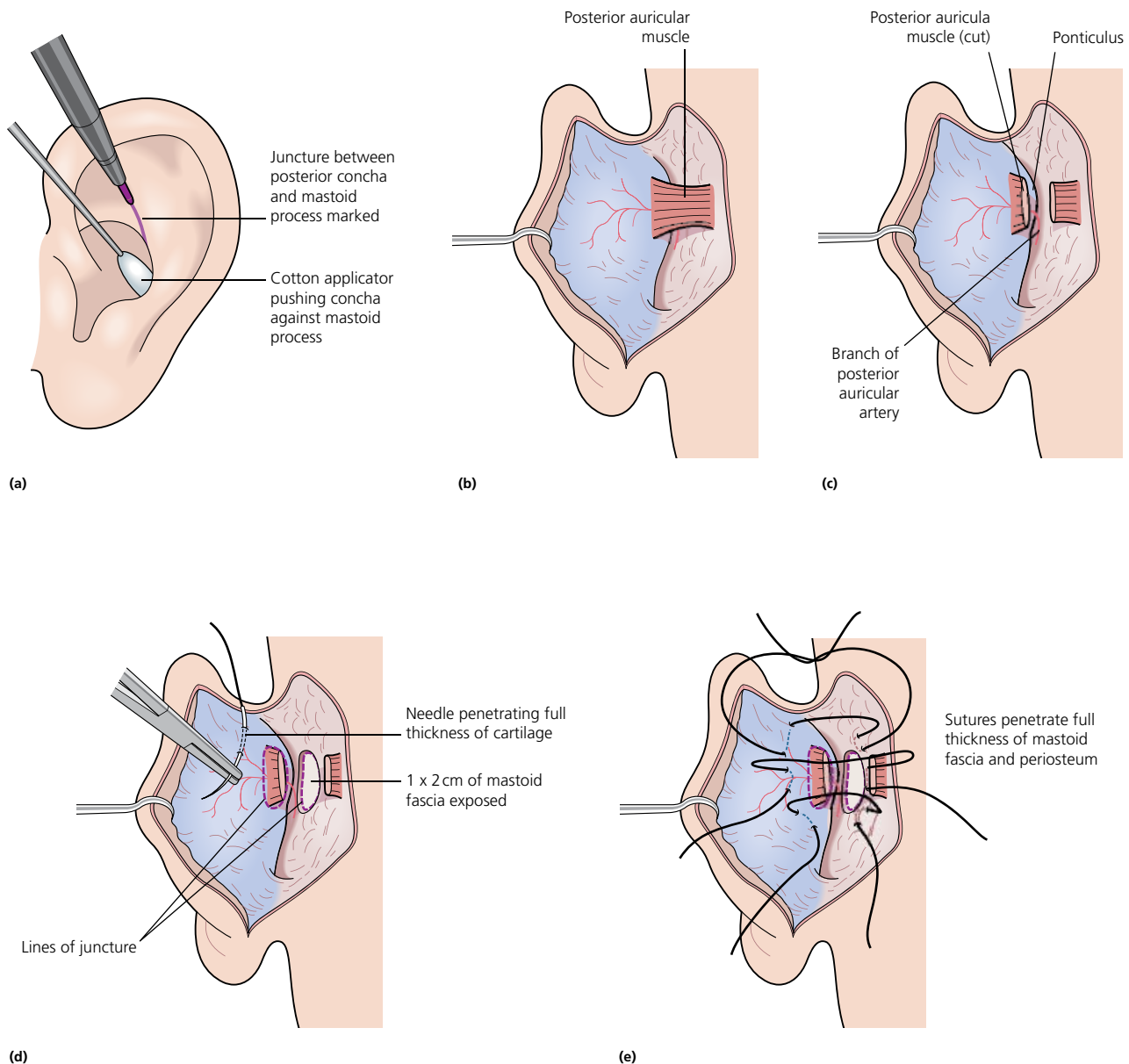


Figure 19.5 Diagrammatic illustration of key steps in the operative technique for placement of conchal-mastoid sutures as originally described by Furnas. Source: Furnas, 1968.¹⁸ Reproduced with permission of Lippincott, Williams & Wilkins.

There are a number of complications associated with otoplasty, which differ depending on the technique used. With cartilage suturing, suture extrusion and granuloma formation may occur, necessitating suture removal and resulting in recurrence of ear prominence. Cartilage irregularities and/or deformity are more likely to occur with cartilage scoring and/or breaking techniques. Haematoma and infection are two early complications that can occur with any technique and merit more detailed mention. With regards to haematoma formation, rapid recognition and appropriate management is essential. In the early postoperative period, onset of severe, often unilateral pain should prompt the immediate removal of the head dressing and if haematoma is confirmed, sutures released to drain the collection. Infection is another

potentially devastating complication of prominauris correction, which mandates aggressive management with intravenous antibiotics, as chondritis and cartilage deformity may ensue if not effectively controlled. Either of the latter two complications can result in significant cartilage destruction, for which correction may involve formal ear reconstruction with rib cartilage.

The authors' preferred method for prominauris correction is therefore a modified Mustardé technique for antihelix formation with or without Furnas' method for conchal setback.²² In the majority of cases, otoplasty is performed under general anaesthesia. The procedure begins with design of a conservative dumb-bell-shaped outline for skin excision on the posterior (medial) side of the auricle. Following infiltration of local anaesthesia to

the posterior auricular surface in the subcutaneous plane, skin excision is performed and the posterior auricular skin is dissected off the perichondrium. If placement of Furnas sutures is anticipated, dissection is extended to the ponticulus (conchal ridge, site of posterior auricularis muscle insertion) and an area of mastoid fascia for suture placement is exposed. Many often use a needle charged with methylene blue to mark the points for recreation of the antihelix. The needle is passed from the anterior (lateral) side and thus tattoos the exposed posterior cartilaginous surface at the desired location for suture placement. The authors do not utilize the latter technique, relying instead on familiarity with the architecture of the cartilage as viewed from the medial surface to identify the conchal border, triangular fossa and scapha, as underscored by Rubin *et al.*²³

The authors prefer to use a non-absorbable braided suture for cartilage fixation. Following antihelical fold formation, conchal prominence is assessed. If deemed excessive, Furnas sutures are placed with no conchal cartilage excision, in an effort to reduce the distance between concha and mastoid. In his original technical description, Furnas divided the posterior auricularis muscle fibres and posterior auricular ligament, which extend from the ponticulus to the mastoid fascia, prior to suture placement.¹⁸ Given that sutures are placed anterior to the muscle insertion on the concha and that great auricular nerve fibres span this area, the authors prefer to not divide these structures, ensuring rather that the posterior auricularis muscle is not strangulated with suture positioning. The latter is thought to avert the postoperative pain that may ensue following placement of conchal-mastoid sutures. If the lobule requires retro-positioning, dissection is extended into the lobe such that a deep suture is placed from both the tail of the helix and the subcutaneous lobular tissue to the floor of the concha. The latter is an effective means to address lower third prominence and avoids tissue excision in and scar on the earlobe. Once haemostasis is assured, the incision is closed with a running horizontal mattress with absorbable suture and a head dressing is applied for 2–5 days. Once the dressing is removed, the patient is instructed to wear a headband for six weeks.

Other auricular anomalies

Although prominauris typically presents bilaterally, other auricular deformities are usually unilateral. For unilateral correction, irrespective of the auricular anomaly, a template of the *normal* ear is made and the size or deformed part on the ear requiring correction is then addressed based on its contralateral counterpart.

In *Stahl's ear* deformity, a third crus projects from the antihelix toward the helical rim at an approximate right angle, flattening the helix and widening the scapha. Aberrant insertion of the intrinsic transverse muscle has been postulated as the aetiological origin of this anomaly.²⁴ For correction of Stahl's ear various techniques have been described, including wedge

excision of the auricular segment occupied by the third crus, excision of the abnormal cartilage with reshaping to approximate the scaphal concavity, and removal of the anomalous cartilage section and substitution in a reversed position.

Cryptotia is a congenital auricular anomaly seldom seen in the white populations, but is common among East Asian populations, with a reported incidence of approximately 1 in 400 among the Japanese. It occurs when the superior pole of the auricle is buried beneath the scalp skin, resulting in a lack of superior auriculocephalic sulcus, as well as associated distorted cartilage architecture.^{25,26} It has been suggested that the invagination of the upper portion of the auricle is attributable to aberrant insertion of the superior auricularis muscle into the upper portion of the helix. Normally this extrinsic muscle extends from the epicranial aponeurosis to the dorsal protuberance of the triangular fossa.²⁷ The deformity of the cartilage results from anomalous intrinsic auricular musculature, namely, intrinsic transverse and oblique muscles, which mould the auricle into its characteristic shape.²⁸ The transverse muscle, which extends from the conchal eminence to the scapha, forms the body of the antihelix and superior crus. The oblique muscle, between the eminence of the concha and triangular fossa, shapes the inferior crus. Although numerous techniques have been described for correction of this abnormality, the underlying principles of reconstruction are similar. That is, cartilage plasty depending on the degree of cartilaginous deformity, and local tissue rearrangement to fill the gap created in the superior portion of the retro-auricular sulcus and posterior aspect of the ear once the superior pole of the ear has been distracted from the scalp and fibrous soft tissue adhesions released. Methods to address the cutaneous defect include Z-plasty, V-Y plasty, advancement flap, a combination of local flap and skin graft or tissue expansion.

In 1970, Cosman *et al.* described a congenital ear anomaly where the ear resembles a question mark secondary to prominence of the upper third of the auricle, deficiency or absence of scapha and helix in the middle third, and partial or complete disjunction of the lobule.²⁹ So-called *question mark ears* are a defining feature of the *auriculo-condylar syndrome*, an autosomal dominant disorder first described by Uuspää in 1978; it is further characterized by mandibular condylar agenesis or hypoplasia and other mandibular anomalies.³⁰ Cosman *et al.*²⁹ proposed correction with cartilage excision along the antihelix–superior crus (thereby reducing auricular height and width) and Z-plasty closure with interdigitation of split helical rim and lobular flaps (for correction of the cleft above the lobule), similar to procedures used for auricular reduction in cases of macrotia. Although satisfactory results were achieved, the authors prefer formal reconstruction with costal cartilage based on principles advanced by Firmin for secondary reconstruction following otoplasty complications.³¹ Firmin states that any deformity involving more than one quarter of the ear, more than two anatomical planes or complex contours necessitates correction with costal cartilage. The skin is addressed with a double type

2 transfixion incision effected at the level of the auricular cleft, in which the superior and inferior portions of the ear are individually transposed and the intermediate mastoid skin recruited for framework coverage.

The terms *constricted*, *cup* and *lop ear* are often used interchangeably to describe variants of a similar auricular anomaly. Tanzer proposed a classification for constricted ears, which is valuable for reconstructive management of this deformity.³² Group 1 involves the *helix only*, where the superior portion of the helix is folded, giving what Cosman referred to as a lidded appearance³³ and Rogers called a *lop ear*.³⁴ The latter can often be addressed with simple excision of the excess skin and cartilage, as auricular height is usually adequate. Alternatively, for cases with a characteristically angulated superior helical rim, the involved portion of rim can be detached and repositioned. The deformed segment of cartilage can be dissected from the scapha and transposed to the posterior surface of the scapha as either a pedicled flap or free graft. In group 2a, the *helix and scapha* are involved, lidding is more significant with increased constriction of the helical rim. Further, features of the prominent ear may be present, including ill-defined superior crus and

enlarged conchal cavity. However, the cutaneous envelope is adequate, such that correction of the malformed cartilaginous structure can be re-draped with the existing skin. The latter often qualifies the *classic lop ear*.

The authors' preferred method of correction for this group is the technique described by Musgrave,³⁵ who elaborated on the procedure first described by Stephenson³⁶ (Figure 19.6). This approach involves complete de-gloving of the superior pole cartilage. Thereafter, radial full-thickness incisions are made in the lidded segment of cartilage, which allows the cartilage to unfurl in a postero-superior direction, thereby restoring auricular height and width. Scoring of the anterior surface of the cartilage can be effected to assist with transposition of the multiple segments created. A conchal cartilage graft is harvested from either the contralateral or ipsilateral (as originally described by Musgrave) ear and used as a curved strut to maintain the cartilaginous segments in place and bridge the gaps in the expanded helical rim. The cutaneous envelope is re-draped over the refashioned cartilage framework and the reconstruction supported with bolsters placed in the scapha beneath the helical rim and on the posterior surface of the ear. If necessary,

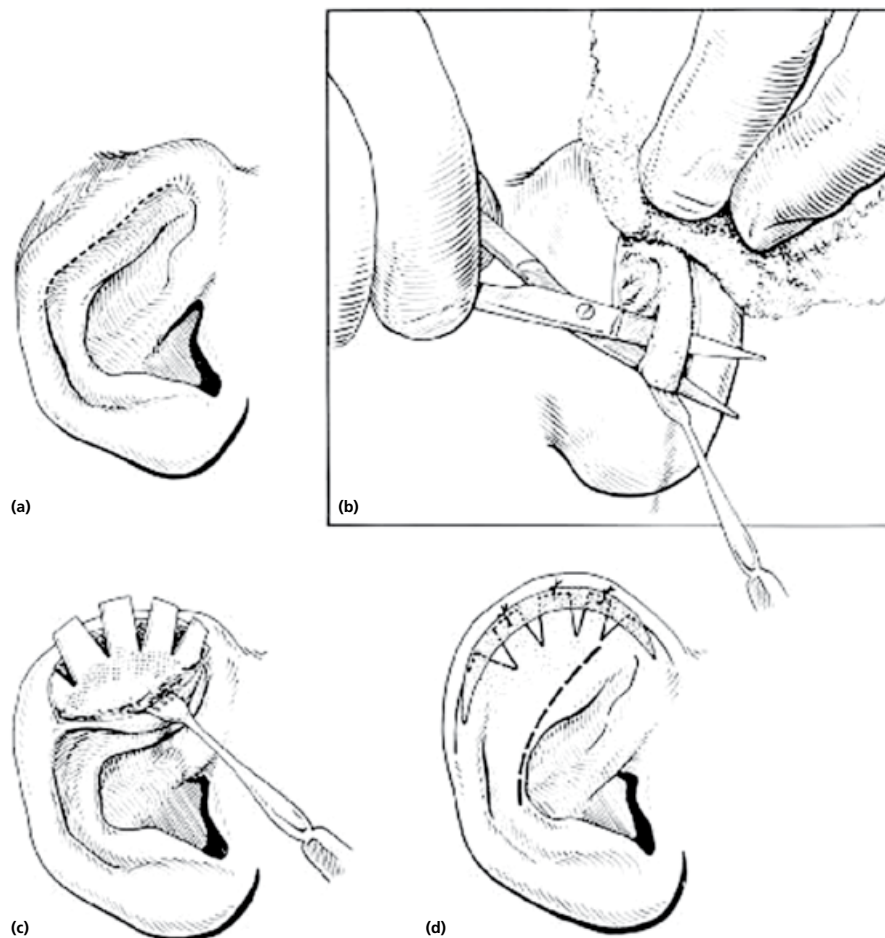


Figure 19.6 Diagrammatic illustration of key steps in the operative technique for lop ear correction described by Musgrave. Source: Musgrave, 1966.³⁵ Reproduced with permission of Lippincott, Williams & Wilkins.

a posterior skin approach can be added to address auricular prominence. Namely, placement of Mustardé sutures for re-creation of the antihelical fold.

The primary distinguishing feature between groups 2a and 2b is *skin deficiency* in the latter subgroup. In this case, the constriction is more significant and the malformed ear has characteristics of both lop and prominent ears, including an overdeveloped concha, deficient superior helical margin, antihelical crura and antihelical body and diminished auricular height (to which the term *cup ear* is usually applied). Indeed, Cosman described the constricted ear deformity with four major features: lidding, protrusion, decreased ear size and auricular dystopia; and advised cartilage expansion and supplemental skin coverage for a size discrepancy of 1.5–2 cm when compared to the normal contralateral ear, which is consistent with a Tanzer type 2b ear. Group 3 is not unlike a conchal-type microtia deformity. As groups 2b and 3 involve deficiency of both skin cover and underlying cartilage, reconstruction necessitates framework fabrication with costal cartilage and recruitment of regional skin, as described in the following section for microtia reconstruction.

Microtia

The pathogenesis of *microtia* remains elusive, with phenotypic expression ranging from pre-auricular skin tags to *anotia* (or complete absence of the auricle). Microtia is a developmental malformation of the external ear with an estimated prevalence between 0.83 and 17.4 per 10 000 births.³⁷ The incidence of microtia differs among ethnic groups, being greatest among Asians (especially Japanese). Microtia occurs 20–40% more commonly in males, involves the right side in nearly 60% of unilateral cases and bilateral deformity accounts for approximately 10% of cases.³⁸ The genetic and cellular mechanisms responsible for the normal morphogenesis of the external ear remain incompletely defined. Microtia appears to be associated with single gene mutations in syndromic and familial cases, whereas in sporadic cases a combination of genetic and environmental factors or polygenic causes are involved. The genetic contribution to microtia is supported by the higher concordance in monozygotic versus dizygotic twins, familial cases with autosomal dominant and recessive modes of inheritance, as well as the more than 18 microtia-associated syndromes for which chromosomal aberrations or single gene defects have been elucidated.

Current theories for microtia development include high altitude, neural crest cell disturbance and vascular disruption.³⁸ Evidence in support of the role of neural crest cells is derived from recent studies on the causative mechanisms of various teratogens on microtia, including retinoids and diabetic embryopathy. The development of microtia following localized vascular insult during embryogenesis is primarily supported by studies of Poswillo,^{39,40} in which unilateral ear and mandibular anomalies resulted following

ipsilateral haematomas at the junction of pharyngeal and hyoid arteries when monkeys were exposed to thalidomide and mice to triazine. This long-standing theory has been challenged by evidence of bilaterality in isolated microtia, as well as the occurrence of non-craniofacial anomalies frequently seen in oculo-auriculo-vertebral spectrum. Johnston and Bronsky⁴¹ disputed Poswillo's original experiments and contended that the development of haematomas was delayed in relation to drug exposure and that significant branchial arch hypoplasia preceded haemorrhage.

Although more commonly an isolated unilateral abnormality, in some patients microtia portends other, perhaps more subtle, craniofacial anomalies. Accordingly, a number of cases present as a component of more complex craniofacial disorders. Most notable are those associated with aberration of first and second branchial arch derivatives, namely the *oculo-auriculo-vertebral spectrum*. Microtia may also constitute an important diagnostic feature of various other craniofacial syndromes, including Treacher Collins, Nager, Townes–Brocks, Kabuki, CHARGE, Meier–Gorlin, branchio-oto-renal and branchio-otic syndromes. Indeed, approximately 20–60% of patients with microtia have other anomalies or an identifiable syndrome.³⁸ Noteworthy associated abnormalities include renal anomalies, cardiac defects, oral clefts, microphthalmia, polydactyly and vertebral irregularities. In view of the latter, clinical assessment of these patients must include observation for these commonly associated features.

Patients with microtia should be treated by a specialist multidisciplinary team including plastic and reconstructive surgeon, otolaryngologist and audiologist. Initial assessment should include a formal audiologic evaluation, with either auditory brainstem response testing in the newborn or audiogram in the child, which are mandatory to determine the degree and type of hearing loss. Microtia with aural atresia is associated with conductive hearing loss, although a small minority may exhibit sensorineural hearing loss despite the discrete embryonic origin of the internal ear. In the presence of an atretic external auditory canal, middle ear deformity and presence of cholesteatoma must be assessed. More advanced radiologic examination may be indicated for elucidation of anomalous temporal bone (middle ear/ossicles) and facial nerve anatomy, which provides anatomic detail essential for any projected otologic surgery. More recent evidence has shown that with regards to academic and career attainment patients with unilateral hearing loss benefit from auditory rehabilitation with an amplification device, as functionally these patients have difficulty with localization of sound and understanding in noisy environments.⁴² Patients with bilateral microtia and conductive hearing loss require early fitting with a bone-conduction hearing device to avert impairment of speech development. The current gold standard for hearing rehabilitation is an osseointegrated bone-anchored hearing aid (BAHA).⁴³ Ideally, this device should be placed 65–70 mm posterior to the external auditory meatus or site of future placement of the ear construct. The authors do not currently advocate surgical restoration of the external auditory conduit (canaloplasty) and middle ear due to the associated

postoperative morbidity, including infection, desquamation, meatal restenosis and inconsistent results in hearing improvement. If canaloplasty is considered, it should follow external ear surgery, as any resulting scarring of the area impedes subsequent reconstruction. In general, at the time of the second stage of auricular reconstruction (sulcus formation), a neo-auditory canal is drilled and covered with local fascial flap and skin graft or directly with skin graft.⁴⁴ However, surgical results are generally poor and as such the latter practice is not universally recommended in patients with bilateral microtia and certainly not in those with unilateral microtia. In a recent report on international trends in the treatment of microtia, authors noted that the great majority of centres did not perform middle ear reconstitution.⁴⁵

Reconstruction

Although Gillies⁴⁶ is credited as the first to propose the use of autogenous costal cartilage for auricular framework fabrication, the modern era of ear reconstruction began in 1959, when Tanzer reintroduced the use of rib cartilage for autologous microtia reconstruction, which is still recognized as the gold standard reconstructive option.^{47,48} Following the original precepts of Tanzer, Brent^{49,50} refined autogenous reconstruction with the introduction of his four-stage technique. The primary advance contributed by Brent was placement of the cartilaginous framework in ideal position *prior* to transposition of the lobule,

which is the reverse of Tanzer's sequence. When the lobular remnant is transposed at the first stage, placement of the ear construct must be adapted to the lobe with a compromise on location. In 1993, Nagata published his modified two-stage approach to autologous microtia reconstruction, wherein the lobule is transposed in the *same* stage as framework fabrication (including the tragus) and placement.^{51–55} Thus, Nagata's first stage combines the first, second and final stage of Brent's technique. In addition, both authors proposed their own respective classification systems to describe the variable appearance of the microtia deformity. A detailed comparison of the latter two classifications and techniques is provided in Table 19.2.^{49–56}

In the authors' practice, auricular reconstruction is realized as a two-stage procedure, as initially described by Nagata and later modified by Firmin.^{57–59} Autologous reconstruction is offered at the minimum age of 9 years, depending on the physical and psychological maturity of the child. At this age, there is usually sufficient costal cartilage for framework fabrication and most children have the requisite level of understanding to be an active member in the treatment plan and postoperative rehabilitation and care. For the first stage of reconstruction, following induction of general anaesthesia, preoperative markings are made. On the unaffected side, two distances are outlined and transposed onto the side with microtia: the distance between the lateral canthus to the most anterior point of the helix and between the lateral commissure to the lowest point of the lobule. The latter two measures determine the anterior- and inferior-most points for placement of the reconstructed ear.

Table 19.2 Comparison of two classifications and techniques for microtia reconstruction

	Brent ^{49,50,56}	Nagata ^{51–55}
Microtia classification		
Types	1. Classical microtia (sausage-shaped vestige, usually with absent external auditory meatus) 2. Atypical microtia (concha, antihelix, tragus, antitragus, with absence of upper portion of ear, with or without presence of external auditory canal)	1. Lobular microtia (ear remnant with malpositioned lobule, without concha, external auditory meatus and tragus) 2. Conchal microtia (lobule, concha, with or without external auditory meatus, tragus, antitragus with incisura tragica, present to variable degrees) 2a. Small type, or 2b. Large type (depending on degree of conchal development)
Operative technique		
Number of stages	3 (stage 2 and 3 combined) or 4 1. Cartilage framework fabrication/placement 2. Transposition of lobule 3. Elevation of ear construct and creation of auriculocephalic sulcus 4. Tragus construction, conchal excavation and contralateral otoplasty, if indicated	2 1. Cartilage framework fabrication (with tragal component)/placement and transposition of lobule (Brent stage 1, 2 and 4) 2. Elevation of ear construct and creation of auriculocephalic sulcus
Age at reconstruction	4–6 years	10 years (or chest circumference at xiphoid, 60 cm)
Skin incision	Small incision anterior to vestige	W-shaped skin incision
Costal cartilage	Contralateral ribs, 6–8	Ipsilateral ribs, 6–9
Framework fixation	Clear nylon sutures	Wire sutures
Dressing	Closed suction drains	Bolsters
Tragus construction	Chondrocutaneous graft from contralateral ear	Rib cartilage
Elevation of ear construct and creation of auriculocephalic sulcus	Occipitalis turnover flap and skin graft with cartilage wedge	Temporoparietal or posterior auricular fascial flap and skin graft with cartilage wedge

Next, the angle between the axis of the nasal dorsum and the normal ear is ascertained and this angle is marked on the abnormal side and used to determine the inclination of the ear construct. Finally, the course of the superficial temporal artery is marked using a Doppler probe. An outline of the normal ear is drawn onto X-ray film, which serves as the template for fabrication of the auricular framework. Once the location of the future ear on the abnormal side is determined, the outline of the normal ear is drawn, potential adaptations of the microtic vestige are assessed and then the appropriate skin approach is selected (Figure 19.7).

The costal cartilage segments of ipsilateral ribs 6–8 are harvested through a 5 cm oblique incision along the costal margin, preserving the posterior perichondrium *in situ*. The cartilaginous base block is fashioned from the synchondrosis of ribs 6 and 7 with cartilage sculpting effected on the surface denuded of perichondrium (as the perichondrial surface ensures cohesion of the cartilaginous block to the recipient bed). Thus, the posterior surface of the harvested cartilage becomes the anterior surface of the reconstructed ear. The helix and antihelix are shaped from the eighth rib. The tragus, antitragus, tragal bar and cartilage block for framework elevation (second stage) are carved from remaining cartilaginous segments, and fixed in place with 38-gauge wires (Figure 19.8). During the carving of the auricular framework, cartilage shavings and unusable cartilaginous fragments are collected, morcelized and placed in a Vicryl mesh sleeve that is then used to reconstitute the costal margin.⁶⁰ Intercostal nerve blocks (chirocaine, 2.5 mg/mL) are carried out for postoperative analgesia prior to inset of the mesh, and the cartilage block for the second stage is banked in a subcutaneous pocket along the inferior margin of the chest incision.

Based on the morphology of the microtia, Firmin⁵⁸ proposed a classification system, perhaps more useful than those of her contemporaries, based on the type of skin incision required for framework placement. Originally, type 1 skin incision was used for typical lobular microtia and involved lobular transposition

with Z-plasty. However, Firmin has now abandoned type 1 skin incision in favour of type 2, transfixion, skin incision. This technical adaptation came from observation of tenuous vascularity of the posterior skin flap. The transfixion incision is indicated for both lobular and conchal-type microtia, where the inferior portion of the auricle is sufficiently developed to accept the lower extremity of the framework. The latter approach consists of a full-thickness transverse incision of the remnant auricle, thereby creating two skin edges. The posterior skin edge is sutured to the inferior skin edge of a corresponding transverse incision in the mastoid skin, and the anterior skin edge is sutured to the edge of the superiorly based skin flap that covers the cartilage framework. Type 3 skin incision is subclassified into type 3a, which is used when the ear is malformed but comparable in size to the contralateral side, such that the deformed cartilage can be dissected from the skin envelope and the framework placed into the cutaneous pocket (sulcus is preserved, single stage reconstruction), and type 3b, which is implemented for cases of anotia or where the microtic remnant cannot be used (e.g. significant auricular dystopia).

Once the appropriate skin incision is made and the amorphous remnant cartilage is removed, the auricular pocket is dissected in the plane deep to the subdermal vascular plexus, with dissection extended beyond the limits of intended framework placement (marked auricular outline) to recruit sufficient skin to adequately cover the contours of the construct without undue tension. The cartilage framework is placed in the subcutaneous pocket over two suction drains. If a temporoparietal fascial flap is required for partial or complete coverage of the cartilaginous framework (e.g. low temporal hairline or inadequate skin envelope in, for instance, cases of secondary reconstruction), it is harvested following dissection of a subcutaneous pocket through a 4 cm chevron-type incision placed at the apex of the temporal fossa, centred over the superficial temporal artery. The fascial flap is then covered with a split-thickness skin graft harvested from the contralateral scalp.

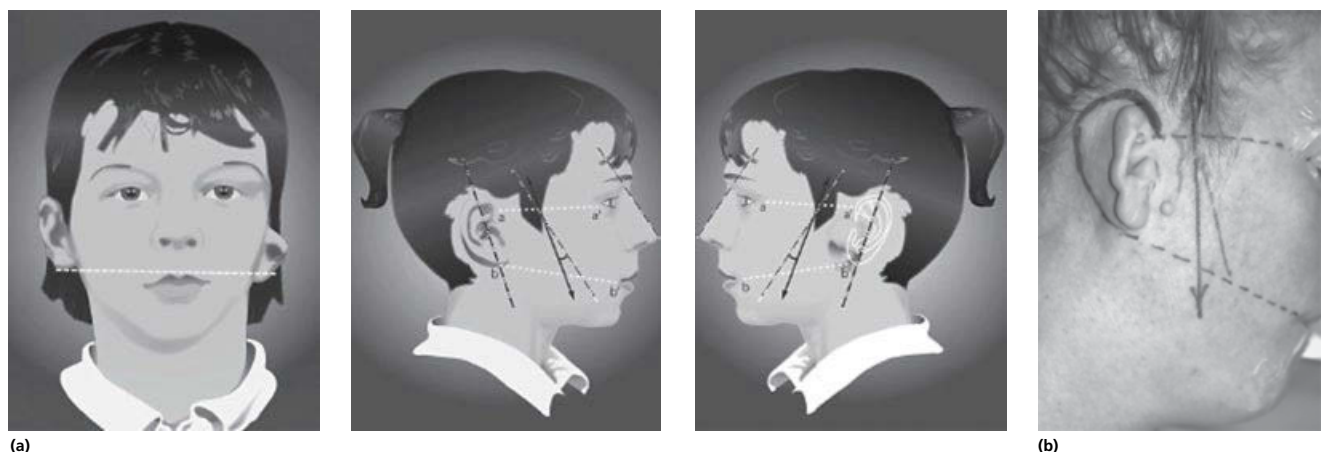


Figure 19.7 Preoperative measurements and markings for assessment of ideal location for the reconstructed ear, as outlined in the text. (a) Estimation of the ideal location. (b) During surgery, landmarks are used to correctly place the reconstructed ear.



Figure 19.8 Framework fabrication. (a) A 5 cm oblique incision is marked along the costal margin ipsilateral to the side to be reconstructed. (b) The cartilage segments of ribs 6–8 are harvested and the framework fashioned as described (c, d). More recently, the authors have modified framework fabrication and no longer sculpt the tragus and antitragus segments as a single unit. Instead, the antihelix and antitragus are shaped as a single component and a cartilaginous strut is made to span the base block, on which the tragus is placed (e). (f) Intraoperative result at the end of the first stage of reconstruction.

Since January 2007, reconstruction for cases of bilateral microtia has been executed simultaneously, with each step of this operative sequence performed in tandem. No dressings are applied to the reconstructed ear(s) to enable constant surveillance of the skin envelope and underlying construct. Patients are admitted to hospital for 4 nights, where the ear(s) is monitored and the vacutainer drains are changed every 4 h to maintain the skin maximally applied to the contours of the framework. Patients are placed on a 24-h course of intravenous antibiotics, followed by oral antibiotics for a total of one week. On day 7, the drains are removed and the patient is discharged from hospital.

The second stage is routinely performed six months later, with the exception of first-stage reconstruction necessitating temporoparietal fascial flap coverage of the construct, for which an interval of 12 months must elapse before framework elevation and sulcus formation. The framework is released through a peri-helical incision extending to fascia. Elevation of the ear construct is buttressed by placement of the previously banked cartilage block behind the antihelix (positioned to form the posterior conchal wall). Although a temporoparietal fascial flap is commonly used for coverage of the framework and cartilage block, the authors prefer a random pattern galeal fascial flap. The retro-auricular skin is advanced toward the sulcus and the

posterior surface of the ear, auriculocephalic sulcus and mastoid region are surfaced with a split-thickness skin graft harvested from the ipsilateral scalp (Figure 19.9). Following the second stage, patients are admitted overnight to complete a 24-h course of intravenous antibiotics, and then discharged on oral antibiotics for 7 days. The dressing remains in place until the first postoperative visit at one week. At two weeks, the auriculocephalic sulcus is fitted with an elastomer ear splint.

The second stage presents an opportunity to carry out other refinements to the ear construct and/or additional reconstructive procedures to enhance facial form and/or symmetry. This combined reconstructive approach has become the authors' routine practice, with complementary procedures including contralateral prominauris correction, ear piercing, fat transfer for soft tissue augmentation, as well genioplasty and malar augmentation to camouflage any associated bony deficiency.⁶¹ Typical results achieved following completion of two-stage microtia reconstruction are shown in Figure 19.10.

Although modifications and refinements in technique have been developed over the last several decades, the optimal management of

several aspects of autologous microtia reconstruction remain unresolved and debated by current leaders in the field of autologous ear reconstruction. Some authorities contend that the second stage of reconstruction should be reconsidered as creation of the auriculocephalic sulcus can result in a loss of framework definition. Further, optimal management of significant bony deficit, as is common in cases of severe craniofacial microsomia, remains in dispute.

Oculo-auriculo-vertebral spectrum

As previously mentioned, microtia is an integral component of a number of syndromes, including the *oculo-auriculo-vertebral spectrum* (more commonly referred to as hemifacial or craniofacial microsomia). In effect, it is now well established that microtia is a *forme fruste* of the craniofacial malformation sequence associated with hemifacial microsomia.⁶¹ However, all structures derived from the first and second branchial arches may be affected, unilaterally or bilaterally to different degrees.



Figure 19.9 Second stage of reconstruction. Early postoperative result achieved following completion of two-stage reconstruction in a patient with unilateral isolated microtia.

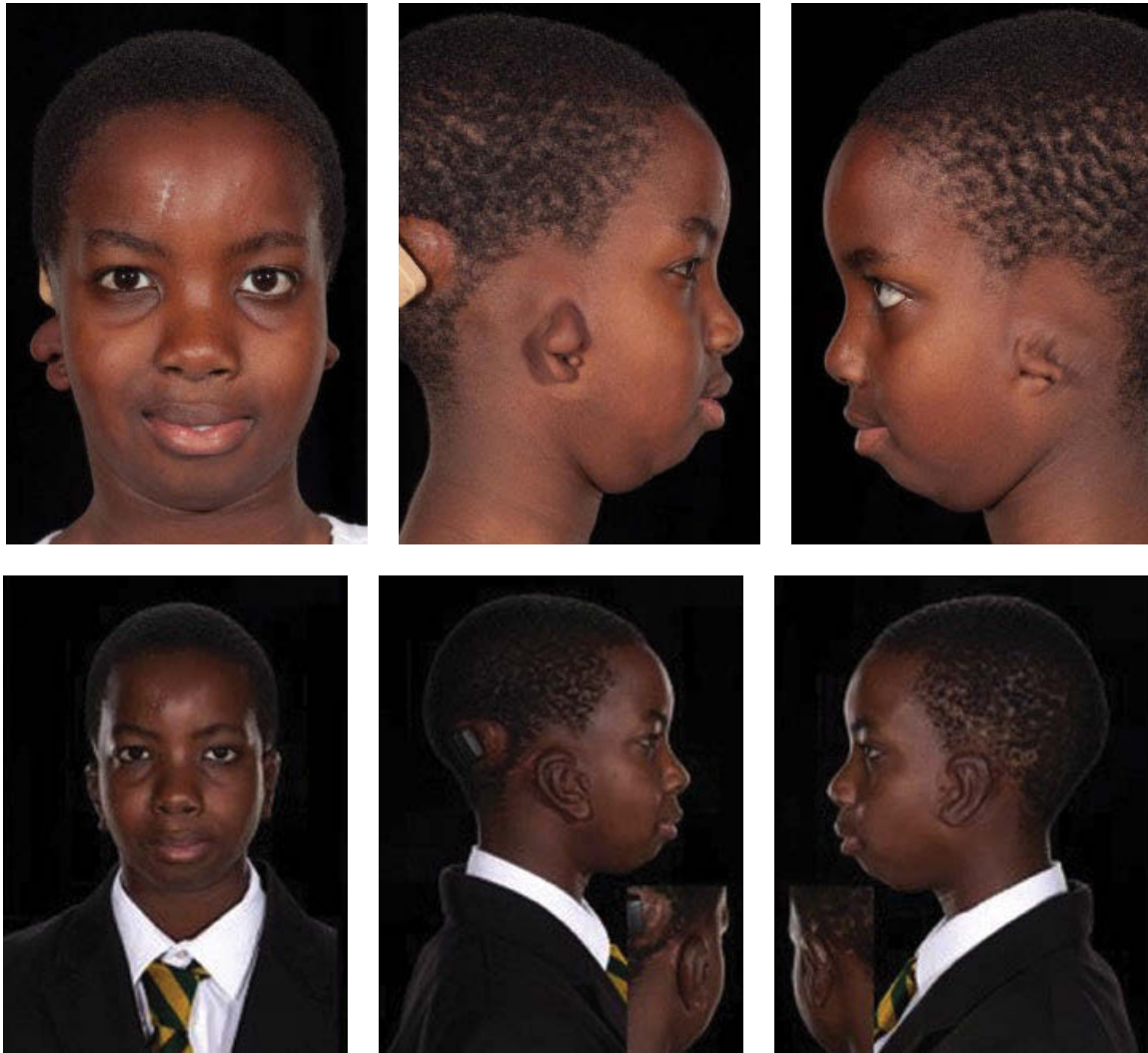


Figure 19.10 Result achieved following completion of two-stage reconstruction in a patient with bilateral isolated microtia, on whom each stage of reconstruction was performed simultaneously for both sides.

Moreover, other extracranial malformations may be present, including vertebral, renal and limb anomalies. In these patients, significant dystopia of the vestige secondary to underlying deficiency of the craniofacial skeleton, notably the temporal bone, as well as soft tissue hypoplasia, renders selection of appropriate location for construct placement difficult. In addition, the external auditory meatus and canal may be in an ectopic position necessitating transposition.⁶³ Thus, achieving a symmetric result presents a particular reconstructive challenge, especially when auricular reconstruction is one of several elements in the reconstructive sequence for facial dysmorphism (Figure 19.11). Moreover, craniofacial deficiency is often associated with an insufficient skin envelope for framework coverage, as well as a low temporal hair-line resulting in hair extending onto the skin that will drape the superior portion of the ear construct. Tissue expansion has been suggested to address the former and temporoparietal flap fascial

harvest for framework cover is generally used to deal with the latter. Although more detailed description of reconstructive modifications in the presence of these features is beyond the scope of this chapter, these general points are mentioned for completeness.

Complications

There are a number of complications associated with autologous auricular reconstruction, related to both donor site and the site of reconstruction. With regards to donor site, the most notable complication is chest wall depression, often cited by proponents of synthetic reconstruction as a primary argument against costal cartilage harvest for framework fabrication. Leaving the posterior perichondrium *in situ* may potentially lessen chest wall

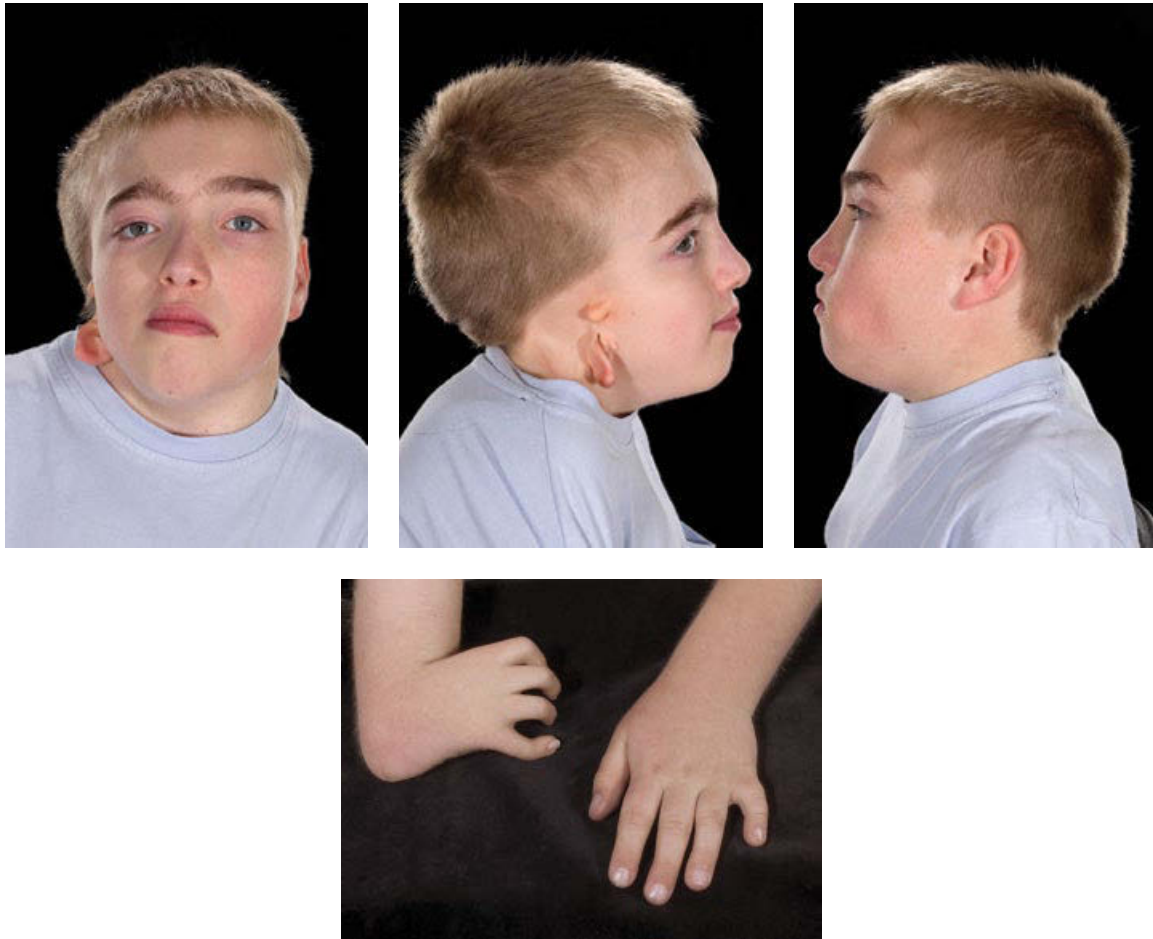


Figure 19.11 Patient with severe manifestation of the oculo-auriculo-vertebral spectrum who presented with significant dystopia of the auricular remnant.

morbidity by promoting cartilage regeneration at the donor site. In our practice, we have found that reconstitution of the costal margin, as previously described, minimizes potential contour deformity. During rib harvest, one may inadvertently create a breach in the parietal pleura, resulting in pneumothorax. This is typically identified during the procedure and the pleural defect promptly repaired.

At the level of the reconstructed ear, the most common complication is exposure of the cartilage framework, which may occur as a result of extrusion of suture/wire material or vascular compromise of the overlying skin envelope. The latter mandates early recognition and appropriate treatment as cartilage resorption and distortion of the final form of the ear construct may result. In general, areas of exposure $>10 \text{ mm}^2$ require surgical management with debridement and cutaneous or fascial flap coverage, depending on the location relative to the contours of auricle and adjacent tissue mobility (Figure 19.12).

Smaller defects can usually be addressed conservatively with local wound care. The most significant complication is infection with consequent cartilage resorption. Infection, usually

secondary to *Pseudomonas*, most often occurs in the presence of a vestigial external auditory canal. Infection may also ensue following construct exposure. Additional complications include haematoma or seroma formation, cartilage framework resorption or fracture.

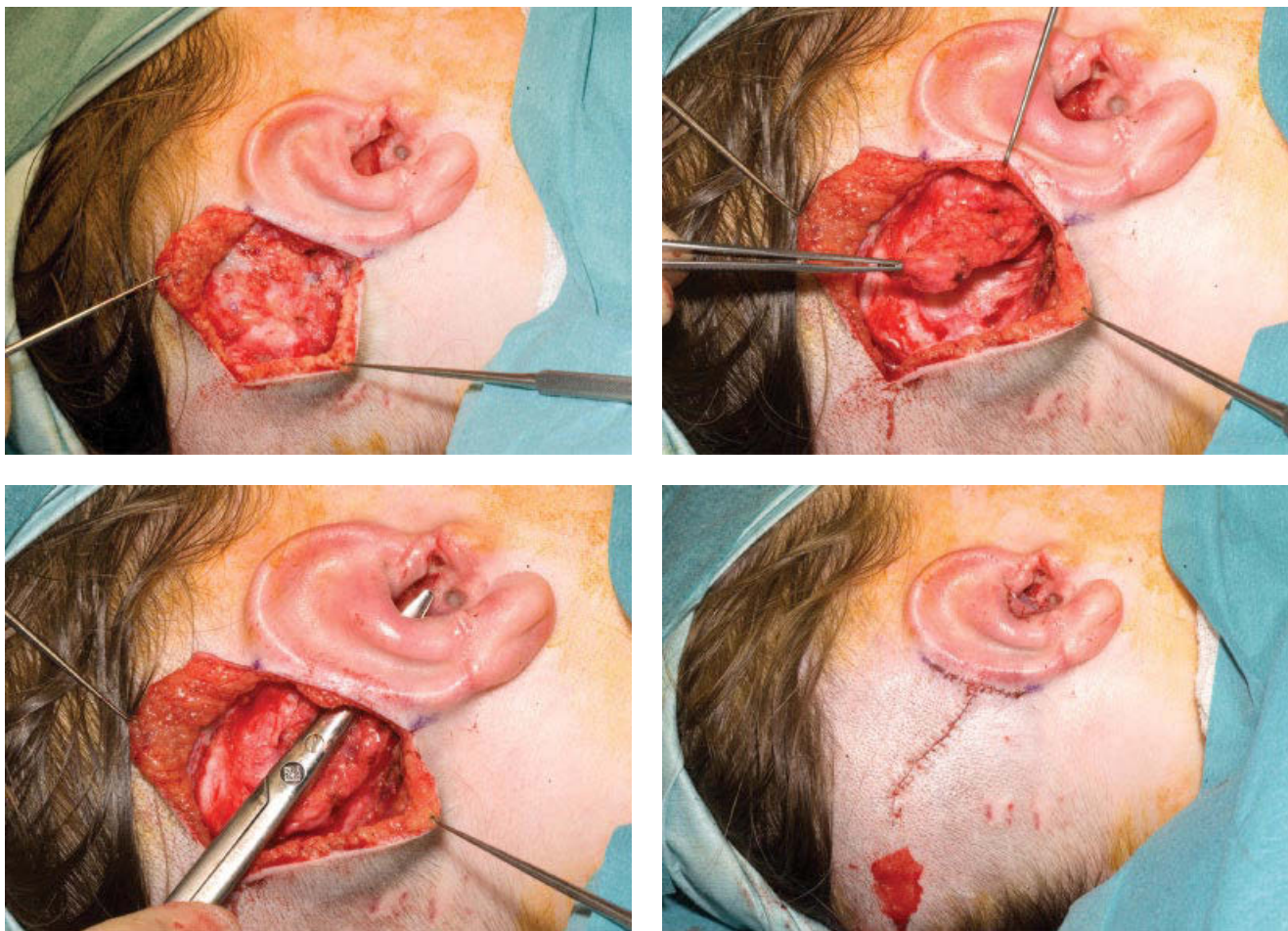
Alternative reconstructive options

The poor results achieved with autogenous cartilage in early reconstructive efforts encouraged others to find alternative options for framework fabrication. Popularized by Cronin *et al.*^{64,65} and refined by Ohmori *et al.*,^{66,67} Silastic was used as a preferred implant material for auricular reconstruction for over two decades. High rates of implant extrusion necessitating explantation, however, limited its continued use and costal cartilage, accredited largely to Tanzer, became the preferred framework material.

Although autogenous reconstruction is now considered the primary reconstructive option in the paediatric population, two other alternative options should be mentioned, which also preclude



(a)



(b)

Figure 19.12 (a) Patient with oculo-auriculo-vertebral spectrum who presented with right unilateral lobular microtia. Twenty-nine days following the first stage of reconstruction, she presented with total necrosis of skin overlying the concha. (b) Coverage of the area of exposed cartilage graft was restored with harvest of a random superficial mastoid fascial flap, which was tunnelled beneath the cartilaginous ear construct to reach the distal extent of the defect. The fascial flap was then grafted with a split-thickness skin graft, harvested from the scalp (skin graft donor site shown).

harvest of costal cartilage for auricular framework construction, namely, composite autogenous/alloplastic reconstruction and prosthetic reconstruction. The former consists of framework fabrication with porous polyethylene (Medpor) and coverage of the framework with a temporoparietal fascial flap, as popularized by Reinisch and Lewin.⁶⁸ The porous structure of this material has proved a major advantage over smooth silicone implants, providing improved adherence to surrounding tissues. Further, fascial flap coverage has decreased the rates of implant extrusion, providing more robust soft tissue support.

Prosthetic reconstruction in children is considered a salvage option following failed secondary autologous reconstruction where costal cartilage or soft tissue coverage remains insufficient for consideration of additional autogenous or alloplastic reconstruction. The latter option involves retention of the silicone auricular prosthesis with osseo-integrated titanium implants.

Finally, further advances in stem cell and tissue engineering to obtain a cartilaginous scaffold, may, in future, obviate the need for costal cartilage harvest and prove the ideal solution for framework fabrication.⁶⁹

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CHAPTER 20

Craniofacial clefts

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Craniofacial clefts encompass a wide range of deformities that is not easily delimited. Only seldom is a true clefting of both the soft tissue and underlying bone involved. One can therefore debate whether or not all diagnoses that are included in this chapter actually should be classified as a craniofacial cleft. Shedding more light on their pathogenesis will undoubtedly change the way we classify these conditions in the future. The occurrence rate of rare facial clefts is estimated to range from 1.43 to 4.85 per 100 000 births. In South America, Africa and Asia, the incidence of rare facial clefts appears to be higher than in Europe, the United States and Canada.

Aetiology

For an increasing number of craniofacial disorders, the genetic background is recognized (Table 20.1). For true midline clefts, it is more likely that a genetic factor is involved, and a crucial role appears to be played by the cranial neural crest cells during embryogenesis. Frontonasal dysplasia and hypertelorism appear to be mediated through excessive Hedgehog activity, which causes enhanced neural crest cell proliferation in the facial prominences.¹⁴ In Treacher Collins syndrome, on the other hand, haploinsufficiency of the protein *Tcof1* is shown to result in increased cell death of neural crest cells.¹⁵ For a significant number of disorders the pathogenesis is still unknown.

Classification

Multiple proposals have been made to create order in the broad spectrum of facial clefts. Tessier's topographic classification is the most frequently used anatomical classification.¹⁶ It differentiates 15 locations for clefts with the palpebral fissure as central landmark; cleft axes 0 (30) to 7 below the fissure represent the facial clefts, and clefts 8–14 above the fissure are referred to as the cranial clefts (Figure 20.1). The system includes a description

of the course of each cleft through both soft tissue and bone, which are rarely affected to the same extent.

Various other proposed classifications take the pathomorphogenesis of clefts into account; of these, van der Meulen's embryological classification is the most comprehensive. In this system the term 'dysplasia' is used since not all malformations are true 'clefts'. The dysplasias are classified according to time and location of developmental arrest.¹⁷

Hypoplasia of the mandible is graded according to the Pruzansky classification¹⁸ and as adapted by Mulliken and Kaban¹⁹ (Table 20.2).

Diagnostics

With the recent progress in three-dimensional ultrasonography and the discovery of pathogenic genes, prenatal diagnostics have gained importance.²⁰ Early recognition of the condition can optimize perinatal care (anticipation of airway problems), timely referral, and parents can be more specifically advised on expectations, treatment and outcome by an experienced team.

For a solid treatment plan, a three-dimensional computerized tomography scan is required for essential information on the condition and position of the osseous structures. An MRI is performed to evaluate intracranial structures, especially in median and paramedian clefts, and in case of suspicion of encephaloceles.

Management principles

General considerations

Surgical treatment of facial clefts is a major reconstructive challenge that is often underestimated. Frequently soft and hard tissues of multiple areas of the face are affected in various degrees of severity. This results in three-dimensional deformities with both aesthetic and functional impairment. In the majority of patients multiple cleft types exist and asymmetry

Table 20.1 Overview of the various types of craniofacial clefts, the causal genetic defect, type of inheritance pattern and incidence

Syndrome	Genetics	Inheritance	Incidence	Reference
Craniofrontonasal syndrome	<i>EFNB1</i>	X-D	1:120 000	1
Frontorhiny	<i>ALX3</i>	AR	Extremely rare	2
Midline cleft	<i>ALX1</i>	AR	Extremely rare	3
	<i>ALX4</i>	AR	Extremely rare	4
Treacher Collins	<i>TCOF1</i>	AD	1:50 000	5
	<i>POLR1C</i>	AD		6
	<i>POLR1D</i>	AD		6
Nager	<i>SF3B4</i>	AD	1:300 000	7
Stickler	<i>COL2A1</i>	AD	1:7500	8
	<i>COL9A1</i>	AD		9
	<i>COL9A2</i>	AD		10
Stickler/Marshall	<i>COL11A1</i>	AD		11,12
Velocardiofacial	22q11.2	AD	1:2000	13

X-D, x-linked dominant; AR, autosomal recessive; AD, autosomal dominant.

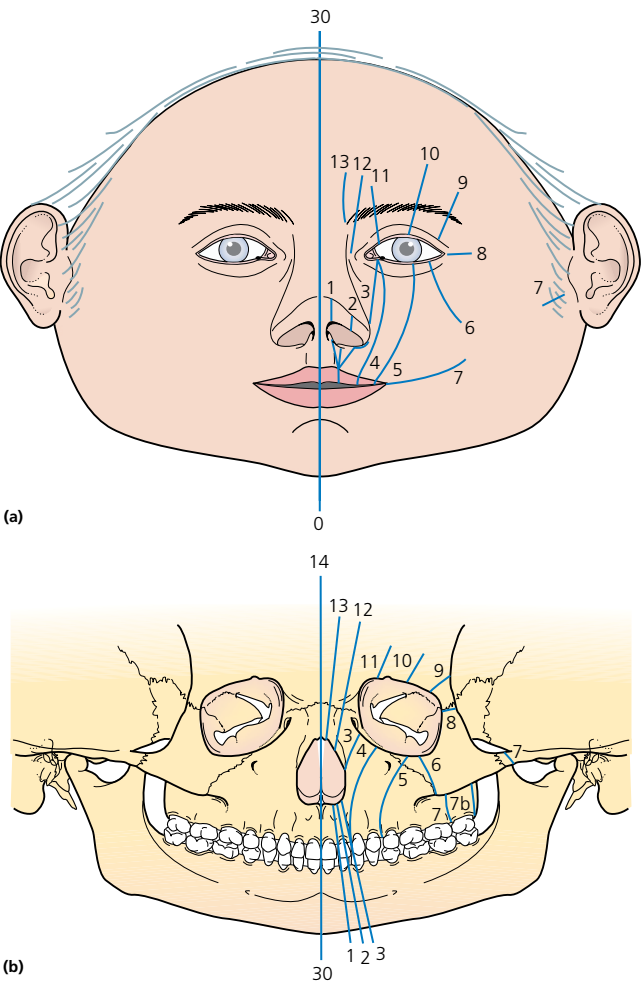


Figure 20.1 Classification of facial clefts according to Paul Tessier, regarding soft tissue (a) and bone (b). Source: Tessier *et al.*, 1976.¹⁶ Reproduced with permission of Elsevier.

Table 20.2 Pruzansky classification for mandibular hypoplasia

Pruzansky	TMJ	Ramus	Glenoid fossa
I	Small	Small	Normal
Ila	Hypoplastic, normal position	Hypoplastic	Hypoplastic
Ilb	Hypoplastic, malpositioned	Hypoplastic	Hypoplastic
III	Absent	Absent	Absent

TMJ, temporomandibular joint.
Source: Mulliken and Kaban, 1987.¹⁹ Reproduced with permission of Elsevier.

can be present. The goal of surgery is to achieve a good functional and aesthetic outcome.

Although these challenging facial deformities are appealing to many surgeons, patients with a facial cleft should be referred to a specialized centre. Even initial small corrections can result in extensive scarring, patchwork or insufficient reconstructions. Moreover, initial excellent results may deteriorate gradually over time due to intrinsic impaired growth or growth disturbance by surgical interventions; in addition mild deformities at birth can become more obvious over the years.^{17,21,22} Therefore, a long-term individual treatment plan by an experienced team is required to optimize end-results at adulthood and limit the number of operations.

Besides growth, the timing of surgery is also influenced by functional problems such as breathing, insufficient protection of the eye and feeding difficulties that require early intervention. Psychological aspects also play a role. ‘School-going age’ and teasing are often seen as a reason for early substantial reconstructions. However, research has shown that the intensity of the psychological burden is not directly related to the objective severity of the deformity.²³

Because of the rarity and enormous variability of facial clefts, it is impossible to establish standardized methods of treatment. However, certain general treatment principles apply as a foundation for each individual treatment plan.^{21,22,24–26}

- manage functional problems early;
- minimize scars and plan them within the borders of the facial units;
- avoid short-term solutions, and take the influence of growth into account;
- establish a good skeletal base for soft tissue coverage;
- attempt to restore the muscle layer by transposition and fixation of dystopic remnants;
- restore mucosal and conjunctival lining;
- use stable fixation to the skeleton for the cutaneous layer in order to optimize and preserve facial contour;
- preferably use techniques for skin coverage that can be reused (e.g. cheek advancement).

Airway management

Acute treatment of a patient with a (cranio)facial cleft is required whenever breathing is obstructed. In most cases, the breathing is normal while the child is awake, but gets obstructed when the child is asleep and the tongue is repositioned and the pharyngeal wall musculature relaxes. During sleep, repeated episodes of reduced (hypopnoea) or blocked breathing (apnoea) may occur, referred to as obstructive sleep apnoea (OSA). A polysomnography is the gold standard to obtain data on presence and severity of obstructive breathing. An upper airway endoscopy is advised in case of OSA to check for all possible sites of airway obstruction, ranging from septum deviation, choanal atresia, nasopharyngeal obstruction, obstruction at the tongue base level or cricoid stenosis.

Mild OSA can often be overcome by placing the baby in prone position. In some types of malformations there is a natural tendency to overcome the OSA, such as in Pierre Robin sequence, whereas in other types such as Treacher Collins syndrome this does not occur. Follow-up with repeated polysomnographies is therefore required for all patients to determine their individual breathing pattern over time and to adjust treatment as required. For newborn patients with severe OSA, a tracheotomy should be considered, as distraction at this very young age can be difficult given the cartilaginous aspect of the mandible. Securing the airway with a cannula allows time for the patient to age, which makes both the anaesthesia and surgery more predictable and safe. Distraction of the mandible in (very) young children has a higher risk of failure, will cause damage to teeth buds and will hamper future surgery which is very likely required. Most surgeons therefore tend to postpone distraction of the mandible until after the first year of age, and will only perform it if a decannulation seems feasible.

Special attention should be given to the palate, which may have a (submucous) cleft. If a cleft is present in a patient with other features of a (cranio)facial cleft, one should not apply the usual protocol regarding timing of palate closure. The chances

of inducing an upper airway obstruction is much higher and may require postponing closure of the palate until growth of the facial skeleton allows unhampered breathing. To determine safety of palatal closure, one can perform an overnight polysomnography with the patient wearing a custom-made palatal plate that mimics the closed palate. If this measurement shows no significant airway obstruction, repair of the cleft palate can usually be undertaken although one has to take into account that some swelling postoperative may temporarily cause additional obstruction. Close postoperative monitoring of the breathing pattern is thus required. Whenever severe OSA is present, one should be aware of the likelihood of associated swallowing and feeding difficulties.

Mandible

A true cleft of the mandible (type 30) is very rare and generally combined with a midline cleft of the lower lip and adherence of the tongue to the floor of the mouth.²⁷

In most cases of a craniofacial cleft with mandibular involvement, the mandible is hypoplastic on one or both sites. If breathing is not at risk, a mandibular distraction can be indicated to correct the occlusion and the facial asymmetry. There is no consensus at which age this should be undertaken and individual factors should be weighed. These factors are, for instance, the degree of asymmetry, problems with chewing, dental status and psychosocial considerations.

Maxilla

Midface hypoplasia is frequently encountered in craniofacial clefts. Depending on the type of cleft the hypoplasia may also be unilateral, causing severe facial asymmetry, particularly in the oblique facial clefts.²¹ If the degree of hypoplasia is significant, advancement with distraction may be indicated. For each patient an individual planning is made on whether a Le Fort I, II, III or a combination of, for instance, Le Fort I with advancement of the zygoma is required.

The orbits

Several abnormal positions of the orbits can be encountered in craniofacial clefts, particularly orbital dystopia and hypertelorism. Orbital dystopia refers to a difference in the vertical position of the orbits, with one of the orbits positioned higher or lower compared to the contralateral orbit. Oblique maxillary clefts often involve a bony defect of the orbital floor, combined with an intrinsic growth restriction in all directions of the maxilla. In unilateral cases, this impaired growth adds to the degree of orbital dystopia over time. Repair of the orbital floor with the insertion of a bone graft is usually undertaken at a young age, and may be combined with bone grafting of the maxillary cleft.²⁶ This grafting of the floor results in a partial correction of the dystopia. Definitive correction of orbital dystopia through a box osteotomy is usually delayed until the age of 6 years, as the osteotomies at the maxillary level are more likely to damage the tooth buds at a younger age.

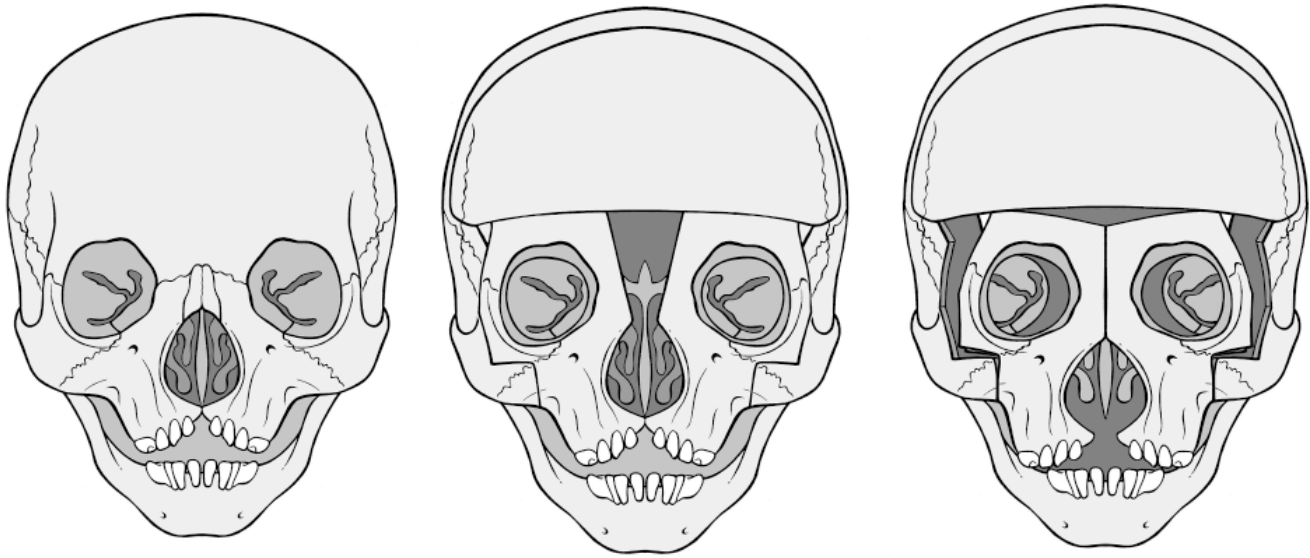


Figure 20.2 Facial bipartition to reduce interorbital distance and widen the alveolar arch.

In hypertelorism, the distance between both orbits is increased due to true lateralization of the orbits, which is commonly encountered in midline facial clefts. It should be distinguished from interorbital hypertelorism (or pseudohypertelorism) in which only the distance between the medial canthi is enlarged. Correction of hypertelorism can be undertaken with an orbital box osteotomy, or with a facial bipartition.²⁸ This last procedure simultaneously corrects the high-arched palate and V-shaped maxilla (Figure 20.2), resulting in a stable outcome in the long term.^{21,22} Following a facial bipartition, temporal depression is often encountered and this can in most instances be corrected with lipofilling. Timing of hypertelorism correction is controversial regarding the occurrence of relapse if performed before the age of 6. Several authors reported no relapse of hypertelorism,^{21,29} but initial undercorrection was a frequent finding. Others state that initial severe hypertelorism and correction under the age of 6 increases the chance of relapse.³⁰

In case of a severe microphthalmia or anophthalmia, early placement of serial orbital conformers should be considered to expand the orbital volume, followed by the insertion of a definite ocular prosthesis with or without a fat–dermis graft.

The eyelids

Semi-acute treatment concerns the protection of the eyes in case of an exposed cornea, which is particularly the case in patients with a large coloboma of the eyelid(s). A coloboma is a localized part of the eyelid characterized by hypoplasia of skin, muscle, tarsal plate, conjunctiva and eyelashes. Initially, the cornea may be protected with the use of lubricating gels and protective eye-wear, but within a few weeks an eyelid reconstruction is indicated. Local flaps for correction of colobomas give good initial results. However, only a minority remain stable over time, resulting in ectropion in the long term. A cheek flap provides good results, replaces like with like and can be reused when

necessary; this in contrast to other local flaps such as, for example, the forehead flap.²¹ Good positioning of the medial canthal tendon is necessary since it often displaces laterally and inferiorly.

The nasolacrimal system is often involved, with partial or complete deficiency in clefts involving the medial orbit. Dacryocystorhinostomia is performed at a later stage. Frequently secondary canthal and eye corner corrections are required.

The nose

A wide variety in presentation of nasal anomalies exist in facial clefts. Specific for midline clefts is the bifid nose. This deformity may be as mild as only a bifid tip of the nose, but may also involve a nose duplication, or even a true separation of both nasal halves in combination with hypertelorism, an encephalocele and absence of all midline structures. If the deformation is that severe, a facial bipartition is indicated which would also correct the widened nasal bridge with the removal of a significant amount of bone at the level of the glabella and the nasal bones. At the same time, a rib graft can be inserted to augment the nasal dorsum and columella as they often have insufficient support. Reduction of the nasal tip can also be done with limited dissection to remove the excess of soft tissue in between the medial crurae and suture the cartilage together to define the nasal tip.

Several options are available to correct the flattened nose, including using autologous material, particularly cartilage and bone, or using alloplastic material, such as silicone. Advantages of using autologous material include the reduced rate of infection, while a disadvantage is the donor site morbidity and resorption of the graft that might occur. Donor sites for autologous grafts are usually the ribs, the calvarium or the iliac crest. Whether or not cartilage should be used for the reconstruction remains unclear and is mostly determined by preference of the surgeon. In a comparative study between bone grafts no obvious

advantage for either graft type was detected regarding outcome or complication rate.³¹

In general, reconstruction is delayed until midfacial growth is nearly complete.³² In clefts with abundant tissue over the nasal dorsum, rearrangement of the skin should be considered with the use of an L-incision.³³ If the midline skin is severely dysplastic, direct excision of the affected tissue and a midline scar cannot be avoided.²⁶ Extensive correction of the nasal deformity, including the use of a forehead flap, should be postponed to adult age, and combined with correction of the nasal septum if so indicated. Prior to this surgery, one has to be certain that the maxilla is in the right position and if this is not the case, the advancement of the maxilla should precede any nasal reconstruction, as it influences tip projection.

In paramedian and oblique clefts a range of alar deformities (true clefting, hypoplasia, upward displacement, broadening, flattening) can be present, but also hemi- or total aplasia of the nose with or without a proboscis (rudimentary tubular 'nasal appendage'); the shape of piriform aperture can be altered or absent. Some use a proboscis for initial (hemi)nasal reconstruction. Since this proboscis consists of dysplastic soft tissue which lacks normal growth, it should be excised; otherwise it only contributes to unnecessary scarring.

Small alar defects can initially be managed with local flaps when sufficient soft tissue is present. Often they require secondary corrections for asymmetry or result in too much scarring.^{21,34}

Encephaloceles and craniosynostosis

In some types of craniofacial clefts, encephaloceles and/or craniosynostosis may be part of the malformation. The encephalocele is usually repaired within the first year of life with the use of a cranial bone graft to close the vault defect. Preoperative evaluation with MRI of the ventricular size and cerebrospinal fluid (CSF) collection at the site of the encephalocele is required and the insertion of a ventriculoperitoneal shunt may be indicated, as the persistent pressure of a CSF collection on a bone graft may cause its resorption.

The cheek

An oblique cleft through the cheek (type 3) is considered the most difficult to repair.¹⁶ Due to shortage of skin, a cheek rotation flap is indicated; it gives much better results than local closure and Z-plasties.^{21,26,35,36} Tissue expanders can be used in large defects and secondary reconstructions to achieve aesthetic subunit reconstruction. An attempt of reconstruction of the muscle layer should always be made,^{35,36} as is the case for macrostomia.³⁷

Associated syndromes

Frontonasal dysplasia

The group of frontonasal dysplasia is rather heterogeneous one. It includes the diagnoses of EFN1-mediated craniofrontonasal syndrome, constituting a distinct phenotype.¹ Discovery of the

ALX3 mutations separated another group of patients within the midline cleft, referred to as frontorhiny.² These patients are characterized by hypertelorism, wide nasal bridge, short nasal ridge, bifid nasal tip, broad columella, widely separated slit-like nares, long philtrum with prominent bilateral swellings, and midline notch in the upper lip and alveolus. ALX4 mutations were discovered in patients with a similar but very mild phenotype.⁴ In 2013, Uz showed mutations in ALX1 to result in an extreme presentation with bilateral microphthalmia, bilateral oblique facial clefts, complete cleft palate next to hypertelorism, wide nasal bridge and hypoplasia of the ala nasi.³

Another separate group within the frontonasal dysplasia cohort are patients with a basal encephalocele, who present with hypertelorism, a bifid nose tip, an incomplete midline cleft of the upper lip and a cleft palate with an encephalocele hanging down in the mouth through the skull base defect.²²

Treacher Collins syndrome

The syndrome is characterized by hypoplasia of the zygomatic arches, mandibular hypoplasia, beaked nose, lower eyelid colobomas or partial absence of eyelashes, downslanting of the palpebral fissures, dental anomalies, various degrees of microtia and atresia of the external auditory canal with anomalies of the middle ear ossicles, speech abnormalities, with or without a cleft palate.³⁸ The facial features present in a symmetric way. Treacher Collins syndrome is caused by mutations in the *TCOF1*, *POLR1D* and *POLR1C* genes and the inheritance pattern is autosomal dominant (Table 20.1).

Patients with Treacher Collins syndrome are highly likely to have OSA^{39,40} and should all be screened with a polysomnography.⁴¹ In a series of 50 patients, 4 required an immediate tracheostomy.⁴² If a cleft palate is part of the phenotype (in 23% in the paper by Thompson *et al.*⁴²), special attention to the breathing pattern should be given prior to closing the palate. Hardly any normal growth of the mandible takes place, prior to or following mandibular distraction.⁴³ There are several options to correct the bony deficiency of the malar complex. Most surgeons prefer autologous bone over implants, especially in children and particularly given the poor soft tissue coverage. Most commonly, cranial bone is used to augment the orbitozygomatic complex and this can be either taken as a pedicled graft on the superficial temporal artery⁴⁴ or as a free graft.⁴⁵ In the series reported by Thompson *et al.*,⁴² no difference in survival of the bone graft was seen between the pedicled and the free grafts. The characteristic nasal deformity may be corrected at an adult age, after completing maxillary advancement if indicated.

Reconstruction of the lower eyelid coloboma and the low position of the lateral canthus is performed with a laterally based flap of the upper eyelid. Jackson⁴⁶ suggested taking a full-thickness flap from the upper eyelid, including tarsus and conjunctiva, while completely releasing the retraction of the lower eyelid. In milder cases, a musculocutaneous flap lined with a mucosal graft is usually sufficient to augment the lower eyelid skin. Fullness to the lower eyelid and upper part of the cheek can be provided through lipofilling.

Most patients with Treacher Collins have a significant hearing deficit based on hypoplastic middle ear cavities and ankylotic or missing ossicles.⁴⁷ Therefore, a bone-anchored hearing aid (BAHA) is often required. The planning of external ear reconstruction and placement of a BAHA should be coordinated so as to not compromise outcome of the two interventions.

Nager syndrome

The typical craniofacial features of Nager syndrome are severe micrognathia, malar hypoplasia, eyelid anomalies such as coloboma and downward slanting, mild anomalies of the external ear, and a low hairline. These anomalies present in a symmetric way. A cleft palate can be present. Functionally, hearing⁴⁸ and breathing are commonly impaired and often a tracheotomy is required. Characteristically, hand anomalies such as hypoplasia of radius and ulna, radioulnar synostosis, thumb aplasia and clinodactyly are present and make the distinction from Treacher Collins syndrome.

The severe hypoplasia of the mandible does not improve in time and thus surgical correction is indicated to improve breathing. Several papers report successful mandibular distraction, allowing the removal of tracheotomies in some patients, but usually not in all.⁴⁹ If a class I occlusion is achieved, an additional genioplasty distraction with hyoid advancement may further improve sleep studies and appearance.⁵⁰

Oculo-auriculo-vertebral spectrum, or hemifacial microsomia

The phenotype of hemifacial microsomia (HFM) is very wide, ranging from a preauricular skin tag and minor asymmetry of the mandible to the full-blown presentation with orbital dystopia, severe asymmetry of the mandible and absence of the temporomandibular joint, soft tissue deficiency, facial nerve palsy, microtia, hearing loss, cleft lip/palate and macrostomia. The various features of HFM are graded in the OMENS classification (orbit, mandible, ear, cranial nerves and soft tissues) with 0 for normal to 3 for severely affected.⁵¹ Facial nerve dysfunction was reported to occur in 22% of HFM patients,⁵² while 10% have an associated cleft lip/palate and 23% macrostomia.⁵³ The malformation has an incidence of 1 in 5000 and usually occurs unilateral but may be present bilaterally in approximately 10–15% of patients. Other frequently encountered congenital malformations are skeletal, cardiac, central nervous system, gastrointestinal, renal, and pulmonary disorders,⁵⁴ or dental anomalies.⁵⁵

The spectrum of oculo-auriculo-vertebral anomalies also includes the Goldenhar syndrome, the phenotype of which is identical to that of hemifacial microsomia in combination with anomalies of the cervical spine and an epibulbar dermoid. The dermoids are usually only removed whenever they interfere with visual acuity.⁵⁶

Children with HFM are more likely than otherwise healthy children to have symptoms of sleep-disturbed breathing.⁵⁷ Since the introduction of distraction of the mandible by McCarthy

et al.,⁵⁸ this is the primary choice of treatment for correcting the hypoplastic side of the mandible. Although adequate lengthening can be achieved, the width of the lower face often remains impaired. This deformity can either be corrected by using autologous bone grafts or with an implant. The discussion on optimal timing for mandibular distraction, however, continues. Early distraction has a higher rate of complication and may be associated with the necessity to repeat surgery at a late age. A critical review of literature clearly showed a lack of evidence to substantiate long-term effectiveness of early mandibular distraction.⁵⁹ To reduce the rate of repeat surgery, overcorrection until the chin points to the non-affected side is recommended. Only the very severely affected mandibles require reconstruction with a free flap,⁶⁰ or the insertion of a rib graft with secondary distraction of the graft or the native mandible.³⁰ Reconstruction of the temporomandibular joint in the class III deformity is a major challenge, with often disappointing results regarding mouth opening. In the severe types of malformations, usually a rib graft is inserted with a cartilaginous part facing the skull base and the interposition of soft tissue (temporalis fascia flap or buccal fat pad) to prevent ankylosis. Implants are in general not applicable in these severely affected patients.

After correcting the skeletal deformity, a significant soft tissue deficiency usually remains. The options for its correction are either serial procedures of lipofilling or a free flap. In a comparative study between both techniques in patients with comparable deformities, a mean number of two sessions for the free flap group (microsurgical revisions and secondary corrections) and four sessions of lipofilling were required.⁶¹ Downside of using a free flap is the donor site morbidity, difficulty in the acceptor site due to previous surgery, and the tendency of the flap to sag. In this group there was an overcorrection of the volume. In the lipografted patients there was a 83% fat take and an almost symmetrical result. Given the lower complication rate and good results, lipofilling is an excellent way to correct the soft tissue deficiency.

Binder

Patients present with the characteristic features of nasomaxillary hypoplasia with flattening of the nose. On examination, the cartilaginous septum is missing and there is hypoplasia of the nasal bones, the anterior nasal spine and pyriform apertures. These anomalies appear not to induce difficulties with breathing. In general, there is no shortage of soft tissue but extensive undermining is required to reduce the mechanical restraints on the graft.⁶²

Good results with various techniques using rib are presented by Chiang *et al.*⁶³ and similarly by Goh and Chen³² who not only restored the nasal dorsum with a strut for the columella, but also augmented the pyriform apertures.

Parry-Romberg syndrome or hemifacial atrophy

Parry-Romberg is characterized by a progressive disappearance of the subcutaneous fat in one half of the face, and may also involve the skin, muscle, cartilage and bone. Patients are more

likely to have neurological abnormalities, such as epilepsy.⁶⁴ Involvement of the skin can present as hyperpigmentation, depigmentation or alopecia of the scalp. The syndrome is often related with linear scleroderma en coup de sabre.⁶⁵ The process usually starts in the first or second decade of life and then stops after several years. Reconstruction is only undertaken when one is sure that there is no more progression of the disease. In the very severe cases, both bony and soft tissue reconstruction is performed, whereas in the mild cases only lipofilling in several sessions may be sufficient.⁶⁶

(Pierre) Robin sequence

There is a large discrepancy in diagnosis of (Pierre) Robin sequence, which makes interpretation and comparison of the numerous studies difficult.⁶⁷ The most generally accepted criteria for Pierre Robin sequence are mandibular hypoplasia, glossoptosis and breathing difficulties. In over 80% of the cases a cleft palate is present, which is generally U-shaped. The isolated Robin sequence is distinguished from the syndromic form, in which the triad is part of, for instance, Stickler syndrome, velocardiofacial syndrome or Treacher Collins syndrome.

To overcome the obstructed breathing, prone positioning is usually sufficient and within several months' time, sufficient mandibular growth resolves the problem. For the minority of patients in whom breathing is insufficient despite prone positioning, the options can be tongue–lip adhesion, nasopharyngeal airway, non-invasive respiratory support, tracheostomy and mandibular distraction. The tongue–lip adhesion has been presented as a successful procedure,⁶⁸ although not routinely advocated or used in several craniofacial centres. Treatment for several months with a nasopharyngeal airway solved the breathing problems in all patients in the London team, obviating any surgical procedure.⁶⁹ Non-invasive respiratory support has very similar results.⁷⁰

The long-term effectiveness of mandibular distraction in patients with Pierre Robin sequence is not proven, as is the case for this procedure in hemifacial microsomia. Numerous case reports are presented to demonstrate its good outcome, but it should be kept in mind that most patients do not require this procedure if given the time to allow for mandibular growth. Distraction should be reserved for those patients with a tracheostomy, an obstruction only at the tongue–base level, and adequate bone quality to allow placement of the distraction device.⁷¹ Decannulation can not always be achieved, however.⁷²

Stickler syndrome and Marshall syndrome

The phenotype in these syndromes consists of ocular (particularly myopia), orofacial (flat midface and cleft palate) and hearing anomalies. Hearing loss is particularly sensorineural and to some degree conductive.⁷³ Given the high risk of retinal detachment, close follow-up by an ophthalmologist is required.

Velocardiofacial, Shprintzen or 22q11.2 syndrome

The velocardiofacial (VCF) syndrome is most commonly encountered in patients with syndromic cleft palate. The facial characteristics encountered are overfolding of the helix and cup ears, prominence of the nasal radix, narrow alar base and bulbous nose tip, fullness of the upper eyelids, malar flattening, elongated lower face height, retrognathia and facial asymmetry.⁷⁴ The syndrome is also associated with developmental disabilities, intellectual disability and a high risk of psychiatric disorders.⁷⁵ Many patients have a mild to moderate immune deficiency, and the majority have a cardiac anomaly. Patients with VCF are likely to have medially displaced internal carotid arteries, which is a risk during pharyngeal flap dissection. In a comparative study, this anomaly was encountered in 44% of VCF patients and in 14% of controls.⁷⁶ The course of the cervical part of the internal coronal artery was irrelevant, however, to the design of the pharyngeal flap and thus preoperative magnetic resonance angiography (MRA) was no longer recommended.

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CHAPTER 21

Craniosynostosis

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History

Hippocrates provided the first description of craniostenosis in 100 BC, noting the variation of skull deformities and correlating them with patterns of cranial suture involvement.^{1,2} There are many historical descriptions of people with unusual head shapes.^{1,2} The Greek statesman and general Pericles had an unusually long narrow head shape and was nicknamed 'Squill Head'. It is possible he had sagittal synostosis. In *The Iliad*, Homer described a man called Thersites whose head was pointed at the top. Aristophanes and Galen both used the term *oxycephalus* for 'tower head'.²

The term *craniosynostosis* was first used by Otto in 1830³ to describe the entity of premature suture fusion. Virchow described how in medieval Saxony, people believed that such children were thought to be changelings or monsters and that it was the devil who made them deaf, dumb and blind.^{4,5} Virchow examined skulls from various museums and published a classification in which he correlated synostosis of specific cranial sutures with head shape.^{2,4,5} What has become known as Virchow's law is that premature fusion of a cranial vault suture (craniosynostosis) inhibits skull growth perpendicular to the fused suture and compensatory overexpansion takes place at patent sutures.^{2,4,5}

The general direction of growth after synostosis is parallel to the fused suture. For example, when synostosis of the sagittal suture occurs, growth proceeds in an anteroposterior direction, leading to scaphocephaly and a long, narrow, boat-shaped skull (Figure 21.1).

Skeletal development

The developing skull is divided into the neurocranium, which surrounds the brain, and the viscerocranium, which develops into the facial bones. The neurocranium is made up of bones that ossify directly, the membranous neurocranium which forms the skull vault, and bones that undergo endochondral

ossification and form the skull base.⁶ The flat bones of the skull grow by the deposition of new bone material at their margins. In their growth phase, these bones do not fuse but remain separated by specialized structures, the cranial sutures. Skull growth is needed to accommodate the size of the growing brain and depends on an exchange of signals between mesenchyme, the osteogenic front and the dura mater (for review, see Slater *et al.*⁷ and Striker and Mundlos⁸).

Normal calvarial growth

Normal growth of the calvarium is complete at about age 2 years, the orbit at age 6 years, and the rest of the facial skeleton at about age 16–17 years. Brain growth drives the enlargement of the skull in infancy. The expanding brain doubles in size in the first six months of life and then doubles again in the second six months and doubles again between 12 and 24 months. The expanding brain creates separating tensional forces in the cranial sutures. The cranial sutures allow for skull deformation during birth and expansion during brain growth. The metopic suture normally starts to close at the age of 2 years. The sagittal suture begins to close after the age of 22 years, the coronal suture after 24 years, and the lambdoid after 26 years. It is within the normal range for them not to fully close until the age of 40 years.⁶

Craniosynostosis: overview

Craniosynostosis, or the premature fusion of one or more cranial sutures, occurs in approximately one in 2000–2500 live births.⁹ It usually results in a change in head shape, and can have functional consequences. Sagittal synostosis is the most common type and is seen in 40–55% of non-syndromic cases.⁹ Coronal synostosis is the second most common (20–25%), followed by metopic synostosis (5–15%); lambdoid synostosis is rare (>5%).⁹ More than one suture is affected in 5–15% of cases.⁹ Recent studies have shown that there has been an increase in the absolute numbers of patients with sagittal and metopic synostosis in the last decade.^{10–14} Metopic synostosis is also becoming

Scaphocephaly

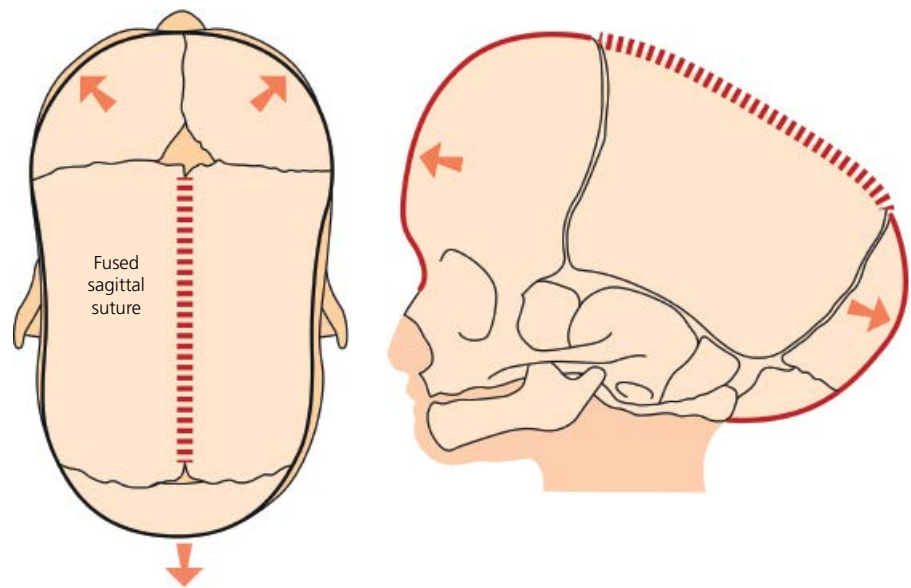


Figure 21.1 Skull growth in sagittal synostosis causes scaphocephaly.

more common.^{10,12,15} Craniosynostosis usually occurs as an isolated condition but can also be associated with more than 160 syndromes.¹⁶ Physical findings can include calvarial and facial dysmorphism, midface hypoplasia, hydrocephalus, deafness, blindness, mental retardation and extremity anomalies.

Classification of craniosynostosis

Primary

- Non-syndromic: sagittal, coronal, metopic, lambdoid, multi-suture
- Syndromic: Crouzon, Apert, Pfeiffer, Saethre–Chotzen, etc.

Secondary

- Metabolic disorder (e.g. hyperthyroidism)
- Malformations (e.g. holoprosencephaly, microcephaly, shunted hydrocephalus, encephalocele)
- Exposure of fetus to teratogenic drugs (e.g. sodium valproate, phenytoin, retinoic acid)
- Mucopolysaccharidosis (e.g. Hurler syndrome, Morquio syndrome)

Molecular mechanisms involved in craniosynostosis

There is good evidence that the primary pathology in craniosynostosis is within the dura mater and not within the bone.^{17–20} Cranial suture fusion and maintenance of patency are dependent on the interplay of transcription factors, cytokines, growth factor receptors, and extracellular matrix molecules. It is now known that the majority of the craniosynostosis syndromes are caused by mutations in genes encoding FGFR-1 (fibroblast growth factor

receptor-1), FGFR-2 and FGFR-3, and the transcription factors TWIST (upstream regulator of FGFRs) and MSX2.^{21,22} Gain-of-function mutations are associated with the human *MSX2* and *FGFR* genes, whereas loss of function is associated with the *TWIST* gene^{21,22} (for a review see Slater *et al.*⁷). Syndromic craniosynostoses exhibit variable clinical and genetic heterogeneity. Recent advances in molecular genetics have led to a discovery of other genes implicated in different craniosynostosis syndromes^{23,24} (Table 21.1).

Mutations in *FGFR2* are the prevalent cause for three overlapping syndromes: Apert syndrome (MIM #101200), Pfeiffer syndrome (MIM #101600) and Crouzon syndrome (MIM #123500).^{23,25–32} These syndromes are characterized by craniosynostosis and varying limb malformations (i.e. midface hypoplasia and complex syndactyly in Apert syndrome, midface hypoplasia, broadened thumbs and big toes, and variable cutaneous syndactyly in Pfeiffer syndrome and facial but no limb abnormalities in Crouzon syndrome).

Parameters of care for craniosynostosis

A multidisciplinary meeting was held in March 2010 in Atlanta, Georgia with the goal of creating parameters of care for individuals with craniosynostosis from the current state of knowledge.^{33–35} These helpful documents set out a team-based approach to the diagnosis, management and treatment of craniosynostosis. Interdisciplinary craniofacial teams are commonly composed of members with expertise in anaesthesia, craniofacial surgery, dentistry, genetics, hand surgery, intensive care, neurosurgery, nursing, ophthalmology, oral and maxillofacial surgery, orthodontics, otolaryngology, paediatrics, prosthodontics, psychology,

Table 21.1 Common gene mutations in craniosynostosis

Syndrome	Causative gene	Inheritance	Clinical features	OMIM reference
Apert syndrome	<i>FGFR2</i> (10q26.13)	Autosomal dominant	Craniosynostosis (usually bicoronal), midface hypoplasia, shallow orbits, beaked nose, hearing loss, complex acrosyndactyly of hands and feet	MIM #101200
Crouzon syndrome	<i>FGFR2</i> (10q26.13)	Autosomal dominant	Craniosynostosis (usually bicoronal), midface hypoplasia, shallow orbits, hearing loss	MIM #123500
Crouzon syndrome with acanthosis nigricans	<i>FGFR3</i> (4p16.3)	Autosomal dominant	Craniosynostosis (usually bicoronal), midface hypoplasia, shallow orbits, hearing loss, acanthosis nigricans	MIM #612247
Pfeiffer syndrome	<i>FGFR1</i> (8p11.23–p11.22) <i>FGFR2</i> (10q26.13)	Autosomal dominant	Craniosynostosis (usually bicoronal), midface hypoplasia, shallow orbits, hearing loss, broad radially deviated thumbs/great toes, partial syndactyly of fingers and toes	MIM #101600
Muenke syndrome	<i>FGFR3</i> (4p16.3) specific mutation Pro250Arg (P250R)	Autosomal dominant	Craniosynostosis (usually bicoronal or unicoronal), midface hypoplasia, hearing loss, variable and low penetrance limb malformations	MIM #602849
Saethre–Chotzen syndrome	<i>TWIST1</i> (7p21.1); <i>FGFR2</i> (10q26.13)	Autosomal dominant	Craniosynostosis (usually bicoronal), syndactyly, congenital heart defects, low frontal hairline, ptosis, small ears with prominent horizontal crurae	MIM #101400
Antley–Bixler syndrome without genital anomalies or disordered steroidogenesis	<i>FGFR2</i> (10q26.13)	Autosomal recessive	Craniosynostosis (usually bicoronal), radiohumeral synostosis, osteodysgenesis, long bone fractures	MIM #207410
Antley–Bixler syndrome with genital anomalies and disordered steroidogenesis	<i>POR</i> (7q11.23)	Autosomal recessive	Craniosynostosis (usually bicoronal), radiohumeral synostosis, genitourinary anomalies, adrenal insufficiency and Addisonian crisis	MIM #201750
Greig cephalopolysyndactyly	<i>GLI3</i> (7p14.1)	Autosomal dominant	Frontal bossing, scaphocephaly, hypertelorism, pre- and postaxial polydactyly and variable syndactyly. Variable phenotype and can include craniosynostosis	MIM #175700
Craniofrontonasal syndrome	<i>EFNB1</i> (Xq13.1), genetic heterogeneity	X-linked	Paradoxically greater severity in heterozygous females than in hemizygous males. Females have frontonasal dysplasia, craniofacial asymmetry, craniosynostosis, bifid nasal tip, longitudinally grooved nails, wiry hair, and abnormalities of the thoracic skeleton, whereas males typically show only hypertelorism	MIM #304110
Carpenter syndrome	<i>RAB23</i> (6p11.2)	Autosomal recessive	Acrocephaly with variable synostosis of the sagittal, lambdoid, and coronal sutures; peculiar facies; brachydactyly of the hands with syndactyly; preaxial polydactyly and syndactyly of the feet; congenital heart defects; growth retardation; mental retardation; hypogenitalism; and obesity. Cerebral malformations, oral and dental abnormalities, coxa valga, genu valgum, hydronephrosis, precocious puberty, hearing loss	MIM #201000
Beare–Stevenson cutis gyrata	<i>FGFR2</i> (10q26.13)	Autosomal dominant	Craniosynostosis, cloverleaf skull. Cutis gyrata variably affects scalp, forehead, face, preauricular area, neck, trunk, hands, and feet. Acanthosis nigricans	MIM #123790

Source: Upton J. Apert syndrome. Classification and pathologic anatomy of limb anomalies. *Clinics in Plastic Surgery* 1991;18(2):321–355. Reproduced with permission of Elsevier.

public health, radiology, social work and speech–language pathology. Ideally, care for patients with craniofacial anomalies is best provided in craniofacial centres by a dedicated team of specialists who see a sufficient number of affected patients to understand the management of complexities. It is important to assess a child with craniosynostosis early and this should begin perinatally.

Prenatal diagnosis

Cranial sutures form at about 16 weeks' gestation. Antenatal ultrasound scans are commonly performed at 20 weeks' gestation and therefore there has not been enough time for growth distortion of the skull to have occurred except in the most severe cases.²³ Most patients with craniosynostosis are not detected during pregnancy.

Craniofacial assessment

Initial history and examination from birth to age

3 months

The aims are: (a) to determine whether craniosynostosis is present; (b) to determine whether there are additional features suggesting an associated syndrome and (c) to assess whether urgent or elective management is required. Craniosynostosis is very heterogeneous in its causes and presentation, and therefore in its management. Most isolated non-syndromic cases present electively, but a minority of syndromic cases present acutely and require immediate intervention.

Questions should be asked about any identifiable causes, such as intrauterine constraint (primiparity, multiple pregnancy, abnormal lie, oligohydramnios),³⁶ teratogenic exposure (e.g. maternal use of oral retinoids, sodium valproate or phenytoin during pregnancy), family history of abnormal head shape and genetic abnormalities. Questions should include whether the conception was normal or assisted, whether the pregnancy was normal, birth history and birth weight. The child's current weight and birth weight should be plotted on a growth chart. Questions must be asked about the airway, feeding, eye protection and elevated intracranial pressure (ICP). It is important to look for evidence of breathing difficulty, choking or vomiting on feeds, failure of eyelids to cover eyes during sleep, or irritability, as these may be indications for acute intervention.

The most common presentation is with an unusual head shape. The head may be long and narrow (scaphocephaly, dolichocephaly), triangular at the front (trigonocephaly), broad and flattened (brachycephaly) or skewed (plagiocephaly). The clinician should examine the hands and feet, looking for congenital anomalies, for example, a broad, radially deviated thumb or big toe in Pfeiffer syndrome, complex syndactyly in Apert syndrome and longitudinally split nails in craniofrontonasal syndrome. The face should be examined for dysmorphic features, including hyper- or hypotelorism, exorbitism, midface hypoplasia, facial asymmetry, ear size, ear position and shape. The skull shape should be examined from the front, back, sides and top. The sutures should be palpated for ridging (associated

with suture fusion). The size, shape and tension of the fontanelles should be assessed. The head circumference should be measured and plotted on the growth chart. The cephalic index (ratio of maximum breadth to maximum length of skull, normally between 0.76 and 0.83 (in boys) or 0.84 (in girls)) provides an objective measurement and requires calipers. Examine the mouth for a cleft palate and a complete general physical examination, including that of the cardiovascular system. Patients should also have an eye evaluation with a fundoscopic examination and visual evoked potentials (VEPs) can be useful.^{37,38} The patient should be reviewed by the genetics team, paediatricians, audiology and ENT. Hearing problems are common. The speech and language team should assess feeding and swallowing. Clinical 2D (and if possible 3D) photographs and further radiological imaging are performed as necessary. The parents should be assessed to see if they have any features suggestive of craniosynostosis.

Deformational posterior plagiocephaly

Craniosynostosis must be differentiated from deformational plagiocephaly, which has become increasingly common after the American Academy of Pediatrics Task Force on Infant Positioning and Sudden Infant Death Syndrome (SIDS) recommended in 1992 that healthy infants be positioned on the back or side, when the child is laid to sleep.³⁹ This 'Back to Sleep' campaign resulted in a dramatic decrease in the incidence of SIDS from 1.2/1000 live births in 1992 to 0.56/1000 in 2001.^{40–42} The skull of a newborn baby is malleable and the back of the head can flatten and cause associated changes in the head shape, which include ipsilateral occipitoparietal flattening on the side where the child tends to lie and apply pressure to the back of the head, ipsilateral frontal bossing and ipsilateral anterior displacement of the ear and contralateral occipital bossing (Figure 21.2).

This deformational plagiocephaly (twisted skull shape) needs to be differentiated from plagiocephaly due to other causes, which can include anterior synostotic plagiocephaly due to unicoronal synostosis, or posterior synostotic plagiocephaly due to lambdoid synostosis (Figure 21.3). Other causes of deformational plagiocephaly include intrauterine constraint (e.g. twin pregnancy), assisted delivery (e.g. forceps) and prematurity. If a child has a deformational plagiocephaly, the skull is often parallelogram shaped when viewed from above, whereas a child with a unilateral synostosis has a failure of growth on one side of the head and so the head shape will tend to be trapezoidal shaped (Figure 21.3).

In a child with a deformational plagiocephaly, the distance from the lateral canthus to the root of the helix will be the same on both sides. In a child with a unicoronal synostosis, this distance will be shorter on the affected side due to a lack of growth. In clinical practice, difficulty most commonly arises in differentiating positional plagiocephaly from lambdoid synostosis, because the changes in orbital shape and forehead symmetry are quite distinctive in unicoronal synostosis. An anteroposterior and lateral skull X-ray can be helpful to exclude a closed cranial suture. If doubt remains, referral to a specialist craniofacial unit is recommended.

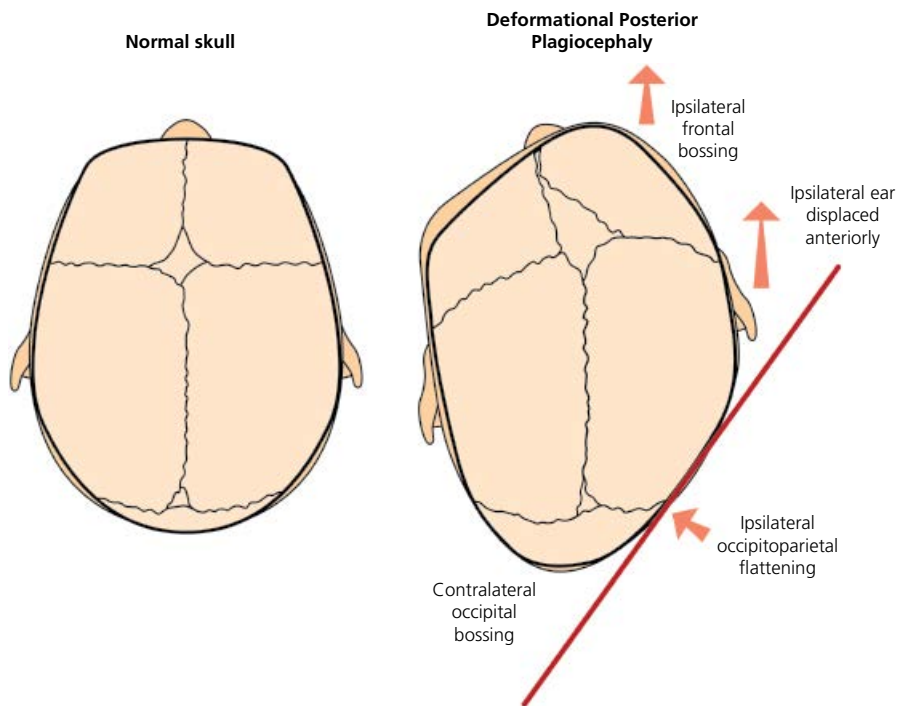


Figure 21.2 Deformational posterior plagiocephaly.

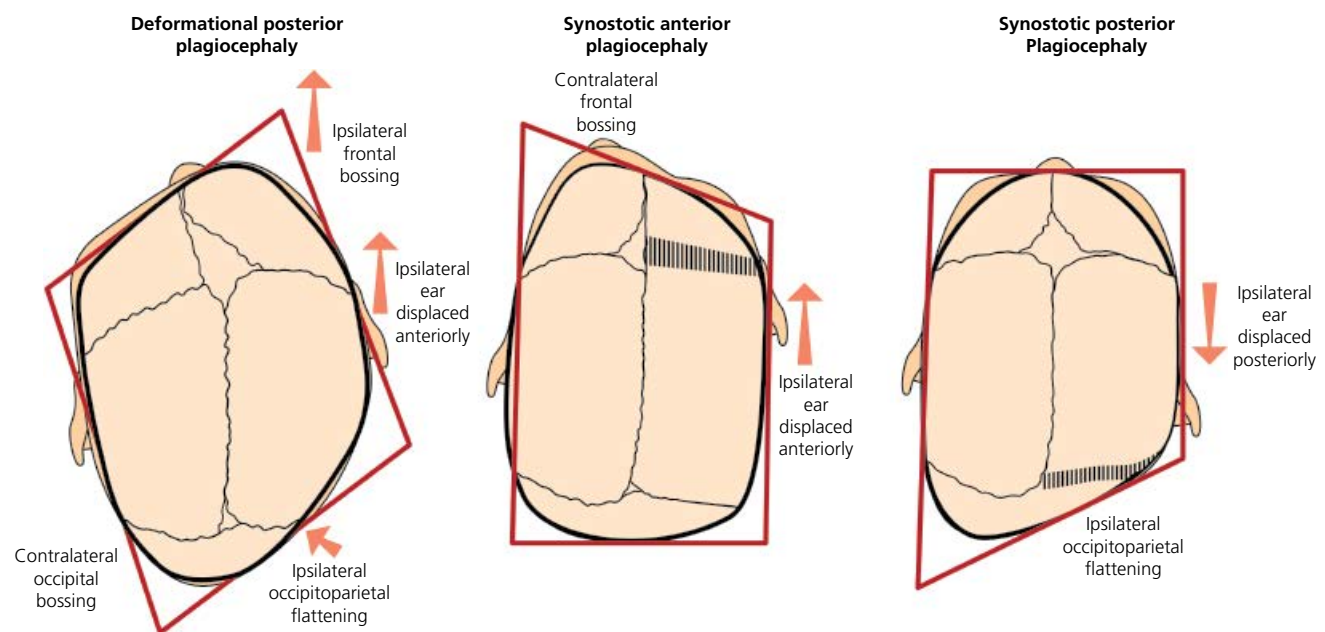


Figure 21.3 Comparison of deformational posterior plagiocephaly and unicoronal craniosynostosis, which causes synostotic anterior plagiocephaly, and unilateral lambdoid synostosis, which causes synostotic posterior plagiocephaly.

Children with deformational plagiocephaly can be initially managed conservatively by turning the head, using supportive pillows, and advice about spending at least 30 min per day of 'tummy time' while the child is awake.⁴² Children should not be kept in one position for a long time, for example in a car seat or carry cot. For infants who fail to improve with repositioning by six months of age and who continue to have severe deformity,

another option is to use a moulding helmet or cranial orthosis.⁴³ The use of moulding helmets is controversial and expensive. For a review of the management of deformational plagiocephaly see Robinson and Proctor⁴³ and Losee and Mason.⁴⁴ There is some evidence that there is an increased prevalence of mild developmental delay in motor development in children with deformational plagiocephaly.^{45–48}

Radiographic imaging

Computed tomography

In cases of single suture synostosis and non-syndromic craniosynostosis, CT scanning and three-dimensional reconstruction using both bone and soft tissue windows is the most useful investigation to examine the patency of each cranial suture.⁴⁹ In patients with complex multisuture craniosynostosis or syndromic craniosynostosis, MRI can be useful to examine the intracranial anatomy in more detail, but it is less good at visualizing the cranial sutures. The brain should be examined to look for anatomical abnormalities (e.g. ventriculomegaly, Chiari malformation, syrinx, agenesis of the corpus callosum) and to assess craniocerebral disproportion.

Sagittal synostosis

This is the commonest of the craniosynostoses (40–60%). Nearly 80% of the sagittal synostoses are non-syndromic and 6% run in families with dominant transmission. It is commoner in males (3.5 : 1), and the prevalence is nearly one in 5000 children. There is growth inhibition in a perpendicular direction to the affected suture leading to a skull shape that is elongated in an anteroposterior direction, causing a boat-shaped appearance (scaphocephaly). There is sagittal ridging, frontal bossing, and a wide interorbital distance due to the unrestricted growth of the patent coronal and metopic sutures. The unrestricted growth at the lambdoid suture causes occipital bulging. The skull base is not involved. Patients with scaphocephaly can present with other clinical signs, such as cerebral palsy, psychomotor retardation, or neurological signs. Elevated ICP is seen in 7–13% of the cases. 3D CT shows suture absence, which may be focal or generalized. It can also be associated with fusion of the metopic suture⁵⁰ (Figure 21.1).

Metopic synostosis

The suture closure starts in the third trimester at the level of the glabella moving upwards towards the anterior fontanelle, and is usually fused by the ninth month, but it may take up to 2 years to fuse. Premature fusion of the metopic suture causes a triangular-shaped head or trigonocephaly (Figure 21.4). There usually is associated hypotelorism. An omega-shaped notch is commonly seen on an axial CT scan in metopic synostosis and this notch is not seen in normal patients. Patients with metopic synostosis can also have midline and forebrain abnormalities. Nearly 75% of the cases identified postnatally are isolated, with 25% being syndromic. Associated syndromes include Jacobsen/11q23 deletion, chromosome 9p deletion, and Opitz C syndrome.

Unicoronal synostosis

This accounts for around 20–30% of craniosynostosis and occurs slightly more often in girls than boys. It was thought to be non-syndromic until the association of the *TWIST1* and Pro250Arg *FGFR3* gene mutations were discovered in some cases.⁵¹ There is

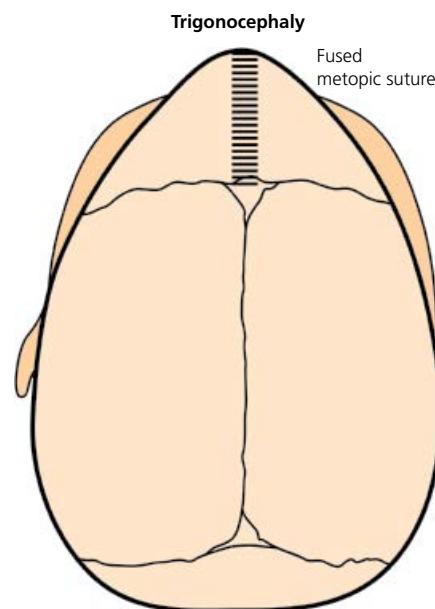


Figure 21.4 Metopic synostosis causing trigonocephaly.

reduced growth on the affected side, which results in flattening of the ipsilateral frontal and parietal bones. There is posterior displacement of the calvarium and resultant superior displacement of the ipsilateral orbital roof and the lesser wing of the sphenoid bone. This results in the 'Harlequin' deformity of the orbit. There is compensatory bulging of the frontal and temporal regions on the side opposite the deformity (Figure 21.3) with anterior displacement of the sphenoid wing and ipsilateral petrous ridge.

Bicoronal synostosis

This is commonly seen in syndromic craniosynostosis and patients present with brachycephaly. The forehead is large and flat. Bicoronal synostosis is commonly seen in with Pro250Arg *FGFR3* gene mutations, where it frequently presents as an isolated feature. In other types of syndromic craniosynostosis, bicoronal synostosis is commonly found in association with midfacial hypoplasia and additional sutural fusions.

Lambdoid synostosis

Lambdoid suture synostosis is a rare form of craniosynostosis with a prevalence of <5% of all craniosynostoses. Unilateral lambdoid synostosis needs to be carefully differentiated from posterior plagiocephaly, as the management of the two conditions is completely different. On imaging there is obliteration or sclerosis of the lambdoid suture. Unilateral lambdoid synostosis creates a trapezoid-shaped head (Figure 21.3). There is ipsilateral occipitoparietal flattening and compensatory growth causing contralateral parietal and frontal bossing, and also ipsilateral occipitomastoid bossing. The ipsilateral ear may be posteriorly displaced.

Multisuture synostosis and cloverleaf skull

Most multiple suture synostoses are associated with syndromes. Common combinations include bilateral coronal with bilateral lambdoid synostoses or bilateral coronal synostoses with sagittal synostoses. When there is pansynostosis, the membranous bones of the calvarium expand between the sutures and give the appearance of a cloverleaf skull or Kleeblattschädel.

Treatment of non-syndromic craniosynostosis

The philosophy of care is a multidisciplinary team approach to correct anatomical deformity in a single stage and monitor for postoperative elevated intracranial pressure.

Most patients with an isolated single suture craniosynostosis have few problems, once this is corrected. With the exception of sagittal synostosis, which is generally treated early, most craniofacial centres treat unsutural synostosis with some form of formal calvarial remodelling to produce a definitive correction of head shape. Opinions on ideal timing vary, but in general the operation is undertaken between six months and 2 years of age. Our protocol is to monitor these patients postoperatively with an annual eye evaluation to look for signs of elevated ICP.

Genetic testing of non-syndromic craniosynostosis patients has led to the discovery of associated genetic mutations. Patients have increased levels of autism and learning difficulties compared to their peers and this may not necessarily be related to elevated ICP. It is thought that up to 15% of patients with non-syndromic craniosynostosis will develop elevated ICP.^{52,53}

For patients with single-suture non-syndromic craniosynostosis, specific operations have been developed that are tailored to correct the characteristic head shapes and these are summarized below:

Sagittal synostosis

Patients can undergo surgery from about four months of age, which involves excision or release of the sagittal synostotic suture and then a device can be applied to mould the head shape. This can be done via an endoscopic-assisted suturectomy, with or without an orthotic helmet moulding device. The other main technique is to use a springs-assisted cranioplasty, which can be performed before six months of age. Two parasagittal osteotomies are made via a short coronal incision on the vertex. The bone with the fused sagittal suture is left intact over the sagittal sinus and three or four stainless steel springs are inserted into the edges of the osteotomies and they push the bone edges apart and remodel the head shape (Figure 21.5). The springs are removed a few months later at a second operation.

Older children between 9 and 18 months of age will need more extensive surgery, for example the Pi procedure (Figure 21.6). A total calvarial remodelling is indicated for a child who presents later, from the age of 18 months onwards. Patients will need pre-operative eye evaluation to look for signs of elevated ICP and will

Spring assisted cranioplasty

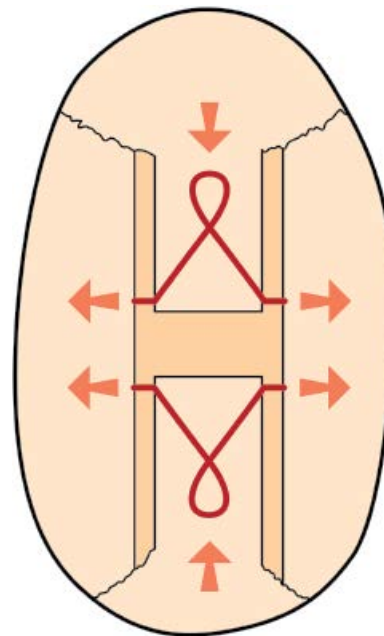


Figure 21.5 Springs-assisted cranioplasty for sagittal synostosis.

also need monitoring postoperatively as a small percentage of patients develop elevated ICP after surgery.

Fronto-orbital advancement

Patients with metopic synostosis, unicoronal synostosis and bicoronal synostosis require an operation to correct the shape of the supraorbital and frontal region. Surgery to correct these craniosynostoses involves different types of fronto-orbital advancement (Figure 21.7).

A fronto-orbital advancement is usually carried out at approximately 9–18 months of age unless signs of elevated ICP are identified earlier. The goals are to provide improved morphology of the forehead and upper orbits and, where necessary, increased intracranial space in the anterior cranial vault for the brain and increased orbital volume, which allows the eyes to be positioned more normally for better protection from exposure.

A coronal (scalp) incision is made, and the anterior flap is elevated either in a subgaleal plane or subperiosteally along with the temporalis muscle. The degree of further dissection depends on the extent of planned surgery. The most common procedures undertaken remodel the fronto-orbital bar (Figure 21.7). At Great Ormond Street Hospital, we favour a much more limited procedure which does not remodel the entire fronto-orbital bar. This limits the amount of orbital dissection required.

A frontal craniotomy is then performed. This allows retraction of the brain so that any orbital osteotomies can be safely undertaken and harvests the frontal bone, which will be reshaped during the procedure.

To undertake a traditional fronto-orbital remodelling, orbital osteotomies are completed across the orbital roof and superior

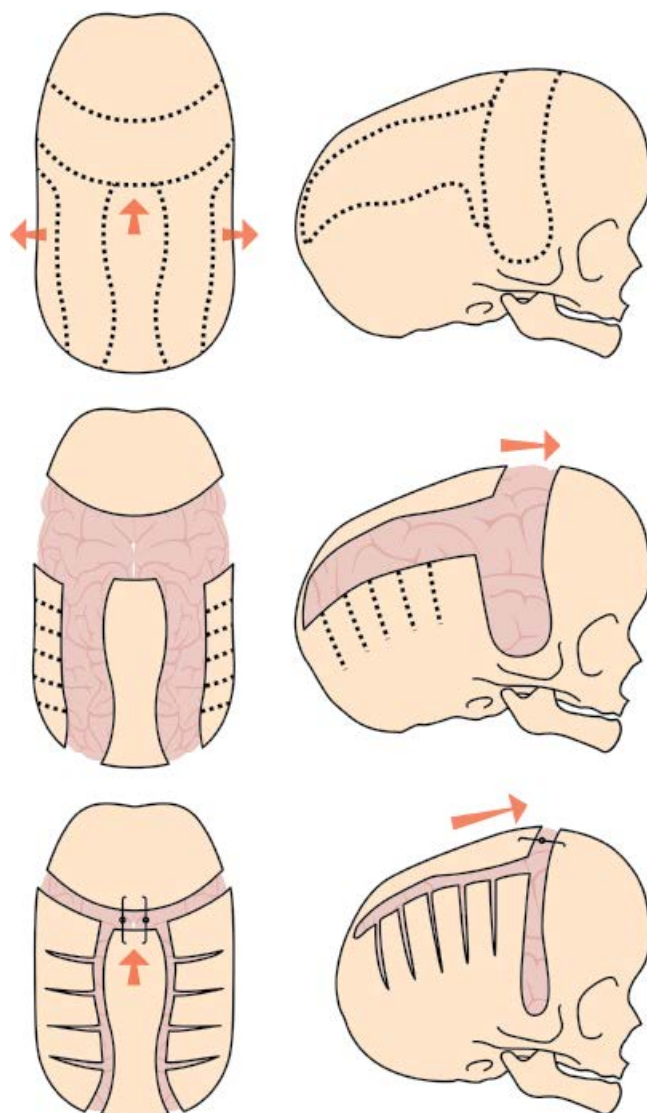
Procedure

Figure 21.6 Pi procedure for the treatment of sagittal synostosis.

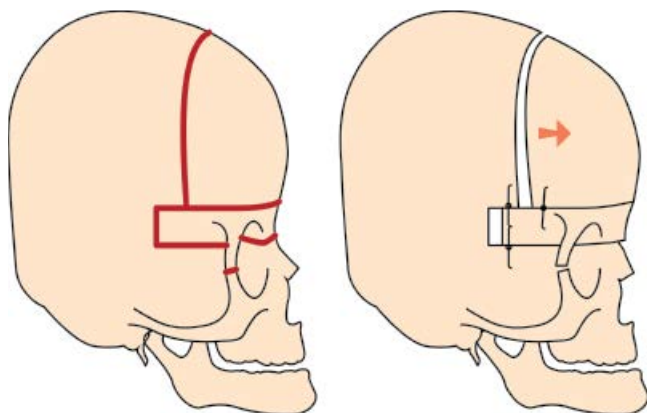
Fronto-orbital advancement

Figure 21.7 Fronto-orbital advancement.

aspect of the medial orbital wall, laterally through the lateral orbital wall and inferiorly just into the inferior orbital fissure. The three-quarters orbital osteotomy units are removed from the field and reshaped and then re-inset. The supraorbital bar is overcorrected. Fixation is generally achieved with resorbable sutures and plates and screws or stainless steel wires. Calvarial bone is cut into segments, which are replaced as grafts to achieve a more normal appearance of the anterior cranial vault.⁵⁴

Posterior cranial vault remodelling

Patients with either unilateral or bilateral lambdoid synostosis benefit from posterior cranial vault remodelling. It is also beneficial in patients with syndromic craniosynostosis who have a bicoronal synostosis and craniocerebral disproportion, and elevated ICP as it leads to an increase in the size of the volume of the skull. It can be performed with or without distractors or springs depending on the requirements of the individual patient. It can also be helpful to correct a Chiari I malformation.⁵⁵

Treatment of syndromic craniosynostosis

The philosophy of care is a multidisciplinary team approach to maximize developmental potential, with staged operations and follow-up into adulthood.

The primary aim of treatment is the prevention and treatment of functional morbidity due to potential problems with vision, airway, elevated ICP, hearing, nutrition, psychological and social development, which can all contribute to developmental delay (Figure 21.8).

Crouzon syndrome

In 1912, Crouzon described a condition featuring craniosynostosis, maxillary hypoplasia, exorbitism, class III malocclusion and hypertelorism.⁵⁶ The hands and feet are usually normal, but subtle anomalies can occur. Crouzon syndrome represents 4.8% of cases of craniosynostosis at birth and the birth prevalence has been estimated to be 16.5 per million births. The syndrome is caused by a genetic mutation generally in the *FGFR2* gene and

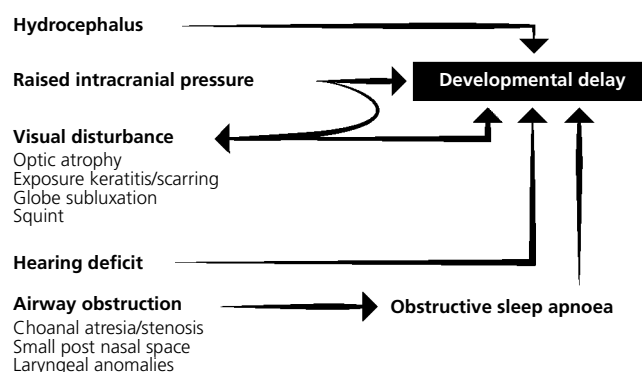


Figure 21.8 Functional problems in syndromic craniosynostosis which contribute to developmental delay.

can arise *de novo* or is inherited in an autosomal dominant fashion. Older paternal age is a risk factor for a *de novo* mutation.⁵⁷ The phenotype can be variable in severity and craniosynostosis can be absent or delayed.⁵⁸ Both coronal sutures are commonly fused, but the sagittal and lambdoid sutures can also be involved. The anterior and posterior cranial base can be short due to early closure of the basicranial synchondroses and thus the posterior fossa is often shallow and cerebellar tonsillar herniation can occur, as well as elevated ICP. The volume of the nasopharynx is also reduced, which can contribute to obstructive sleep apnoea (OSA).

Apert syndrome

In 1906, Apert reported a series of cases with a brachycephalic head, severe syndactyly of hands and feet and a cleft palate.⁵⁹ Apert syndrome or acrocephalosyndactyly type I is characterized by craniosynostosis, turribrachycephaly, hypertelorism, downslanting palpebral fissures, exorbitism, midface hypoplasia, class III malocclusion, cleft palate and complex syndactyly of the hands and feet. Apert syndrome represents 4.5% of all craniosynostosis cases and the birth prevalence is 15.5 per 1 million births.⁵⁸ The syndrome is caused by a one of two *FGFR2* (10q26.13) mutations involving amino acids (Ser252Trp and Pro253Arg), and can arise *de novo* or is inherited in an autosomal dominant fashion. Older paternal age is a risk factor for a *de novo* mutation.⁵⁷ The anterior cranial fossa is shortened and widened and the orbital volume is reduced, causing exorbitism. There is a wide-open calvarial defect from the root of the nose to the posterior fontanelle in the mid-sagittal plane. The intracranial volume is increased compared to controls. Cloverleaf skull has been reported in association with Apert syndrome and occurs in about 4% of cases. Globe subluxation can occur. Cleft palate commonly occurs and the palate is high arched, with an anterior open bite and dental overcrowding. There is no association with cleft lip. Upton described a classification of the Apert hand (Table 21.2).⁶⁰ Additional anomalies can include fusion and malformation of other joints, including the elbows and shoulders, cervical vertebral fusions, congenital airway malformations, deafness and neurodevelopmental abnormalities. There is a greater downward slant of the lateral canthi and a distinctive, S-shaped upper eyelid ptosis. The quality of the skin frequently varies from normal, with acne and hyperhidrosis being prominent features.

Pfeiffer syndrome

The original report of Pfeiffer syndrome in 1964 described a family of three generations in whom eight individuals had a syndrome consisting of cranial vault abnormalities, broad thumbs and great toes and partial syndactyly of the hands and feet.⁶¹ Additional anomalies can include ankylosis of the elbow and other joints, congenital airway malformations, deafness and neurodevelopmental abnormalities.⁶² There is a characteristic facial appearance: a wide cranial vault with a flat occiput, broad high forehead, midface hypoplasia, a small nose with a depressed nasal bridge and orbital hypertelorism. Patients often have exorbitism due to shallow orbits. It affects approximately 1 in 100 000 individuals.⁶³ The syndrome may be caused by mutations in the fibroblast growth factor genes *FGFR1* (chromosome 8p11.2–p11)⁶⁴ or *FGFR2* (chromosome 10q26.13).^{65,66} The mutation can arise *de novo* but can also be inherited in an autosomal dominant fashion. Males and females are affected equally. The spontaneous mutation is thought to be associated with advanced paternal age.⁵⁷ Pfeiffer syndrome is considered type V of the acrocephalosyndactyly syndromes.⁶³ Respiratory, ocular, otologic and neurologic problems are key factors for these patients and indicators of the need for treatment.

Functional problems associated with syndromic craniosynostosis

Neurological problems

Elevated intracranial pressure is a serious functional problem. It can have a slow onset and be difficult to recognize in a paediatric population. Untreated craniosynostosis with elevated ICP will cause papilloedema and eventually optic nerve atrophy and blindness. There is no consensus on what normal levels of ICP are in a paediatric population, as this is not routinely measured in healthy children. Elevated ICP results from a number of different factors, including craniocerebral disproportion, abnormalities of cerebrospinal fluid dynamics, impaired cerebral venous drainage secondary to generalized cranial base stenosis and hydrocephalus.^{54,67}

Hydrocephalus is common and is characterized by a progressive enlargement of the ventricles over time, which is different from non-progressive ventriculomegaly related to brain maldevelopment.⁶⁸ There is good evidence that OSA secondary to upper airway obstruction and venous hypertension exacerbate elevated ICP in patients with syndromic craniosynostosis.^{69,70}

Table 21.2 Upton's classification of the Apert hand (1991)

Deformity	Type I	Type II 'Mitten hand'	Type III 'Hoof hand'
Thumb radial clinodactyly	Present	Present	Present
Index radial clinodactyly	Absent	Present	Present
First web syndactyly	Simple (non-osseous)	Simple (non-osseous)	Complex (osseous)
Complex 2–3–4 syndactyly and symbrachyphalangism	Present	Present	Present
4–5 Syndactyly	Simple, incomplete	Simple, incomplete	Simple, complete

Source: Upton, 1991.⁶⁰ Reproduced with permission of Elsevier.

The Chiari I malformation (cerebellar tonsillar herniation) is often found in patients with syndromic craniosynostosis and has a mechanical rather than developmental aetiology and can be caused by a small posterior fossa (which in itself could be due to lambdoid suture fusion) or by venous congestion because of obstruction to venous outflow across the abnormal skull base.^{70,71} Brain anomalies that can occur include: defects of the corpus callosum, porencephalic cysts, mega cisterna magna, focal cortical tissue loss and brain herniation through multiple areas of the skull base due to elevated ICP.⁷²

Patients with Crouzon syndrome usually have normal intelligence, with about 3% having intellectual compromise.⁷³ Patients with a mild Apert or Pfeiffer phenotype can have normal intelligence and those with a severe phenotype can have severe neurodevelopmental delay.^{58,74}

Ocular problems

Primary ophthalmic findings in Apert, Crouzon and Pfeiffer syndromes include exorbitism due to shallow orbits, increased interpupillary distance, strabismus, refractive error, iris colobomata, cataracts, nasolacrimal duct obstruction and ptosis. Secondary findings include amblyopia, corneal exposure, globe herniation, optic atrophy and papilloedema.⁷⁵ Major causes of visual loss are amblyopia, exposure keratopathy and optic atrophy, due to elevated ICP.⁷⁶ Patients with syndromic craniosynostosis must undergo regular ophthalmologic assessment.

Otological problems

Hearing loss is common. External auditory canal anomalies can occur, with stenosis or atresia of either the bony or cartilaginous parts of the auditory canal, and malformations of the middle ear and ossicles,⁷⁷ with concomitant conductive hearing loss. CT scanning can be helpful in defining the anatomical abnormalities.⁷⁸ There is a higher incidence of middle ear effusions in patients with Apert syndrome, which may be related to the presence of a cleft palate.⁷⁹

Respiratory problems

Airway pathology can include midface hypoplasia, nasopharyngeal stenosis, choanal atresia or stenosis, and tracheal anomalies.^{73,74,80,81} These represent a significant source of potential morbidity and mortality. Airway obstruction can cause failure to thrive, suboptimal neurological development and developmental delay. Disruption of sleep states in OSA, particularly loss of sustained quiet sleep, has been shown to impair growth hormone release.⁶⁷ There is a strong correlation between the presence of hydrocephalus requiring ventriculoperitoneal (VP) shunting and severe upper airway obstruction: 69% of patients with VP shunts also required tracheostomy.⁸⁰

Many children with syndromic craniosynostosis need some form of airway support during infancy or childhood. A drop off the growth curve can be a useful predictor of the need for both airway and feeding intervention in children with craniosynostosis.⁸⁰ A nasogastric tube or a gastrostomy tube may be required.⁵⁴ Lymphoid

hypertrophy occurs at approximately age 3–6 years and children whose airways become more obstructed at this age often benefit from tonsillectomy and adenoidectomy. Frequent monitoring for signs of upper airway obstruction is required. The use of nasopharyngeal airways, continuous positive airways pressure (CPAP) and tracheostomy may be required to maintain an adequate airway.

Congenital heart defects

It is not uncommon for children with syndromic craniosynostosis to have congenital heart defects.⁷⁴ This leads to an increased risk when undergoing surgery or general anaesthesia. Some children with congenital heart defects have the potential for either right to left or bidirectional shunts. Given that venous air embolism is common with craniosynostosis repair, the risk of coronary or cerebral air embolism is significant in these children.

Surgical management of syndromic craniosynostosis

Most children with syndromic craniosynostosis require surgery to protect function, expand the cranial vault, advance the midface and then to make final adjustments to appearance.

The management can be subdivided into 'crisis interventions' (e.g. for threats to airway, threats to vision and elevated ICP); 'early interventions' for hydrocephalus, visual disturbances, hearing deficit, OSA, which all contribute to developmental delay; interventions in childhood to change appearance in order to support psychosocial development and wellbeing and 'late interventions' to improve aesthetic appearance at the completion of growth. The sequence of surgical interventions is summarized in Table 21.3.

Management in infancy

Management in infancy is generally directed towards the treatment and prevention of functional crises. Most commonly, cranial vault expansion is undertaken to treat cranio-cerebral disproportion and elevated ICP.

Table 21.3 Sequence of craniofacial surgical procedures in syndromic craniosynostosis

Age	Procedure
6 months to 3 years	Crisis intervention (ICP, orbital protection, airway), cranial vault expansion, midfacial or frontofacial advancement
4–8 years	Correction of midface deformities, primary and secondary cranial vault procedures, and adjunctive procedures
9–12 years	Correction of midface deformities, secondary cranial vault procedures, and adjunctive procedures
13–17 years	Late correction of midface deformities. Adjunctive procedures and orthodontic therapy
Over 17 years	Orthognathic surgery

Broadly there are two philosophies of surgical management at this stage:

- A fronto-orbital advancement can be performed (Figure 21.7). Proponents of this approach cite the advantages of the increased cranial volume achieved along with some degree of orbital protection. If the frontal advancement does not relapse, midfacial advancement can be achieved at a later stage with a subcranial Le Fort III osteotomy (age 4–10 years), thus avoiding the need for a second transcranial procedure (Figure 21.9).
- The other technique is to perform a posterior vault expansion (either an acute advancement with bone grafts, or gradual advancement using springs or distraction devices). Proponents of this approach believe it is a functionally better technique, because it can produce greater increases in cranial volume than frontal advancement and there is some evidence that the change in posterior cranial fossa anatomy treats Chiari malformations. Performing a posterior expansion at this stage leaves the forehead untouched, which is a great advantage from a technical point of view if a frontofacial advancement osteotomy is required at a later date.

In children with severe exorbitism, elevated ICP and upper airway obstruction, frontofacial advancement by distraction can be considered as early as four months of age.⁹⁷

Management of midface deformity in childhood and adolescence

Severe midfacial retrusion is usually corrected in childhood or adolescence. There are two commonly adopted strategies. The first is to perform a frontofacial advancement, which is most commonly performed as a monobloc osteotomy (Figure 21.10). The osteotomy effectively cleaves the entire facial skeleton and forehead from the anterior skull base. Advancement of the monobloc segment increases intracranial volume, enlarges the

orbits, increases upper airway volume and advances the midface. The procedure can be performed conventionally holding the advanced segment in position with bone grafts, plates and screws, but is nowadays most frequently undertaken by internal or external distraction techniques (Figure 21.11). The procedure is effective and has been successfully adapted to deal with the anatomical facial flattening seen in Apert syndrome by converting the monobloc to a facial bipartition advancement.⁸²

The second strategy is to advance the forehead and midface separately in two operations (first a fronto-orbital advancement (Figure 21.7) followed at a later date by a subcranial Le Fort III osteotomy (Figure 21.9). The Le Fort III can also be performed conventionally or by distraction.

There is good evidence to suggest that frontofacial advancement produces better functional outcomes and aesthetic results than a two-stage forehead advancement and Le Fort III. The disadvantage of this approach is its higher complication rate.^{98,99} The monobloc osteotomy results in a communication between the anterior cranial fossa and upper nasal spaces, which increases the risk of ascending infection, persistent CSF leaks and mucocoele formation. A separate forehead advancement and Le Fort III procedure reduces these risks. Opinion remains divided about the best approach.

Orthognathic procedures for definitive occlusal correction

The mandible has normal growth potential in children with syndromic craniosynostosis, but the maxilla does not. This leads to a class III malocclusion, often with an anterior open bite. Although monobloc and Le Fort III procedures performed in childhood and adolescence go some way to correcting this intermaxillary discrepancy, there is often a residual class III malocclusion. A Le Fort I osteotomy often combined with a mandibular ramus osteotomy

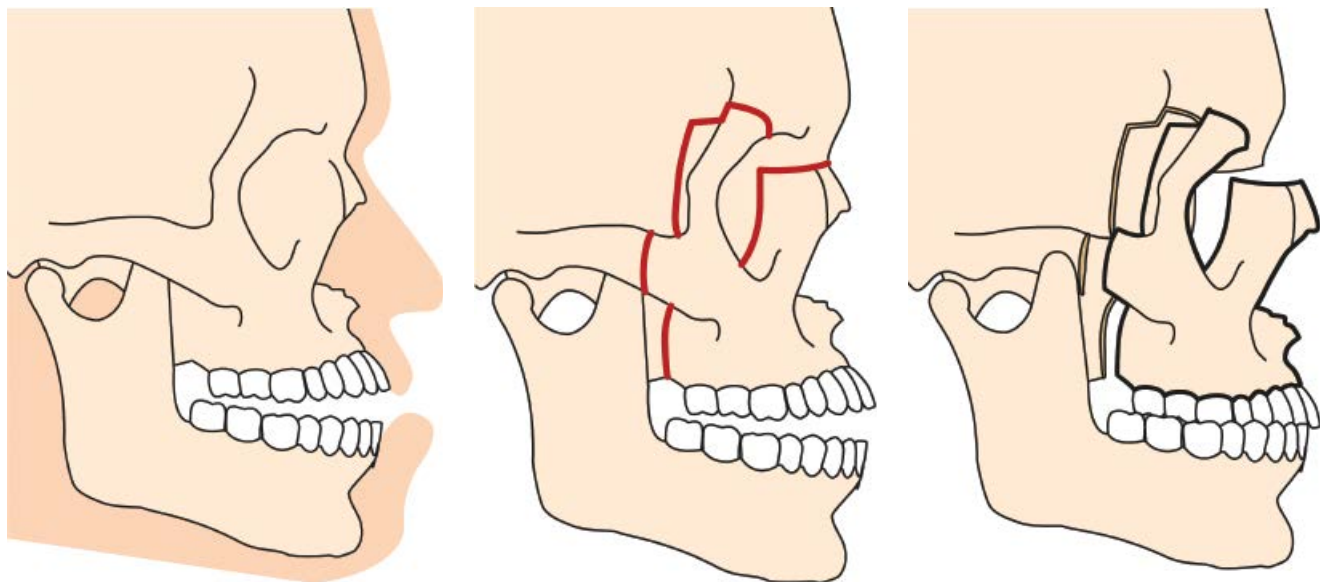


Figure 21.9 Subcranial Le Fort III midface advancement.

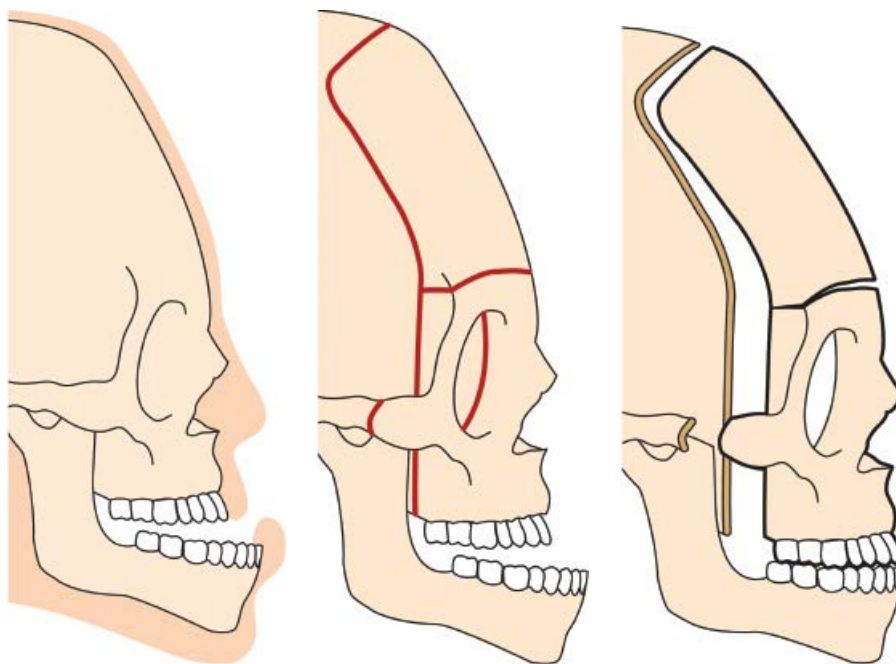


Figure 21.10 Frontofacial advancement with monobloc osteotomy.

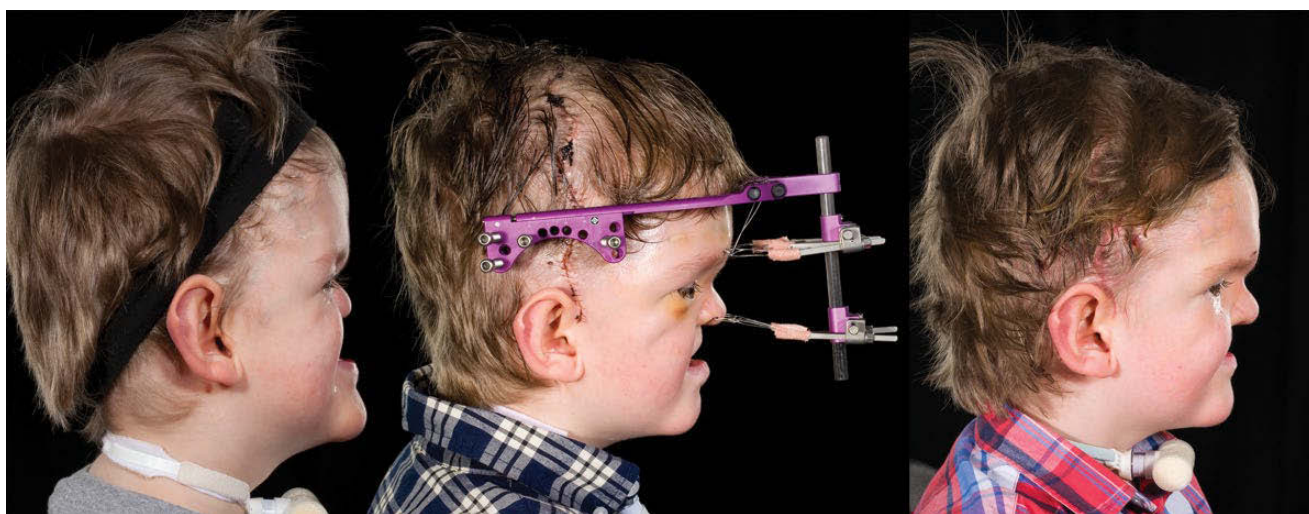


Figure 21.11 Frontofacial advancement by distraction using the external RED device.

(bimaxillary osteotomy) is generally required in combination with genioplasty. About 30% of patients with syndromic craniosynostosis require orthognathic surgery and this is carried out in conjunction with orthodontic treatment and planned for completion at the time of skeletal maturity (age 16–18 years).

Complications of surgery for syndromic craniosynostosis

Complications of craniofacial surgical procedures are listed in Table 21.4. Early series looking at complications for all types of craniofacial surgery quoted mortality rates of between 1 and 2%.^{89,90} In a more recent multicentre review, the mortality had fallen to 0.1% for transcranial procedures and 0.3% for subcranial procedures.⁹¹

These figures are similar to our own experience of a 0.11% mortality rate for all craniofacial procedures undertaken in the last 5 years.⁸³ Higher mortality rates are seen in reviews quoting complications for frontofacial advancement, demonstrating the greater risks associated with major surgery.^{83,91–94} The risk of death or significant morbidity most commonly arises from bleeding (either exsanguination or postoperative intracranial bleeding) or less commonly postoperative airway complications. Well-defined protocols and strategies for addressing perioperative blood loss are important.⁹⁴ It is our practice to routinely use a cell saver to recycle lost blood during major craniofacial procedures and run a strict protocol for blood replacement and investigation of blood loss post operatively.

Table 21.4 Complications of craniofacial surgical procedures

Early	Bleeding
	Airway
	Infection (e.g. meningitis)
	Neurosurgical (e.g. stroke, elevated ICP)
Intermediate	CSF leak ⁸³
	Bone loss (e.g. frontal bone) ^{83,84}
	Osteomyelitis/sequestrum
	Plate infections
Late	Mucocele ⁸³
	Late bone loss
	Relapse
	Seizures ^{82,83,85}
	Premature ageing ^{86,87}
	Radiation exposure from multiple CTs ⁸⁸

Conclusions

Modern craniofacial surgery, despite the risks of serious complications, has transformed the lives and developmental potential of children with syndromic craniosynostosis. The treatment of craniosynostosis requires a dedicated multidisciplinary team because of the complex interrelated functional and anatomical problems caused by the condition. It is only with the help and advice of members of this team that craniofacial surgeons can make informed decisions about the timing of surgery and the use of different surgical techniques.

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CHAPTER 22

Orthognathic surgery

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Introduction

Orthognathic surgery involves the surgical manipulation of elements of the facial skeleton to restore anatomic proportion, facial harmony and a functional relationship in patients with dentofacial skeletal disproportion. Successful orthognathic surgery requires multidisciplinary input involving the patient, consultant orthodontists and maxillofacial surgeons, laboratory technicians and others (e.g. liaison psychiatrist, restorative dentistry consultants, dental hygienists, ENT surgeons and nurses). It is commonly used for the treatment of dentofacial discrepancies when orthodontic treatment and cosmetic procedures are insufficient to answer the patient's concerns.¹ Orthognathic surgery is involved in the final correction of syndromic craniofacial patients, congenital disorders such as hemifacial microsomia and acquired disorders such as hemifacial atrophy (Figure 22.1).

Surgery is part of a larger, more complex treatment pathway that usually involves both pre- and postsurgical orthodontics. Orthognathic treatment seems to provide good outcomes at relatively low cost.²

Indications for treatment

Treatment is indicated where a patient is handicapped as a result of dentofacial deformity. This may manifest in various ways:

- Compromised jaw function: The patient with an anterior open bite often complains that they are unable to bite into a sandwich. Improving the bite may also improve dental health.
- Discrimination due to facial and dental appearance: This may be subjective and difficult to quantify. Assessment is often helped by the involvement of a liaison psychiatrist.^{3–5}
- Sleep apnoea: Bimaxillary advancement is a recognized and effective treatment for sleep apnoea.⁶

There is no evidence that orthognathic surgery is a treatment for temporomandibular joint disorders.⁷

Treatment planning for the correction of dentofacial disproportion involves consideration of multiple variables. The ultimate aim is to address the patient's concerns and have a satisfied patient at the end of treatment.

Overall treatment aims can be broken down as follows:

- 1 Achieve optimal dental occlusion/function and harmonious, balanced facial aesthetics.
- 2 Optimize future oral health.
- 3 Minimize treatment morbidity.
- 4 Maximize the possibility of a stable result.
- 5 Eliminate other symptoms of dentofacial deformity.
- 6 Patient satisfaction.

It is important that patients prioritize what they wish to achieve and that they have realistic expectations of the likely outcomes. Patients attend a new group-style clinic⁸ where they have an opportunity to question the team and a past patient. The more informed a patient prior to commencement of treatment, the greater their likelihood of satisfaction at the end of treatment.^{1,9}

General assessment¹⁰

Clinical evaluation of the face requires that the clinician knows what to look for. Leonardo da Vinci called this 'sapre verde'.¹¹

When assessing a patient it is important to ensure that a natural head position is adopted. This is the position taken by the head when standing straight and looking into a mirror placed at eye level with the interpupillary line parallel to the floor. Assessment is undertaken by systematic examination in full face view and in profile.

Frontal facial evaluation (full face)

Facial shape

Facial index is the ratio of facial height to facial width (Figure 22.2). Facial height is the distance from trichion to soft tissue menton. Facial width is the distance between the



Figure 22.1 Pre- and post-op views of a patient treated for hemifacial atrophy.

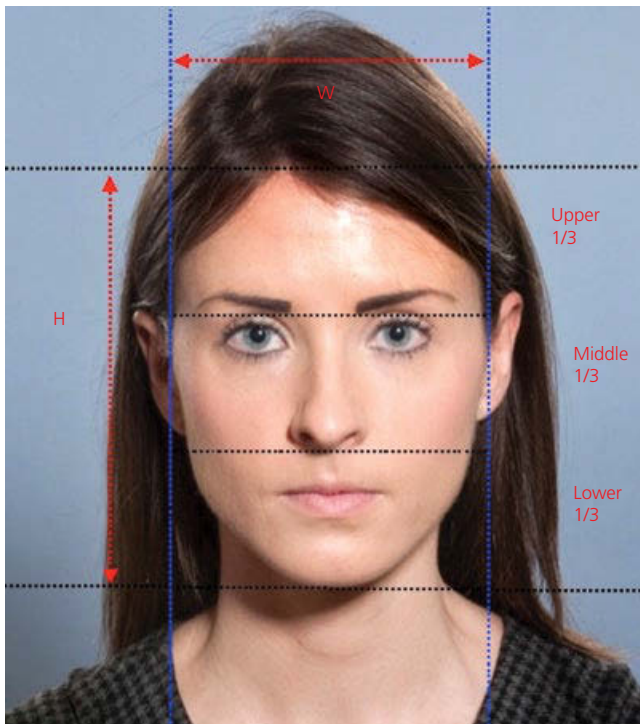


Figure 22.2 Assessment of facial shape and vertical proportions. *H*, facial height; *W*, facial width.

lateral-most aspect of each zygomatic arch. For males, facial index should approximate to 1.35:1 and for females to 1.3:1.

Vertical proportions

These are assessed by facial trisection to upper third – hairline trichon to nasion, middle third – nasion to subnasale and lower third – subnasale to soft tissue menton (Figure 22.2). Anterior lower facial third is often slightly greater than the middle third, especially in males.

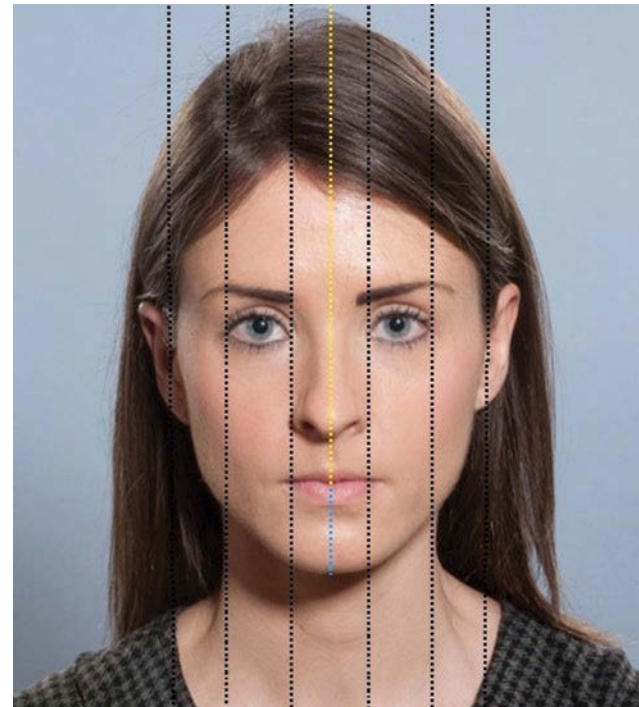


Figure 22.3 Assessment of transverse proportions and facial symmetry. Facial and maxillary midlines are demonstrated by the yellow line. Mandibular midline is indicated by the blue line.

Transverse proportions

Transverse proportions are assessed by the rule of fifths (Figure 22.3). Each fifth is approximately the width of an eye. Mouth width should be equal to the distance between the medial iris margins. Alar base width should be equal to the intercanthal distance.

Anteroposterior assessment

Increased scleral show is a sign of midface deficiency. Paranasal hollowing/flatness is a sign of maxillary hypoplasia (Figure 22.4).

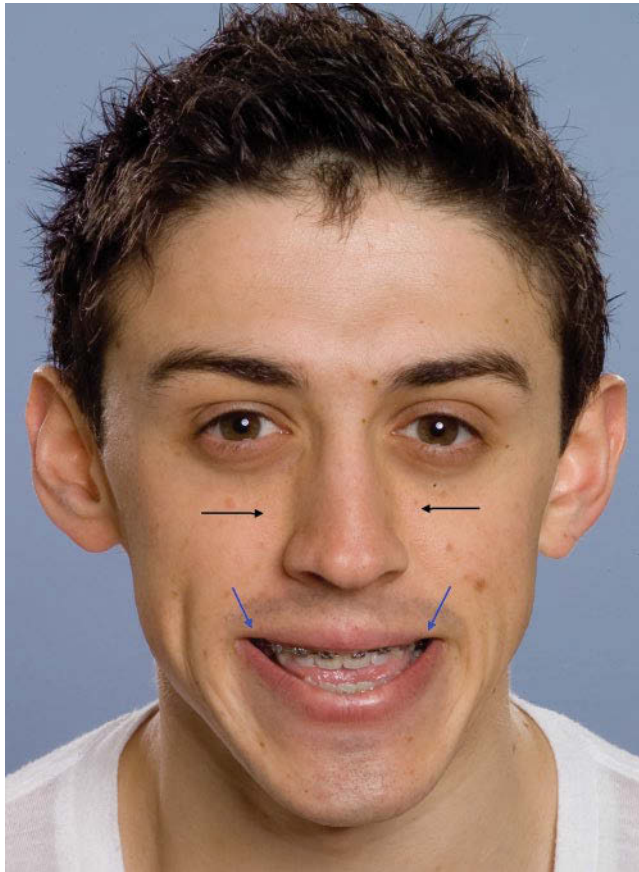


Figure 22.4 Maxillary hypoplasia related paranasal hollowing (black arrows) and dark buccal corridors (blue arrows).

Facial symmetry

Facial midline is the line connecting the middle of the philtrum of the upper lip (Cupid's bow) and nasion (see Figure 22.3). Minor asymmetry is considered normal.

The transverse occlusal plane should be parallel to the interpupillary line in the absence of a transverse maxillary cant or vertical orbital dystopia. Patients with a vertical orbital dystopia will have acquired a compensatory, horizontal head tilt and this will persist after surgery.

Maxillary midline

This is assessed in relation to the middle of the philtrum of the upper lip (Cupid's bow) and facial midline (see Figure 22.3).

Mandibular midline

Mandibular dental midline is assessed in relation to the chin's midpoint and facial midline (see Figure 22.3).

Dark 'buccal corridors' on smiling

These may be due to transverse maxillary deficiency, palatally inclined maxillary posterior teeth and maxillary retrognathia (see Figure 22.4).

Maxillary incisors and upper lip

Maxillary central incisors are approximately 9–12 mm in length. Tooth show is normally 2–4 mm, at rest. Incisal edge wear reduces incisor exposure. Reduction will also occur in cases of gingival hypertrophy and with a more incisal gingival attachment.

Vertical lip position depends on upper lip length and vertical maxillary incisor position (see below). With closed lips, the lower lip should cover the incisal third of the maxillary incisors.

The relationship of the upper lip to the upper incisors helps determine adequacy of maxillary position.

Anteroposterior lip position

Lips may be:

- held together at rest and therefore termed 'competent';
- apart at rest by more than 3–4 mm but able to be closed together with conscious effort – 'potentially competent'; or
- unable to be held together due to interposed teeth or relative jaw malposition and therefore 'incompetent'.

Lip animation

A strap-like lower lip often retroclines incisors. This commonly occurs in class II division 2 cases in the presence of a deep labio-mental fold.

Lip shape

Everted lips may be due to interposed proclined maxillary incisor teeth. Flat or backward sloping lips give an 'aged' appearance to facial profile. Full lips are less likely to significantly alter position with anteroposterior dental movement. Thin lips are more likely to 'flatten' with incisor retraction. Vermillion show of the lower lip is 2–3 mm more than in the upper lip.

Upper lip length and smile curtain

Normal upper lip length is 18–22 mm. Upper lip length usually comprises one third of anterior lower face height (see Figure 22.2). An increased muscular capacity to raise the upper lip higher than average when smiling may lead to excessive show of the gingival tissues and a 'gummy smile'. If the lip–incisor relationship at rest is correct, this smile curtain may have to be accepted. The Le Fort I incision tends to reduce the smile curtain slightly, at least temporarily.

Facial profile evaluation

Anteroposterior position of maxilla

The Frankfort plane is an important cranial base reference point and is taken to represent the true horizontal (Figure 22.5). It is the plane between the superior margin of the external auditory meatus and the inferior most point of the infraorbital margin. The Frankfort plane is parallel to the floor when a patient has adopted the natural head position.

Facial vertical line is perpendicular to the Frankfort plane from soft tissue nasion, with the patient in natural head position. Subnasale is on this line.

Anteroposterior chin position

Soft tissue pogonion (i.e. most anterior point on the symphysis) should be 0 ± 2 mm to facial vertical¹² (see Figure 22.5). It is vital to assess the soft tissue thickness anterior to the bony chin, as an overprojection of the chin may be entirely due to the soft tissue thickness.

Anteroposterior lip position

The aesthetic line joins the nasal tip to soft tissue pogonion (see Figure 22.5). The upper lip should be 4 mm and the lower lip 2 mm behind this line in adults. This is very dependent on nasal and chin projection. The harmony line runs from soft tissue pogonion to touch the upper lip (see Figure 22.5). It should bisect the nose.

Relationship of upper lip to nasal columella

Nasolabial angle is formed between the nasal columella and the upper lip slope.¹² The average value ranges between 85 and 120°.

Relationship of lower lip to chin

Labiomental angle is formed between the lower lip and chin. It is determined by the lower incisor inclination and anterior lower face height.^{12–14} The average value varies between 110 and 130°.

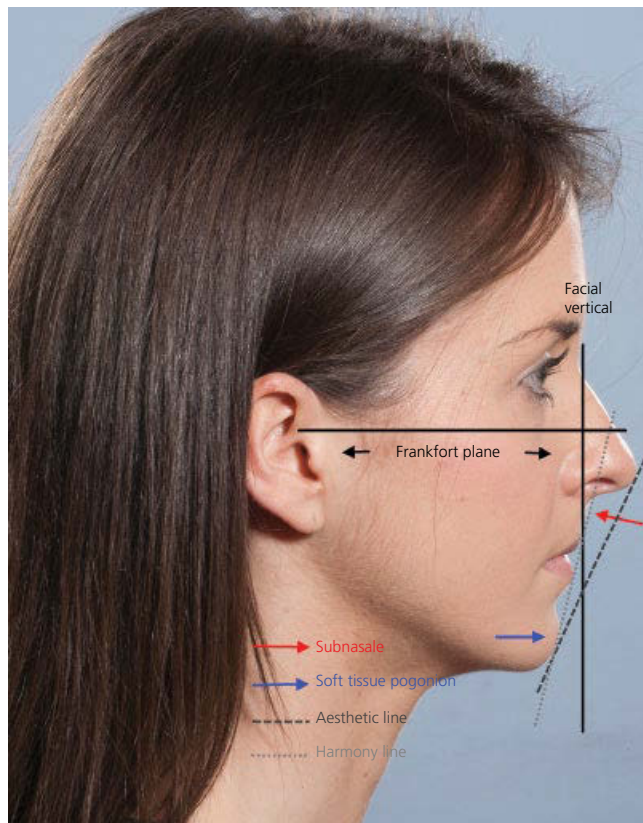


Figure 22.5 Evaluation of facial profile.

Relationship of chin to submental plane

Lip–chin–submental plane angle averages 90–110°. This is obtuse if mandibular retrognathia, retrogenia, excessive lower lip projection or increased submental fat are present ('double chin').

Dental occlusion

A malocclusion can be defined as 'an appreciable deviation from ideal occlusion' and is described as misalignment of teeth or incorrect relation between the teeth in the two dental arches. Malocclusions may be accompanied with a skeletal discrepancy, warranting orthognathic surgery to 'normalize' the relationship between the maxilla and mandible.

Occlusion refers to the manner in which the opposing teeth meet. There are a number of ways a malocclusion may be classified including based on molar relationship (Angle's classification) and based on incisor relationship (British Standards Institute classification).

Angle's classification

This was described by Edward Angle in the 1890s and describes the molar relationship in the anterior–posterior direction between the mesiobuccal cusp of the maxillary first molar and mesiobuccal groove of the lower first molar (Figure 22.6).

The molar relationship can be class I, II or III and different molar relationships can exist on either side of the mouth.

- *Class I (neutroclusion)*: The mesiobuccal cusp of the permanent maxillary first molar occludes in the buccal groove of the permanent mandibular first molar.
- *Class II (distocclusion/post-normal)*: The mesiobuccal cusp of the permanent maxillary first molar occludes mesial to the buccal groove of the permanent mandibular first molar.
- *Class III (mesiocclusion/pre-normal)*: The mesiobuccal cusp of the permanent maxillary first molar occludes distal to the buccal groove of the permanent mandibular molar.

A half unit class II or class III molar relationship is also occasionally described.

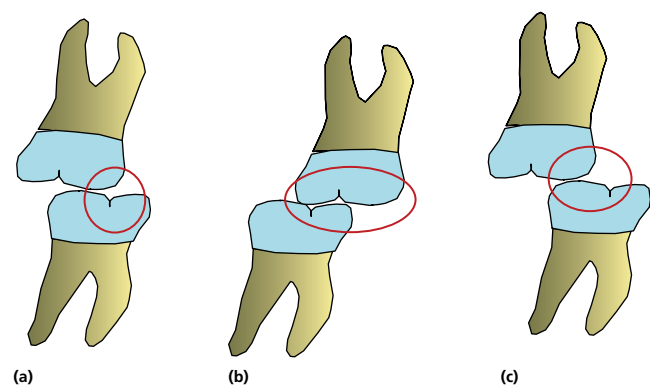


Figure 22.6 Angle's molar relationship in the anterior–posterior direction between the mesiobuccal cusp of the maxillary first molar and mesiobuccal groove of the lower first molar. (a) Class I; (b) class II; (c) class III.

Incisor classification

The British Standards Institute describes the relationship between the lower incisor edges and the cingulum plateau of the upper central incisors when the teeth are in centric occlusion (in contact). The incisor relationship may be class I, class II division I, class II division II or class III (Figure 22.7).

- **Class I:** The lower incisor edges occlude with or lie immediately below the cingulum plateau of the upper incisors.
- **Class II:** The lower incisor edges lie posterior to the cingulum plateau.
 - Division I: The upper incisors are proclined and the overjet is usually increased.
 - Division II: The upper incisors are retroclined and the overjet is usually reduced.
- **Class III:** The lower incisor edges occlude anterior to the cingulum plateau. The overjet is reduced or reversed.

Other useful terms

Overbite is the degree of vertical overlap between the incisors when the teeth are in centric occlusion. It is measured relative to the incisor edges. Ideally, the upper incisors should overlap a third of the lower incisor crowns (Figure 22.8).

An **anterior open bite** exists when the upper incisors fail to overlap the lower incisors when the teeth are in centric occlusion.

Overjet is the horizontal overlap of the incisors. It is measured in millimetres between the labial surfaces of the upper

and lower incisors (Figure 22.9). The overjet is normally a representation of the thickness of the incisal edges (i.e. 2–4 mm). A reverse overjet exists if any of the lower incisors lie ahead of the upper incisors, leading to an anterior crossbite.

The orthognathic patient

Orthognathic surgery can be defined as ‘the correction of functional and aesthetic consequences of severe dentofacial deformity through a combination of orthodontic and surgical dentistry’. The overall treatment goal is to produce a functionally stable and balanced occlusion, in an aesthetically pleasing and harmonious face – in addition to satisfying the patient’s wishes or desires.

The patient journey

Potential orthognathic patients will often have a series of appointments prior to the commencement of orthodontic treatment (Figure 22.10). This whole journey takes approximately 2½–3 years, with, on average, 18–20 months of presurgical orthodontics followed by roughly 9–10 months of postsurgical orthodontic treatment.

The joint consultation

The patient meets the multidisciplinary orthognathic team including:

- consultant orthodontists,
- consultant maxillofacial surgeons, and
- others (e.g. liaison psychiatrist, restorative consultants, dental hygienists, ENT surgeons, nurses and lab technicians).

Factors influencing the decision to proceed with orthognathic treatment

Patient factors

Age

This influences the timing of treatment. Combined orthodontic and surgical treatment is ideally commenced on patients in whom growth has ceased. This is normally at age 18 in females and 21 in males.

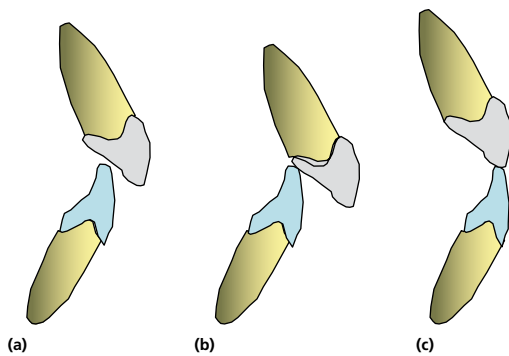


Figure 22.7 Incisor relationship as described by the British Standards Institute. (a) Class I; (b) class II; (c) class III.

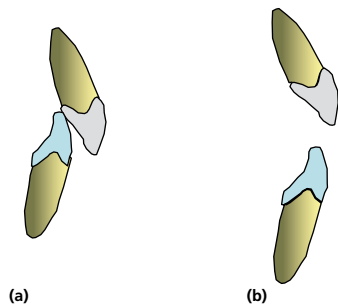


Figure 22.8 Overbite is measured relative to the incisor edges. (a) Deep overbite; (b) anterior open bite.

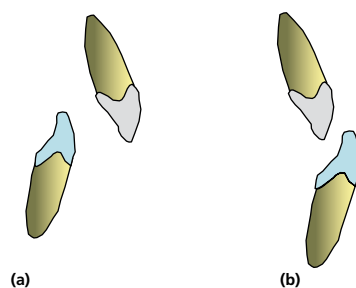


Figure 22.9 (a) Increased overjet (class II division I incisor relationship). (b) Reverse overjet (anterior crossbite).

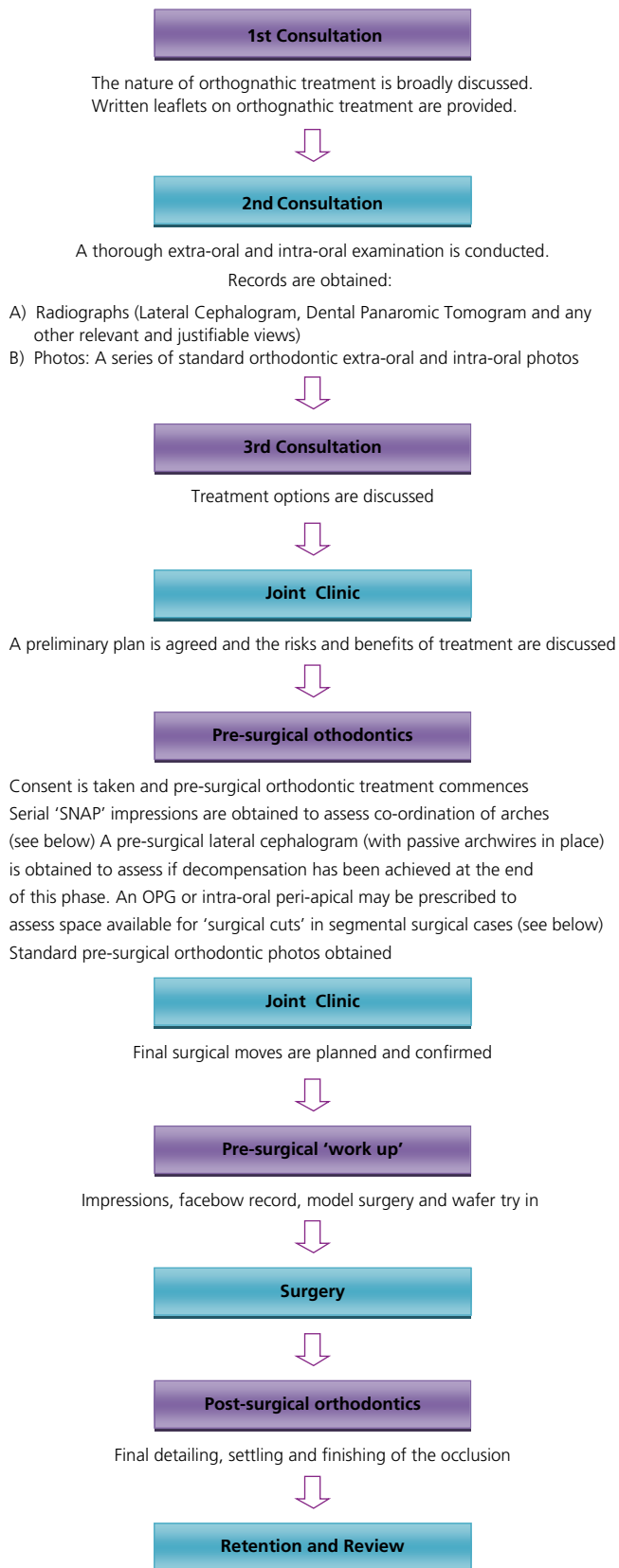


Figure 22.10 Appointments prior to the commencement of orthodontic treatment.

- Skeletal III cases tend to get worse with growth.
- Skeletal II cases (with mandibular retrognathia) often improve with growth.
- Vertical discrepancies (e.g. anterior open bite) get worse with growth.
- Transverse discrepancies become more obvious following a growth spurt.

If orthodontic treatment in a skeletal III case commences prior to the cessation of growth, the mandible may continue to develop in the anterior-posterior direction, creating a larger skeletal III discrepancy with time. Relapse of a corrected skeletal III pattern is also often a reflection of further mandibular growth.

When there is any doubt concerning changes to the extent of a developing skeletal pattern or occlusion, the options are to:

- watch and monitor,
- take serial height measurements, or
- obtain serial records (standardized photos, radiographs and study models).

Gender

Gender will influence timing and profile considerations.

Patient complaint(s), psycho-social factors and medical history

These help identify if orthognathic treatment is suitable and will be of benefit.

Clinical factors

- Severity of skeletal discrepancy in all three planes: anterior-posterior (skeletal II or III), vertical and transverse (degree of hard and soft tissue asymmetry).
- Soft tissue findings (e.g. extraoral soft tissue asymmetry, upper lip to incisor relationship at rest and smiling, gingival show on smiling, tongue and lip activity/tone, nasio-labial angle, nasal tip elevation and alar base length).
- Dental findings, such as oral health and features of the presenting malocclusion. This includes crowding, degree of dentoalveolar compensation, presence of centreline discrepancies and arch coordination.
- Anticipated stability of any treatment options will also influence the final decision. For example, a patient presenting with a retrognathic mandible on a skeletal II base, complicated by the presence of small mandibular condyles, may benefit from an advancement genioplasty and orthodontic treatment to camouflage the skeletal II discrepancy.

Other factors

The aetiology of the presenting malocclusion and findings of other investigations (radiographic/imaging and blood test results) will also help determine the suitability of any case.

Risks of orthodontic treatment

Along with the surgical risks, all patients are informed of the following risks associated with orthodontic treatment with fixed appliances:

- **Teeth:** Decalcification (permanent scarring of teeth), root resorption (root shortening), pulpitis and devitalization of teeth (inflammatory reaction affecting nerve supply to tooth).
- **Periodontium:** Gingivitis (reversible gingival inflammation), periodontitis (gum disease), recession.
- **Other:** Dental relapse, failure to close pre-existing spaces, ankylosis (failure of a tooth to move).

Most of the dental and periodontium risks can be managed with preventative oral hygiene and dietary advice.

Presurgical orthodontics

The aims of orthodontic treatment with fixed appliances are to:

- align the arches,
- decompensate arches,
- coordinate arches, or
- facilitate segmental surgery.

Aligning the arches

The aim is to straighten the teeth and eliminate crowding. Levelling of the teeth is achieved by flattening the 'curve of spee', which describes the curvature of the mandibular occlusal plane (occlusal alignment of the teeth). In anterior open bite cases, there are two approaches that may be adopted:

- The occlusal planes may be levelled presurgically and bimaxillary surgery undertaken thereafter (a Le Fort I posterior maxillary impaction $\pm$ mandibular osteotomy).
- The arches may be aligned in segments to enable segmental maxillary surgery to correct the anterior open bite along with a mandibular osteotomy.

Decompensating the arches

Dentoalveolar compensation occurs when a mismatch between the jaws in one or more planes of space is compensated by the inclination of the teeth and may be observed in all

or some of the planes. Decompensation is undertaken to eliminate features of dentoalveolar compensation and to maximize surgical moves, enhance postsurgical aesthetics and stability of occlusion.

Anterior-posterior

In a skeletal III case, the lower incisors are often retroclined and upper incisors are proclined. Conversely, in a skeletal II case, the lower incisors may be proclined.

Decompensation puts the upper and lower incisors at the correct inclination to their respective bases and removes 'compensation' for the skeletal pattern. This makes the appearance of a pre-existing class II or III incisor relationship worse preoperatively (Figure 22.11).

Transverse

A mismatch between the position of both or either jaws may result in the development of a buccal crossbite (where the upper teeth lie in a more palatal position relative to the lowers). As a result, the upper molars may be tipped buccally and lowers lingually in an attempt to obtain some contact with each other (Figure 22.12).

Vertical

In the presence of an anterior open bite, the incisors often lie in an extruded position.

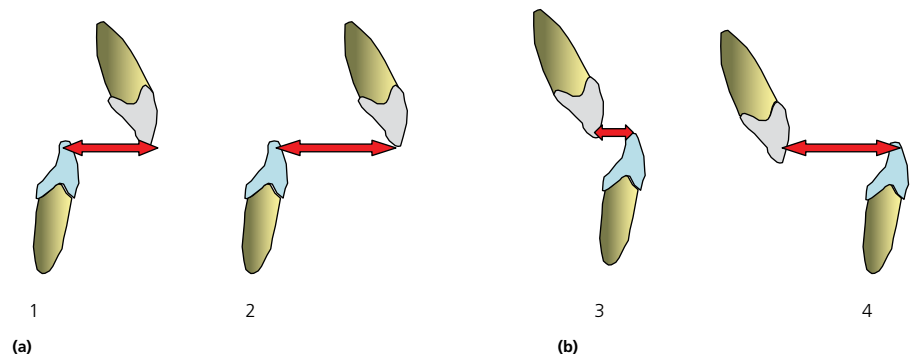
Coordination of arches

As explained above, expansion or contraction of the arches may be required to ensure the teeth intercusate and occlude appropriately once the jaws have been repositioned. Coordination of the arches is checked with serial 'SNAP impressions' of the upper and lower teeth with the fixed brace *in situ* to assess if the arches will be coordinated in all three planes.

Facilitating segmental surgery

There is normally a space requirement of 3–4 mm in between the roots and crowns of the canines and first premolars. This enables the surgeon to make the necessary cuts without damaging the teeth on either side.

Figure 22.11 Decompensation. (a) Class II division I incisor decompensation – overjet increases presurgery. (b) Class III incisor decompensation – reverse overjet increases presurgery.



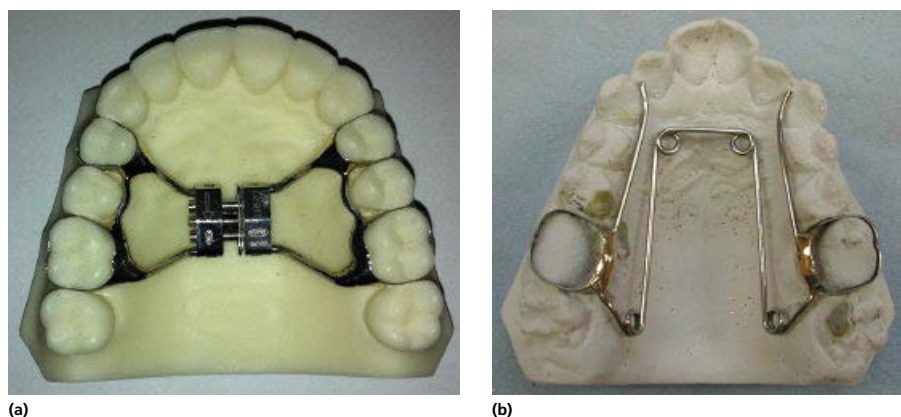


Figure 22.12 (a) Rapid maxillary expansion (RME) appliance with Hyrax screw and orthodontic bands on the upper premolars and first molars. (b) Quadhelix expansion appliance with orthodontic bands on the upper first molars and palatal expansion arms.

Presurgical 'work-up'

The presurgical work-up is undertaken once the final surgical moves have been confirmed at the joint clinic consultation. At this stage the following are obtained:

- facebow record,
- impressions of the maxillary and mandibular dentition,
- occlusal registration – products such as Occlufast (a silicone-based recording material) are often used to obtain the facebow registration.

A facebow records the relationship between the maxillary dentition and condyles using 2–3 reference points. The impressions are used to cast up models of the maxillary and mandibular arches. Model surgery is subsequently undertaken enabling the fabrication of acrylic wafers (occlusal guides). In single jaw procedures, one wafer is required to relate the mobilized maxilla to the fixed mandible. In bimaxillary cases, two wafers are fabricated. The second ensures the jaws relate to each other accurately once surgery on the mandible is complete.

Ball-hooks on the orthodontic archwires enable ligation of the wafers intraoperatively and the use of elastics in the postsurgical orthodontic phase.

Postsurgical orthodontics

Patients require written and verbal support in the immediate post-op phase.

- 1 day–4 weeks post-op: Light directional (class II or III elastics) and or settling intermaxillary elastics are used to maintain or improve the final occlusion.
- One month plus: Final finishing and detailing of the occlusion is undertaken.

Surgical techniques

Le Fort I maxillary osteotomy

The Le Fort I osteotomy was initially described as an access osteotomy. In 1867, Cheever performed Le Fort I osteotomy to aid resection of nasopharyngeal masses.^{15,16} In 1926, Wassmund

described this procedure to correct dentofacial deformity. He did not complete maxillary down fracture and instead used orthopaedic traction to achieve his results.¹⁷ Auxhausen described complete mobilization of the osteotomized maxilla in 1934.¹⁸

Modern maxillary osteotomies were popularized after anatomically based work by Bell *et al.* in 1969.¹⁹ Following this work it was realized that the maxilla could be osteotomized and segmented safely with the mucosal pedicle providing the vascular supply.

Technique

The patient is carefully draped such that the whole face can be visualized (Figure 22.13). Tumescence solution is infiltrated.

Vertical maxillary dimensions are assessed with reference to a measurement against a fixed point that will not be changed by the procedure to be performed. We prefer using the medial canthus as the fixed point.²⁰ A metal ruler is used to measure the distance to the orthodontic archwire as it crosses the lateral incisor (see Figure 22.13).

An incision is made through the mucosa to bone from the midline to the anterior edge of the zygomatic buttress bilaterally. Posteriorly, the incision is angled superiorly to maintain a wide mucosal pedicle for the mobilized maxilla^{21,22} (Figure 22.14a). The flap is raised with care taken to protect the inferior orbital nerves. Anteriorly, the muscles are left attached to anterior nasal spine and the incision is made through mucosa only (Figure 22.14b). The anterior nasal spine is removed with an osteotome and remains attached to the nasal floor, muscles and septum. The floor of the nose is elevated and protected with two Howarth's periosteal elevators (Figure 22.14c). Lateral subperiosteal dissection is extended to the posterior maxilla as far as the lateral pterygoid plates.

A reciprocating saw is used to make a bone cut parallel to the occlusal plane from the maxillary tuberosity to the pyriform aperture (Figure 22.14c), remembering that the maxillary artery is related to the upper third of the pterygoid plates. A Wolford step osteotomy is a modification that could avoid a high posterior cut and avoids ramping of the maxilla.²³ A curved osteotome is used to undertake pterygomaxillary dysjunction.



Figure 22.13 Draped patient. Note exposure of whole face and intubation with a nasoendotracheal tube. Vertical maxillary reference measurement from medial canthus to archwire being taken with a metal ruler.

Finally, before down fracturing the maxilla the nasal septum and the vertical plate of the vomer are separated from the maxilla with a guarded septal chisel. The maxilla is then down fractured with finger pressure on the incisor teeth. To mobilize the maxilla a 4' × 4' Raytech gauze swab is packed posteriorly between the osteotomy cuts around the maxillary tuberosities. This not only safely strips the mucosa but also acts as a fulcrum for the application of upwards pressure on the anterior maxilla. This is a safe and effective way of stretching the soft tissues and mobilizing the maxilla. The mobilized maxilla is placed into the surgical guide and intermaxillary fixation applied (Figure 22.14d) and stabilized against the mandible in its new anterior–posterior position. The vertical position is attained either by trimming bone for an impaction or by placement of an interpositional graft for an inferior repositioning. Care needs to be taken around the floor of the nose as the pyriform aperture may require sculpting to avoid widening of the nasal base (Figure 22.14e). The septum should lie passively and, together with the inferior turbinates, may need trimming to achieve this. Adequacy of the vertical change is assessed with reference to the presurgical measurement from medial canthus to archwire.

If a segmental osteotomy is planned, segmentalization should be performed from a superior approach. Midline cuts should be made in a parasagittal position where the bone is thin and is away from the thick midline raphe, making mobilization easier.

It is important that the mandibular condyles are seated in their fossae when the maxilla is positioned and fixed. Pressure should be placed at the angle of mandible and not the chin when holding the maxillary/mandible complex in position, applying the fixation. Titanium plates are adapted to the new bony contours of maxilla and secured on each side at the nasomaxillary buttresses adjacent the pyriform rim and the zygomaticomaxillary buttresses posteriorly (Figure 22.14f). The intermaxillary fixation is released and position of the maxilla and the occlusion is checked. Closure is with 3/0 Vicryl and, if the upper lip is short, a V-Y midline closure can be used to increase lip length.

Indications

- Midfacial hypoplasia
- Maxillary excess
- Asymmetry
- Anterior open bite correction
- Transverse discrepancy.

Limitations

- Movements greater than 10 mm are difficult and potentially unstable.
- Inferior repositioning of maxilla is unstable.
- Posterior repositioning is difficult to achieve and will reduce nasopharyngeal airway space.

Versatility

- Extremely versatile, especially segmented osteotomy.

It is possible to carry out higher level maxillary osteotomies at Le Fort II, Le Fort III and transcranial monobloc levels. However, control of the occlusion and vertical position can be difficult. In 1971, Kufner described the 'quadrangular' midface osteotomy to correct asymmetry in patients with midface recession and a normal nasal skeleton.^{24,25}

Bilateral sagittal split osteotomy (BSSO)

Mandibular sagittal split ramus osteotomy can be used to correct asymmetry as well as anteroposterior discrepancies.²⁶ Since the original description, the technique has gone through a series of modifications to enable a predictable sagittal split to be achieved. The three seminal changes are the Dalpont,²⁷ Hunsuck²⁸ and Bell-Epker^{29,30} modifications.

Development of the BSSO

The original description by Trauner and Obwegeser²⁶ describes two parallel horizontal osteotomy cuts on the ramus, one lingual and above the lingula and the second buccal and below the level of the lingula in a sagittal plane.

Dalpont (1961)²⁷ overcame the problems of poor bone contact and difficulty of accessing the posterior ramus buccally by

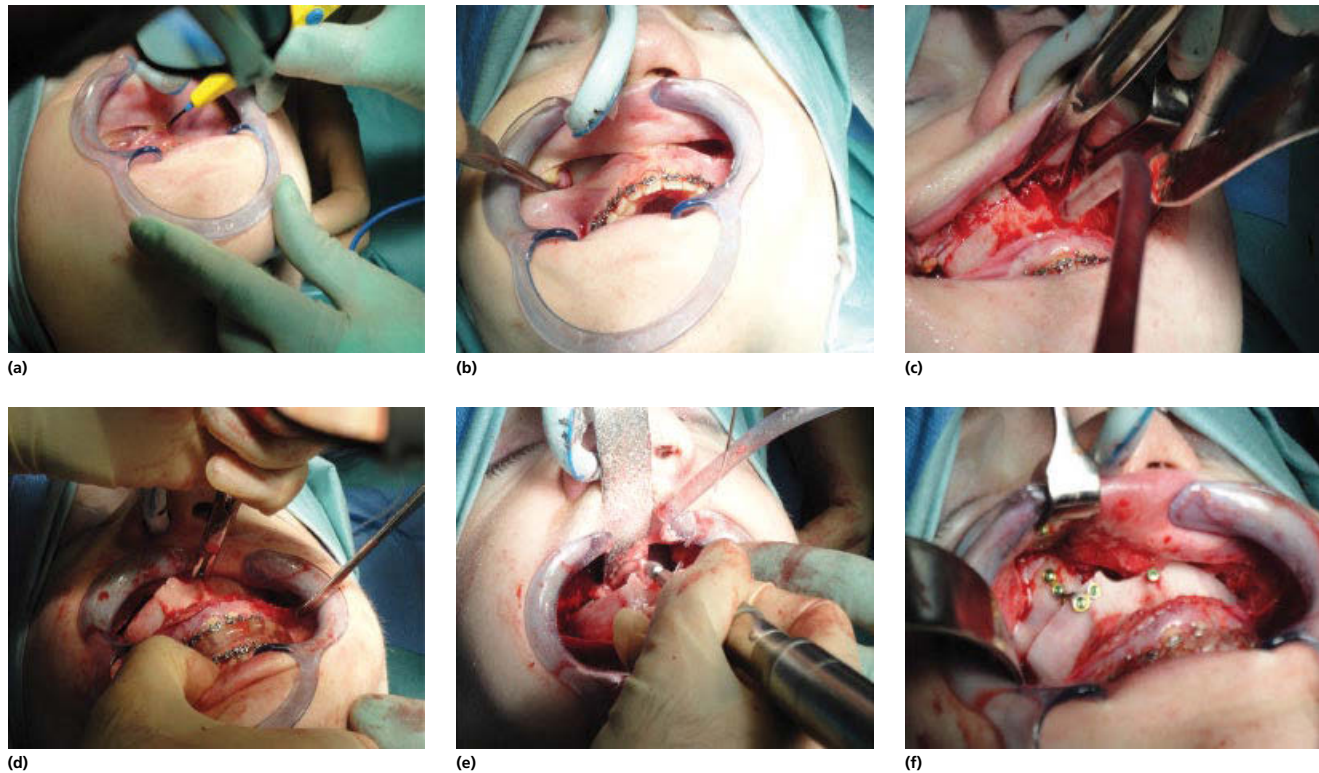


Figure 22.14 (a) Le Fort I incision. (b) Anteriorly the incision is through mucosa only in order to maintain attachments and viability of the anterior nasal spine. (c) Howarth's elevators retracting the nasal floor as bone cuts are being made. (d) Mobilized maxilla being manoeuvred into a surgical guide/wafer. (e) Sculpting of the pyriform aperture. (f) Fixation plates *in situ*.

advancing the buccal cut to the molar region. In 1968, Hunsuck²⁸ shortened the medial cortex cut by limiting it to the mandibular foramen in the lingular fossa. Bell and Schendel (1977)²⁹ and Epker *et al.*'s (1978)³⁰ further modification involved dividing both buccal and lingual cortices at the lower border of the mandible below the anterior extension of the buccal cut, resulting in a more consistently favourable split.

With the development of piezoelectric saws and endoscopically assisted techniques Sader³¹ has reverted to the original Obwegeser procedure in certain cases, requiring small moves to benefit from the lower risk of nerve injury.

The technique

A Featherstone-type mouth gag maximizes access to the lingual aspect of the ramus. If a bimaxillary procedure is to be undertaken the mandibular bone cuts are made before commencing maxillary surgery but left incomplete so the mandible remains unsplit. The mandibular osteotomy can then be completed without the use of a mouth gag, thus avoiding undue pressure on the maxilla after it has been fixed in its new position.

An incision is made on the lateral aspect of the external oblique ridge as it begins to ascend the ramus to the level of the distal aspect of the lower first molar tooth. It is important to maintain a wide mucosal pedicle to the maxilla. From an anterior, buccal approach the temporalis muscle is stripped off the

coronoid process (Figure 22.15a). The lingual dissection starts high up on the medial ramus so that the lingula is approached from above, thus retracting and protecting the inferior alveolar nerve. The position of the mandibular foramen in the lingular fossa is indicated by the almost invariable presence of perineural and perivascular fat (Figure 22.15a). Bone cuts are made according to the Dalpont²⁷ (Figure 22.15b), Hunsuck²⁸ and Bell-Epker^{29,30} modifications with a reciprocating saw. The external oblique ridge guides the position of the cuts. The saw is placed at a 45° angle with the tip blade just above and behind the lingula. The saw cut is made in a downwards direction and continued along the inner aspect of the external oblique to the first molar region where it tapers out (Figure 22.15b). The mouth gag is removed to improve access to the lower border of the mandible. A vertical cut is made through the buccal cortex only, to join with the previously made cut. The inferior dental nerve runs in the buccal cortex in 10% of patients therefore care needs to be taken with the depth of the cut.³² Finally, a saw cut through the buccal and lingual cortex at the lower border is made.

The split is completed using osteotomes and the nerve should remain on the distal, lingual segment (Figure 22.15c).

The muscles of mastication are stripped from the lower border and medial aspect of the proximal condylar segment. Once the osteotomized segments are completely mobilized, the

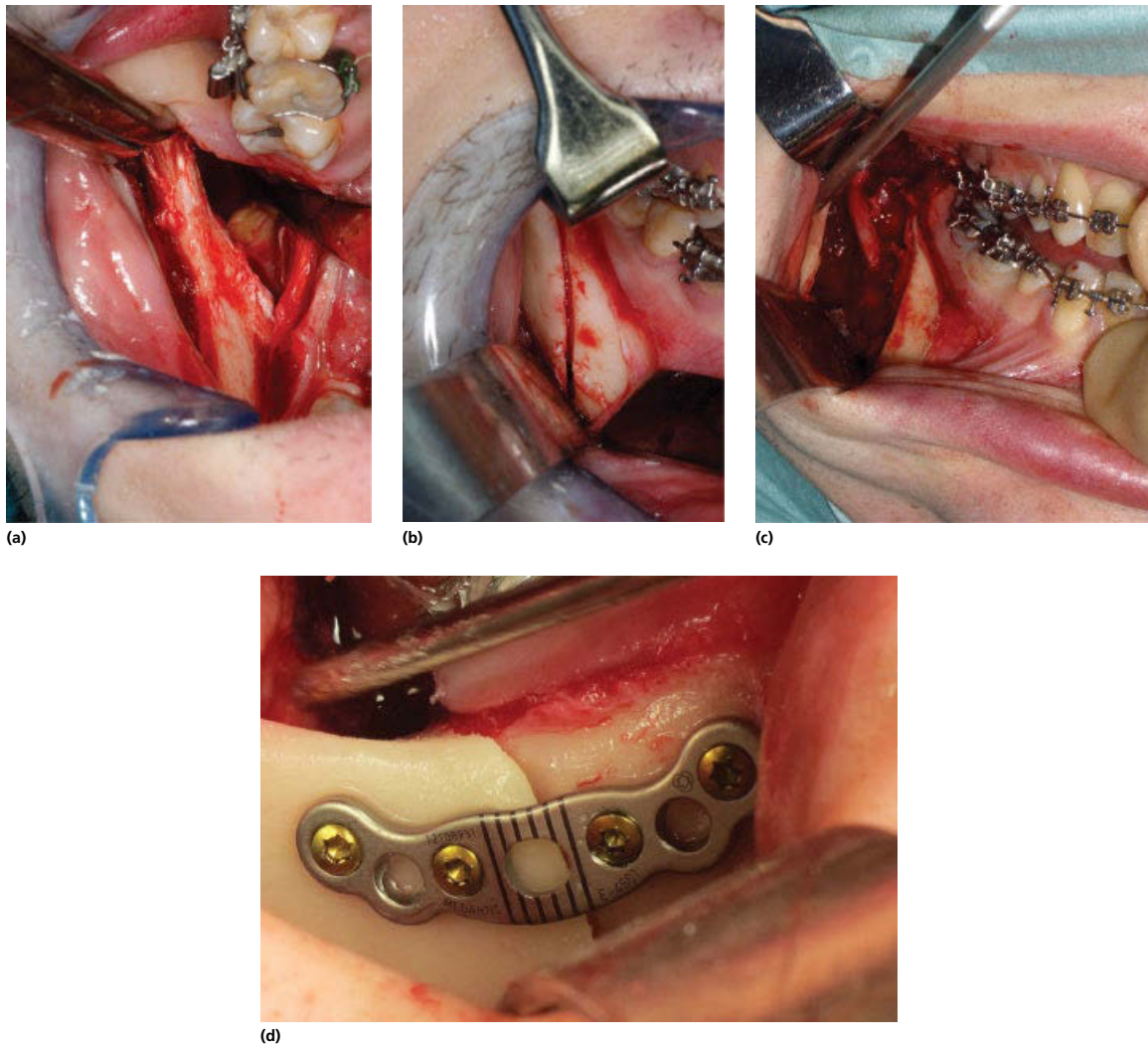


Figure 22.15 Bilateral sagittal split osteotomy. (a) Ramus exposure with medial dissection exposing the perineural fat. (b) Buccal bone cut. (c) Split mandible with inferior alveolar nerve on the distal, tooth-bearing segment. (d) Completed osteotomy with bone fixation *in situ*.

distal, lingual segment is placed in the mandibular surgical guide and intermaxillary fixation is applied. If the mandible is being set back, bone is trimmed from the proximal condylar segment prior to fixation. If counterclockwise rotation of the proximal condylar segment inadvertently occurs, resultant stretch of the pterygomasseteric sling can result in instability of the osteotomy.³³ In order to avoid this, the lower borders of the mandible should be aligned across the cut separating the proximal and distal segments when fixation is applied. Lateral torque of the condyle can be a problem, particularly with rigid bicortical or lag screw fixation. Bone fixation plates (Figure 22.15d) have been designed with a degree of lateral flexibility to allow the attached muscles some degree of freedom to attain a passive condylar position within the glenoid fossae. The anaesthetic can be lightened to provide muscle tone when the fixation is being applied. The surgical guide is removed and the occlusion checked. Closure is with 3/0 Vicryl.

Indications

- Mandibular retrognathia
- Mandibular prognathia
- Mandibular asymmetries.

Limitations

- Movements greater than 10 mm are difficult and potentially unstable.
- Setback will reduce airway and tongue space.
- Unfavourable cervico-mental soft tissue changes with setback.

Versatility

- Extremely versatile and most commonly performed mandibular osteotomy.

The most common orthognathic treatment plan involves bimaxillary Le Fort I and BSSO osteotomies. Excellent results can be achieved (Figures 22.16 and 22.17).



(a)



(b)

Figure 22.16 (a) Preoperative class III skeletal base malocclusion. (b) Postoperative results.

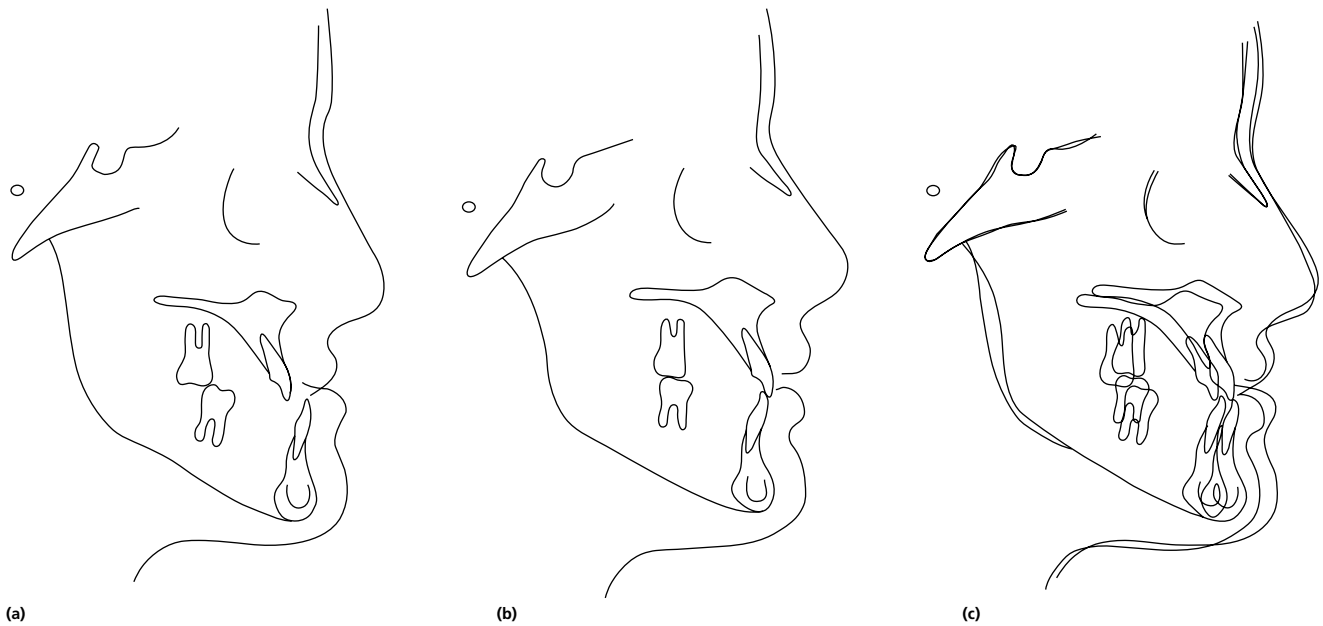


Figure 22.17 (a) Preoperative lateral cephalometric tracing. (b) Postoperative lateral cephalometric tracing. (c) Superimposed tracings showing extent of skeletal and soft tissue changes.

Other mandibular osteotomies

Ramus osteotomies

Caldwell and Letterman (1954)³⁴ described an extra oral approach to a vertical osteotomy of the mandible allowing repositioning of the ramus under direct vision. Modern vertical subsigmoid osteotomies are performed via an intra-oral approach with fibreoptic/endoscopic guidance using an oscillating saw.³⁵ Mandibular prognathism correction is achieved with minimal risk to the inferior alveolar nerve – paraesthesia is reduced by two thirds.³⁶ It is indicated in an asymmetric move where it is important to minimize proximal segment torque. In large asymmetric moves, a vertical ramus osteotomy may be performed on one side and a sagittal split osteotomy on the side of advancement. Only small advancements of the mandible are possible when mixing these two techniques.

Total subapical osteotomy

This is indicated in cases where the discrepancy lies within the dentoalveolar segment of the mandible and the relationship of the chin to the maxilla does not require alteration. It relies on being able to exteriorize and protect the inferior alveolar neurovascular bundle. The procedure is aided by the use of a piezoelectric saw. The moves are stable as the pterygo-maxillary sling is not transgressed. It is an ideal technique for the skeletal class II patient who has an acceptably positioned chin point. At least 10mm height between the lower border of the mandible and the apices of the molar teeth is required. The bone cut should be 4mm below the root apices.³⁷

Complications

In 1998, Dimitroulis³⁸ proposed a simple classification of complications related to orthognathic surgery (Table 22.1). The important intraoperative and postoperative complications are discussed below.

Relapse

Stability can be defined as the propensity for orthodontically manoeuvred teeth and osteotomized bone segments to remain in their new positions. Relapse is a tendency to revert to pre-treatment and presurgical relations.

The keys to stable, successful treatment are:

- appropriately planned and executed orthodontic preparation;
- an understanding of the mechanisms and limitations of the adaptive processes at the bone/muscle interface; and
- an appreciation of the stability of various surgical procedures^{39,40} (Table 22.2).

Some degree of relapse is inevitable, but good orthodontic preparation can minimize and sometimes mask the extent of surgical relapse.

Condylar positioning at time of fixation is also important in stability of osteotomies. It is impossible to perfectly align the condyles to their pre-osteotomy positions, and they will thus be subject to altered loading stresses. Consequently there is always some degree of adaptation and remodelling of the condyles post surgery. The degree of remodelling will relate to stability of the orthognathic treatment. Females presenting with high maxillary-mandibular plane angle, class II division 1 malocclusion with small mandibular condyles seem to be at greater risk of

Table 22.1 Classification of complications related to orthognathic surgery

Preoperative	Intraoperative	Postoperative
Communication Diagnosis	Haemorrhage Nerve injury	Acute phase <2 weeks Chronic phase >2 weeks
Laboratory work Patient preparation	Unfavourable osteotomy Jaw malposition	Relapse Neurological dysfunction Poor aesthetics Bone healing Fixation

Table 22.2 Hierarchy of stability of surgical moves³⁹

Movement	Stability	%
Maxilla up Mandible forward ^a Chin in any direction	Very stable	>90%
Maxilla forward Maxilla asymmetric	Stable	>80%
Mandible back Mandible asymmetric move	Stable with rigid fixation	>80%
Maxilla forward and mandible back Maxilla up and mandible forward Mandible back	Unstable	Major relapse is possible
Maxilla down Maxilla wider		

^aShort and normal face height only.

condylar resorption and lysis.⁴¹ The use of bicortical position screws or lag screw techniques carry a higher risk of torquing the condyles laterally, resulting in displacement of the condyles and early disruption of the occlusion and increases relapse. The use of semirigid fixation with 2 mm mini-plates and monocortical screws reduces this risk.⁴²

Minimizing stretch of the pterygomasseteric sling is also important in maximizing stability.³³ Muscle adaptation after reattachment of the stripped tissues is a complex process and the presence or relative absence of intermediate muscle fibres seems to play a role in stability.⁴⁰

Managing anterior open bites can be challenging.⁴³ This deformity is inherently unstable after either surgical or orthodontic correction and is managed with a differential maxillary impaction creating bilateral posterior open bites which can be corrected orthodontically. The problems of pterygomasseteric sling transgression are avoided. Small anterior open bites can be managed with mandibular osteotomies and counterclockwise rotation of the distal mandibular segment using rigid bicortical fixation.

Nerve injury

A wide range of incidence in postoperative paraesthesia following orthognathic surgery has been published. In one such study, overall neurosensory deficit in a sample of 46 ramus splits at the

end of one year was 15%.⁴⁴ Another author reported 8.3% deficit at one year from 218 splits.³⁷ The severity of disturbance is closely related to the measured distance between the neurovascular bundle and the buccal cortical plate. In 91% cases with severe disturbances the distance between the inferior alveolar nerve and the buccal cortical plate was less than 1.2 mm.⁴⁵ Other variables include the degree of advancement, intraoperative bleeding, presence of wisdom teeth, bad split, nerve manipulation, rigid fixation, concomitant surgery, systemic disease and age of patient.⁶

Long-term sensory deficit occurs in approximately 10% of patients undergoing BSSO.⁴⁴ Interestingly, approximately 10% of the nerves are intimately related to the buccal cortex.^{32,36} The deficit is usually partial but can unfortunately manifest as causalgia.

Plate infection

This is a common complication and literature shows wide variation in incidence. One study suggests 13.7% of 570 cases had infected plates.⁴⁶ Mandibular plates have a higher risk of infection when compared to maxillary plates.

Bleeding

Red cell transfusion is generally not required if good technique is followed and soft tissues are handled carefully. Hypotensive anaesthesia also plays a role. Mean haemoglobin drop of just 1.7 g/dL in 82 bimaxillary osteotomy cases has been reported.⁴⁷

Other

Disastrous complications reported range from unfavourable osteotomy cuts, to bleeding from maxillary artery injury, blindness and even avascular necrosis of part or all of the jaw.⁴⁵

Advances in orthognathic surgery

Endoscopic approach and fixation

Advances in instrument development, for example, right-angled saws and screwdrivers and more widespread use of the endoscope in head and neck surgery raise the possibilities for endoscope-assisted osteotomies.⁴⁸ Possible indications might include endoscope-assisted vertical subisigmoid osteotomy in cases requiring mandibular setback where preservation of lower lip sensation is paramount. A two thirds risk reduction to the integrity of the inferior alveolar nerve can be achieved.³⁶

Piezoelectric saw

Piezoelectric, ultrasonic bone surgery systems utilize a selected frequency range between 28 and 36 kHz. They are active on mineralized tissues only, thereby limiting the risk of soft tissue injuries to anatomically important structures such as nerves, blood vessels, dura and Schneiderian membrane.

The modulated piezoelectric signal allows tissue relaxation and very high precision cutting. Narrow, smooth bone cuts in

the presence of irrigation result in less risk of hard tissue necrosis and bone trauma and allow for faster bone healing.

Piezoelectric systems are likely to take their place as technology that is complementary to manual and motorized cutting instruments. They may be of particular use in segmental osteotomies, total subapical mandibular osteotomies and also for the buccal cut in BSSO to minimize risks to the inferior alveolar nerve and adjacent teeth.³¹

Summary

An understanding of the differing abilities of patients to adapt to change, both physical and psychological, and the complex relationship of hard tissue and soft tissue changes is required to obtain optimum results in orthognathic treatment.

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CHAPTER 23

Facial reanimation

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Introduction

Facial paralysis presents a complex challenge to the reconstructive surgeon. Since Sir Charles Bell first described the course of the facial nerve in 1821, our understanding has led to the development of the vast array of surgical options to treat patients with facial paralysis. The fundamental problem when considering facial palsy is that there is currently no conceivable way to restore the 17 muscles of facial expression successfully. The second challenge facing the surgeon is that no patient is the same; not only the aetiology behind facial paralysis dictates which operations are available, the age group, the anatomy available and, most importantly, what the patient wants, all make the decision-making process vital.

The presentation of facial nerve paralysis also adds to the conundrum, ranging from isolated marginal branch palsy to complete bilateral palsy. The approach to the management of any patient with facial paralysis should be to break the face down into three units: the forehead, the eyes, and the mid- and lower face. In a young child, the skin quality and soft tissues are far superior to that of an elderly patient, thus problems encountered are different. Frequently, there is minimal asymmetry at rest and therefore focus needs to be directed at restoring dynamic function. In the elderly patient, skin elasticity is poorer and when combined with wasting of the soft tissues and muscles, the asymmetry at rest may be strikingly obvious and static treatment options may yield the desired outcome.

What is the importance of facial palsy reconstruction? The impact facial paralysis has on an individual can be devastating and can be broken down into functional and psychological factors.^{1,2} If a patient is unable to close their eye and the cornea is insensate, the globe will dry, leading to ulceration and ultimately blindness. Diminished oral continence, poor speech and obstructive nasal breathing are other functional problems encountered. A child who is unable to smile or convey emotion effectively may be subject to bullying and psychological trauma, and an adult with profound asymmetry at rest may be perceived as being angry, causing the patient to become socially recluse.

The aim of this chapter is to give the reader a strategy on how to approach facial paralysis. The anatomy of the facial nerve and other important structures of the face will be reviewed as this provides the foundation on which the evolutions of surgical techniques have been based. The information provided should enable a surgeon to have a methodical approach to patient management but it must be stressed that the information should be used as a guideline and that each patient will require a customized approach to a greater or lesser extent. To the reconstructive surgeon it combines the epitome of what we seek to achieve: a functional outcome where one has to consider the aesthetic result as the face is the centre of human interaction.

Anatomy of the facial nerve

The facial nerve (CN VII) develops embryologically from the second pharyngeal branchial (hyoid) arch. The motor division derives from the basal plate of the embryonic pons, whereas the sensory division arises from the cranial neural crest. The sensory portion, known as the intermediate nerve, is responsible for supplying taste to the anterior two thirds of the tongue, secretory and vasomotor fibres to the lacrimal, submandibular and sublingual glands, the mucous membranes of the nose, mouth and, finally, sensation over the external auditory meatus and posterior skin of the ear. Of clinical importance, the supranuclear innervation is bilateral to the muscles of the forehead and eyes, but only contralateral to the muscles of the lower face; this accounts for the sparing of the forehead muscles in cases of supranuclear lesions in comparison to nuclear or infranuclear lesions. The facial nerve arises from the brainstem at the junction between the pons and medulla oblongata with the intermediate nerve and is closely related to the abducens (CNVI) and vestibulocochlear nerves (CNVIII), which lie in close proximity. The course of the nerve can be simply divided into intracranial and extracranial.

Intracranial course

As the facial and intermediate nerves leave the brainstem, they course laterally towards the cranium, entering the petrous temporal bone through the internal auditory meatus, travelling with the acoustic and vestibular nerves for approximately 8–10 mm. The facial nerve then enters the fallopian canal by itself which is subdivided into three segments and is approximately 30 mm long. The labyrinthine segment is the narrowest and gives rise to the greater petrosal nerve, from the geniculate ganglion, to innervate the lacrimal gland parasympathetically. Next is the tympanic segment and the junction between the two is of clinical importance as the acute angle formed between the segments is a site where shearing can commonly occur. The mastoid segment is the third segment before the nerve arises through the stylomastoid foramen.

The second branch of the facial nerve within the facial canal is the nerve to stapedius, providing motor innervations of the stapedius muscle. The final branch is the chorda tympani, responsible for taste sensation in the anterior two thirds of the tongue; these branches are useful when examining the facial palsy patient as the clinician can determine the site of nerve injury based on end-organ functionality.

Extracranial course

After emerging from the stylomastoid foramen, the facial nerve passes through the parotid gland. The most immediate branch given off is the posterior auricular nerve, innervating the small muscles of the posterior ear and branches to the posterior belly of the digastrics muscle and stylohyoid muscle. The nerve divides into temporo-fascial and cervico-fascial divisions approximately 1.3 cm from the stylomastoid foramen. A useful clinical landmark for locating the trunk is at a point slightly anterior and inferior to the tip of the tragus; the nerve typically lies between 1 and 1.5 cm deep to this. Another consistent landmark is the course of the frontal branch, which travels from 0.5 cm below the tragus to 1.5 cm above the lateral brow.^{3,4} The zygomatic branch is frequently located 1 cm above the zygomatic arch and the buccal branch 1 cm below, whereas the marginal mandibular branch runs 1 cm below the inferior border of the mandible. The facial nerve separates into the five main branches at the pes anserinus.

The temporal branch courses cranially to cross the zygomatic arch within the superficial musculoaponeurotic system (SMAS), lying deep to frontalis but superficial to the deep temporalis fascia, coursing within or just deep to the temporoparietal fascia, SMAS continuation in the temporal region. The temporal branch is responsible for the innervation of frontalis, orbicularis oculi and corrugators supercilii. An important efferent limb from this branch is responsible for the corneal reflex.

The zygomatic branch proceeds cranially, angled towards the lateral angle of the orbit. Here it supplies the orbicularis oculi. Multiple branches passing lateral and medial to the zygomaticus major, as they radiate from the main trunk into the orbicularis oculi muscle. It is vital for not only for the

lower lid tone but also the blink reflex. This commences medially and spreads laterally.

The buccal branch is the largest branch and passes forward below the orbit and divides into deep and superficial divisions. The superficial branches lie superficial to the SMAS and supply procerus. The deep branches pass beneath zygomaticus and quadrates labii superioris, supplying them both. Further deeper branches go on to supply buccinator and orbicularis oris.

The marginal mandibular branch courses beneath the SMAS towards the chin, passing beneath depressor angulis oris (which it supplies). Here it supplies the depressor labii inferioris and mentalis.

The cervical branch is the most inferior branch and passes towards the neck over the mandible beneath the SMAS. It primarily innervates the platysma. Of the five main branches, the marginal mandibular is most susceptible to injury during facial dissection due to its variable branching pattern and the multiple procedures performed in this area.^{5,6}

There is much variability in the branching patterns of the facial nerve and numerous interconnections between them and caution must always be exerted when making a surgical approach to expose the nerve. Davis *et al.*⁷ described the multiple branching patterns of the facial nerve in 350 dissections and concluded that a consistent finding was that both frontal and marginal mandibular nerves are end-nerves.

There are 23 facial muscles, of which 17 are activated in facial expression. When considering the muscles involved with producing a smile, the most important are zygomaticus major, depressor anguli oris and the buccinators. Broadly speaking, the musculature of the face can be divided into four layers according to Freilinger⁸ from superficial to deep:

- Layer 1: Depressor anguli oris, zygomaticus minor, orbicularis oculi
- Layer 2: Depressor labii inferioris, risorius, platysma, zygomaticus major, levator labii superioris, alaeque nasi
- Layer 3: Orbicularis oris, levator labii superioris
- Layer 4: Mentalis, levator anguli oris, buccinators.

Anatomy of potential donor nerves for facial reanimation

When discussing the anatomy of the facial nerve it is worth spending some time considering other local nerves of the face. Frequently, another donor nerve is required when either there is absence of a functioning facial nerve or if it is inappropriate to consider its use. Here, three other potential donor nerves will be considered.

Masseteric nerve

The masseter is one of the principal muscles of mastication. It arises from the zygomatic arch and the maxilla and inserts into the coronoid process and the ramus of the mandible. The muscle is divided into a superficial and deep portion and is innervated

by a branch of the mandibular division of the trigeminal nerve (CN V). The masseteric nerve arises from the anterior division of the mandibular nerve, which arises from the cranium through the foramen ovale. The masseteric nerve passes laterally in front of the temporomandibular joint lying deep to the temporalis tendon. It crosses the mandibular notch with the masseteric artery and enters the masseter on its deep surface and branches within the muscle, reaching the anterior border. The nerve can be found intraoperatively by delicately spreading the muscle fibres of the masseter which are oriented downwards and backwards superficially, then downwards and forwards in the deep portion of the muscle. It is important to isolate suspected branches and use a nerve stimulator to confirm its function.

Hypoglossal nerve

The hypoglossal nerve (CN XII) nucleus is located in the medulla oblongata. It passes through the cranium to emerge from the hypoglossal canal. The extracranial course is closely related to the vagus nerve and passes between the internal carotid artery and jugular vein, lying on the carotid sheath. It passes deep to the posterior belly of the digastric muscle and enters the submandibular region, coursing lateral to the hyoglossus muscle and inferior to the lingual nerve before it branches to innervate the tongue. It innervates all of the muscles of the tongue except palatoglossus, which is innervated by the vagus nerve. When considering using the hypoglossal nerve as a potential donor nerve, it is advocated that a terminal branch is used to minimize any loss of function.

Spinal accessory nerve

The spinal accessory nerve (CN XI) is responsible for the innervation of the sternocleidomastoid and trapezius muscles. The nerve is unlike other cranial nerves in the fact it arises from the upper cervical spinal cord and enters the skull through the foramen magnum. Its intracranial course leads it to emerge from the jugular foramen. In the neck it crosses the internal jugular vein at the level of the posterior belly of digastric muscle and continues to travel caudally, piercing and supplying sternocleidomastoid and then terminating to supply trapezius.

Anatomy of potential recipient vessels for functional muscle transfers

The arterial supply of the face is predominantly via branches of the external carotid artery and the ophthalmic artery. Consideration here will be given to the branches, which arise from the external carotid artery as these are of more practical use when free muscle transfer is proposed. The three main facial arteries most suitable for arterial anastomosis are:

- the facial artery,
- the superficial temporal artery, and
- the transverse facial artery.

The facial artery arises in the carotid triangle just distally to the lingual artery behind the ramus of the mandible. It passes deep to the digastric and stylohyoid muscles and along the posterior border of the submandibular gland, superficial to the hypoglossal nerve. At the point of the antero-inferior angle of the masseter it crosses over the body of the mandible and proceeds tortuously towards the angle of the mouth. In the face it branches to give rise to the inferior and superior labial arteries and terminates as the angular artery and the lateral nasal artery.

The superficial temporal artery arises from the external carotid artery when it bifurcates to also give rise to the maxillary artery. The superficial temporal artery originates at the level of the parotid gland behind the neck of the mandible. It then courses cranially just anterior to the tragus, crossing superficially to the zygomatic process and is covered by the auricularis anterior muscle and a dense fascia. The temporal and zygomatic branches of the facial nerve cross the artery and it is accompanied closely by the auriculotemporal nerve which lies immediately posteriorly. It terminates by branching to form the parietal and frontal arteries.

The transverse facial artery arises from the superficial temporal artery before it emerges from the parotid gland. It travels transversely across the face, running between the zygomatic arch superiorly and the parotid duct inferiorly superficial to the masseter muscle.

When planning free functional muscle transfer (FFMT), good knowledge of the vascular supply is critical. Facial anatomy is by no means consistent and frequently secondary options are required; even in some cases direct end-to-side anastomosis to the external carotid artery may have to be considered and preoperative assessment with a Doppler can be helpful.

Aetiology of facial paralysis

Congenital

Birth trauma

There is an incidence of 1.8 out of every 1000 children born with facial nerve paralysis in North America.⁹ Several factors increase the chance of facial nerve injury during childbirth, including instrumentation, birth weight over 3.5 kg and primigravida. The exact mechanism of injury is unknown but likely to be caused by traction being placed on the nerve. The prognosis is excellent, with over 90% of children recovering completely without intervention.¹⁰

Mobius syndrome

Mobius syndrome is characterized by concomitant bilateral facial nerve and abducens nerve (CN VI) palsies. This leads to complete, bilateral facial paralysis leading to extreme difficulty in expressing emotion as well as oral incontinence and social ostracism.

CHARGE syndrome

The major features of CHARGE syndrome are: coloboma, chonal atresia, cranial nerve abnormalities and auditory anomalies. The minor features include distinctive facial dysmorphism, facial

clefting, tracheoesophageal fistula, congenital heart defects, genitourinary anomalies, developmental delay and short stature. Although not all anomalies may be present, at either least four major features or three major and three minor features must be present.

Hemifacial microsomia

Hemifacial microsomia covers a variety of congenital facial anomalies which arise as a lack of development of one side of the face. It is characterized by a hemifacial soft tissue deficiency and poor development of the mandible, maxilla and external ear.

Infectious

Bell's palsy

Bell's palsy represents 80% of the caseload of a facial palsy surgeon.¹¹ There is no known causative factor, although there is evidence that herpes simplex virus plays a role. Incidence is around 30 cases per 100 000 per year, and is slightly higher in pregnant women (45 per 100 000). Eighty per cent of people will completely recover within 2 years. Of the 20% remaining, the problem ranges from having a complete dense facial paralysis to synkinesis.

Ramsey–Hunt syndrome

This syndrome is caused by varicella zoster virus involving the facial nerve. It is distinguished clinically from Bell's palsy by the presence of vesicles and pain in the ear, along with a myriad of other less common symptoms. Although far less common than Bell's palsy, the prognosis is worse and most patients do not recover from the facial paralysis and develop chronic neuralgia.

Otitis media/mastoiditis

An acute infection of the middle ear or mastoid bone can very rarely lead to facial nerve paralysis. This is likely to be secondary to localized inflammation and compression of the nerve in the facial canal. Prompt recognition and treatment yields favourable outcomes and in select cases mastoidectomy may be indicated.

Cholesteatoma

A cholesteatoma is a non-malignant slow-growing skin cyst. It causes destruction of the inner ear through chronic infection and by local pressure effects as the cyst grows. Facial paralysis develops in less than 3% of cholesteatomas.

Lyme disease

Lyme disease is caused by *Borrelia burgdorferi*, which is transmitted to humans by infected ticks. The symptoms are initially non-specific, including headache, malaise and fever; however, facial paralysis can develop in up to 11% of cases. The prognosis is excellent, with more than 99% of patients going on to full recovery without surgical intervention.

Traumatic

Trauma to the face or cranium represents the second most common cause of acquired facial palsies. In blunt trauma with no associated bony involvement, the type of injury is likely to be a neuropraxia and full recovery is to be anticipated. A penetrating injury where there is a transection of the nerve warrants urgent surgical exploration and an attempt at primary nerve repair within 3 days. Beyond this the muscles can no longer be stimulated due to prolonged leakage of acetyl choline from the severed nerve ends. If the wound is grossly contaminated, it is prudent to tag the severed nerve ends, which will allow for easy identification beyond this timeframe and allow for delayed repair. When there is an injury of the temporal bone the prognosis is less favourable unless the onset of the facial palsy is delayed. In temporal fractures the incidence of associated facial nerve injury is between 5 and 10%, of which 40% will recover poorly. In this group of patients, the diagnosis of a facial nerve injury may be difficult due to other intracranial injuries, making the assessment difficult or impossible.¹² Iatrogenic injuries may occur with soft tissue, bony or intracranial surgery.

Neoplastic

The facial nerve may be prone to compression at any point along its course by a space-occupying lesion. The most common cause is secondary to compression by an acoustic neuroma followed by glomus, facial neuromas and carcinomas (skin as well as salivary gland). Manipulation of the facial nerve is frequently required in the removal of these tumours, which may lead to further injury.

Systemic and neurological

A brainstem injury may result in permanent facial paralysis due to disruption of the motor nucleus. A central facial palsy may be caused by a lacunar infarct that affects the fibres of the internal capsule going to the facial nucleus. The nucleus itself may be prone to ischaemic injury secondarily to an infarct of the pontine arteries.

Many systemic diseases may lead to facial paralysis as a consequence of disease progression. Conditions include multiple sclerosis, Guillain–Barré syndrome, autoimmune disease and diabetes. Treatment of the underlying cause is the primary management in this category.

Clinical examination

Clinical examination of the facial nerve is best undertaken in a logical routine, working from the top of the face downwards. Most pertinent information can be ascertained by observing a patient as you greet them and they talk. Resting asymmetry may be difficult to assess in the paediatric population and therefore asymmetry may only become apparent during interaction with a child and attempting to get them to smile or speak.

Beginning at the brow, look for asymmetry of the wrinkles of the forehead at rest and then ask the patient to raise their

eyebrows to exaggerate this. A particular area, which requires close examination, is the eye. Ask the patient to close their eyes as tightly as possible. The upper eyelid predominantly achieves eye closure and the lower eyelid acts to maintain the lid margins in contact with the globe and assist tear drainage. Begin with assessing the condition of the globe; for signs of epiphora, corneal irritation or ulceration. During the introductory period of the consultation it should be possible to see if the blink reflex is still intact and whether the Bell's reflex is effective. The height of the palpebral aperture should be compared between the two sides and measured during eye closure. When assessing the lower eyelid, the position of the lacrimal duct should be assessed as it normally should be lying pressed to the globe, however, when there is a moderate degree of ectropion it may be ineffective at lubricating the globe, leading to epiphora. The assessment of corneal sensation is paramount as an insensate eye, which is unable to close effectively, carries a high risk of corneal damage, ulceration and scarring which may ultimately result in blindness.

The mid-face must be examined at rest and during animation. The position of the nose, alar symmetry, the presence of a nasolabial fold, the position of the philtrum and mouth must all be noted at rest. This should be repeated while asking the patient to perform a closed mouth smile, an open mouth smile and while attempting to show all their teeth. Internal examination of the mouth is prudent as it will give the surgeon a better idea of dental hygiene and the presence of dental caries. Functional assessment of speech is performed by asking the patient to repeat labial sounds of 'b', 'p' and 'm'. Patients are also asked to pucker their lips and blow out their cheeks.

Not all patients will have a dense unilateral palsy and more subtle signs should be sought. The presence of synkinesis is particularly troublesome for patients, and the most common symptom is involuntary eye closure during smiling. Dyskinesis may also be present and signs of involuntary muscle twitching must be noted. Other potential cranial donor nerves should also be examined if relevant to the case.

A thorough discussion with the patient should then be performed if possible to elicit their symptoms. Typically this will start with the patient listing the symptoms in order of severity. In the paediatric patient this discussion mainly involves the parents and what they have noticed. It is often useful to ask the parents to bring photos or videos of the child from home as they may be somewhat apprehensive during the consultation. Parents can often feel negative emotions of guilt if their child has difficulty smiling; it is important to consider the psychological support a family may require before, during and after treatment of their child. In the adult group, they may report functional or psychological symptoms; eye-watering, drooling, difficulty eating or poor speech may be the primary concern. For some, the psychological implications of not being able to smile may be more concerning or the feeling that they are being perceived as angry or hostile may lead them to social isolation.

It is important to ascertain the patient's or parents' expectations and modify this at an early stage if appropriate. A tailor-made

patient-focused approach usually provides the most successful outcomes. After the consultation it is useful to ask the patient to have both photographic and video documentation. Each unit varies in their requirements, but this serves as a vital tool in assessing outcomes of intervention.

Many scoring systems have been proposed over time and the general consensus is that there is no ideal system. The most popular is the House–Brackmann scale, originally described in 1985 and modified in 2009 to form the basis of the Facial Nerve Grading System 2.0.¹³ It is a widely accepted system, simple, sensitive, accurate and reliable, which grades facial function in six steps from normal (HB I) to total paralysis (HB VI).

Diagnostic studies

Diagnostic studies can play a critical role in the assessment of cranial nerves either preoperatively, postoperatively or during the monitoring nerve recovery. If there is any doubt regarding a facial nerve paralysis or a potential donor nerve paralysis, it is prudent to perform further testing.

Radiological imaging in the form of a CT scan or MRI scan can be useful to exclude a space-occupying lesion. Typically this has normally been performed prior to referral. Physiological MRI scans can be used to assess cortical remodelling for situations where relearning how to use a nerve to carry out a different function needs to be assessed. If the facial palsy is part of a syndrome, it is sometimes prudent to perform an ultrasound scan to confirm the presence of a donor muscle; in second arch syndromes extensor digitorum brevis (EDB) may be absent.

Several electrophysiological tests can help guide the clinician. Electromyography (EMG) is most commonly used to assess the function of different branches of the facial nerve or potential donor nerve in comparison to the normal side. EMG is often combined with electroneurography (ENoG), which assesses nerve conductivity in comparison to muscle activity. The nerve excitability test (NET) is used mainly to determine the prognosis of nerve recovery in unilateral facial nerve palsy. The facial nerve is stimulated at the angle of the jaw with increasing current until a barely perceptible twitch is seen in the face. This is compared with the normal side and a difference of more than 2–3.5 mA is considered abnormal. Due to the invasive nature of these tests, patients may not be able to tolerate the investigation, particularly in the paediatric group.

Management

When considering the management of facial paralysis the clinician must have a set of priorities to which they adhere. First, protection of the eye is paramount. Drying out of the globe can lead to corneal ulceration and ultimately blindness if untreated. Second, oral continence and speech should be restored. Third, facial symmetry should be restored to address the aesthetic

appearance of the patient and consequently, the psychological impact of the paralysis.

Patients should be treated differently on an age criteria. In the paediatric group the eye and forehead do not usually require surgical intervention and the focus of attention should be on smile restoration. In the ageing patient, treatment options begin to become more limited as the ability of a nerve to regenerate diminishes with age as does the chance of retraining the brain if a different nerve is used. The gold standard should be to recreate symmetrical spontaneous movement; this may not be possible in all cases, and many treatment options exist to help improve the circumstance.

Broadly speaking, the management options can be broken down into non-operative and operative treatments. When considering the operative management this can further be subdivided into static or dynamic options. Static treatments will produce improve resting symmetry but cannot provide reanimation of the face, and are useful tools commonly reserved for the elderly population where skin quality is poor and there are more frequent comorbidities which exclude multiple lengthy operations. Dynamic options provide reanimation of the face and involve either local muscle transpositions or FFMTs.

Non-operative management

In cases where a spontaneous recovery is expected, this represents the mainstay of treatment. In permanent partial or complete facial paralysis, this is a key adjunct to the operative management and is by no means exclusive. In patients complaining of difficulty in eye closure at night, particularly in the absence of Bell's phenomenon, simple taping of the eyelids closed may be all that is required to relieve the symptom. It is crucial to keep the eye hydrated and this may be achieved by using topical eye-drops or lubricants.

Botulinum toxin type A provides a reversible chemical method of denervation and may be used for two different patient groups: first, in patients complaining of asymmetry and second, those complaining of synkinesis. When focusing on brow asymmetry or overactivity of the depressor, Botox is often the initial first step in a patient's management as it is minimally invasive, provides rapid visual results and is reversible. The dose is variable between patients and that it should be administered to the unaffected side when attempting to correct symmetry. Typically, patients achieve a maximum response at two weeks and the dose should be repeated every three months and titrated as necessary. Botox is effective in treating synkinesis, and the affected muscle groups may be directly injected to diminish their action. Dyskinesis is what a patient will complain of if there is involuntary twitching of muscle groups as they recover or fail to completely recovery. Botox is administered directly, typically requiring a lower dose, to the affected muscle groups and similarly repeated every three months and titrated as necessary. The key is to avoid paralyzing the muscle as this may increase asymmetry and lead to an unhappy patient outcome; if this occurs, simple reassurance is all that is required.

Operative management

Treatment in children

Since the first description of a vascularized, innervated functional muscle transfer by Harii in 1976,¹⁴ a two-stage reconstruction has become the gold standard, especially in children. In the first stage, a cross-facial nerve graft is connected to a functioning terminal branch of the facial nerve, typically a buccal branch, on the unaffected side and tunnelled subcutaneously under the upper lip to the affected side (Figure 23.1a). The donor nerve ideally should leave minimal deficit therefore a pure sensory nerve is used, usually the sural nerve from the leg. The nerve graft is then allowed to regenerate across the face, which normally takes around 3–6 months and is monitored clinically with Tinel's sign. This may be unreliable in young children as they may not fully understand the test. Also, Tinel's sign is a more sensitive indicator of sensory nerve regrowth rather than motor nerve, therefore it is important to educate the parents that this is not necessarily a sign of surgical failure. In the second stage of the surgery the patient then receives a vascularized muscle flap that will be innervated by the cross-facial nerve graft over a 6- to 12-month period (Figure 23.1b). Typical muscles used are the pectoralis minor,¹⁵ gracilis¹⁶ and latissimus dorsi, as these leave as little donor morbidity as possible. At this stage the patient will slowly regain the movement on the affected side of the face.¹⁷

In bilateral congenital facial paralysis, the facial nerve is not available as a motor nerve to drive the new muscle, therefore, a different cranial nerve may be used (e.g. V, IX or the XII). A muscle must be chosen with an appropriate pedicle length and excellent results can be obtained using latissimus dorsi or gracilis.¹⁸ This operation can be performed in one stage, with the muscle flap being raised while the face is being dissected to expose an appropriate vascular pedicle and donor nerve before proceeding to the other side (Figure 23.2). It is preferential to operate on these cases when they are young as the effect on cortical remodelling and relearning is beneficial.

When embarking on facial reanimation in children it is important to consider the impact the surgery and hospital appointments will have on a family and the child. The average time taken to complete the treatment is in the region of 2 years and this should ideally be completed before educational commitments become affected and before the child becomes fully aware of their palsy and the psychological implications begin to take hold. Surgery can be started from the age of 4 years and it is beneficial to allow time for the family to consider the surgical options in detail, therefore early referral is prudent.

Management in adults

When considering the operative management of an adult with facial paralysis it is best to think of the treatment options by breaking the face down into logical subunits consisting of the brow, the eye and the mid- and lower face, separating these into static and dynamic options.

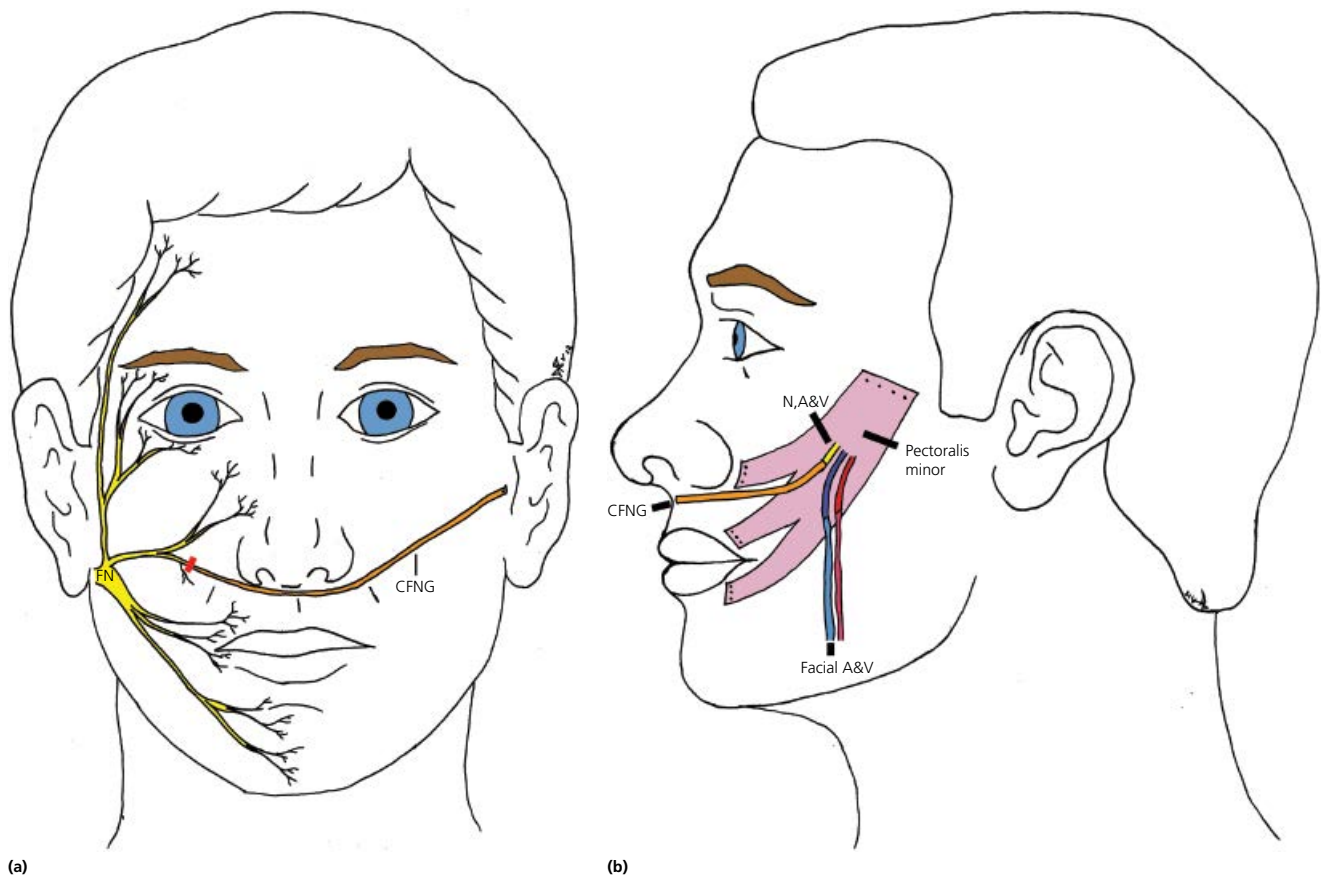


Figure 23.1 Stage 1 and 2 facial reanimation. (a) A cross-facial nerve graft (CFNG), usually a harvested sural nerve, is joined to a functioning buccal branch of the facial nerve (FN) on the contralateral side and tunnelled subcutaneous to the preauricular area on the affected side. (b) A free functional pectoralis minor flap is raised through an axillary approach with its neurovascular pedicle. The flap is inset on the affected side where the thoraco-acromial artery and vein (A&V) are anastomosed to the facial artery and vein (FA&V) and the medial pectoral nerve is connected to the CFNG.

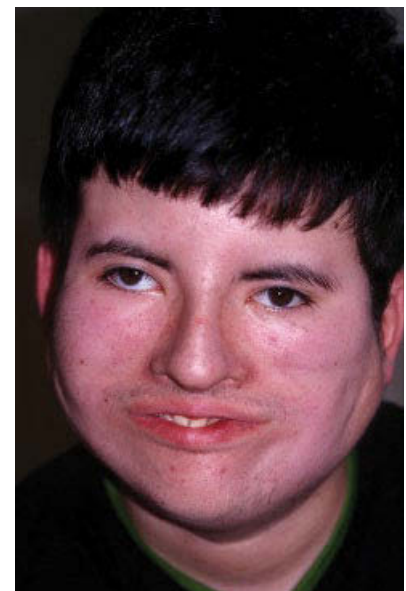


Figure 23.2 Mobius syndrome. (a) Preoperative photograph showing bilateral facial paralysis. (b) Postoperative photograph after bilateral facial reanimation using latissimus dorsi and the masseteric nerve.

Management of the brow

The only management options available for the brow are static; resting symmetry is the goal of treatment. Correction of brow ptosis may be approached surgically either directly or via an endoscopic approach.¹⁹ Direct brow lift incisions include brow, coronal or mid-forehead approaches. Dermadesis or a direct brow lift through a superciliary brow incision are well recognized as the favoured method in the setting of facial paralysis and can correct large discrepancies at the expense of a visible scar. An ellipse of skin and frontalis muscle is excised and pexy of orbicularis to the periosteum improves durability of the lift. The supraorbital nerve is at risk and must be carefully preserved but even so, patients often complain of numbness. A coronal brow lift is approached through an incision in the scalp 4–6 cm posterior to the anterior hairline extending to the pinna. Elevation in the subgaleal plane is performed and 1–2 cm of scalp is removed by incising anterior to the initial incision. The scars are usually inconspicuous but the amount of lifting is much less than the direct brow incision and patients often complain of paraesthesia posterior to the incision. A receding hairline, or family history of male-pattern baldness, may be a relative contraindication to a coronal brow lift, in which case a mid-forehead brow lift through a deep brow rhytid may be more appropriate.

Endoscopically assisted brow lifts may be useful for small amounts of brow ptosis and are therefore not satisfactory in older individuals with significant ptosis. In contrast to the direct methods involving skin excision, endoscopic brow lift suspends the periosteum in an elevated position until adherence by healing is obtained. Elevation of both brows and the complete frontal region appears necessary to address the hyperfunction observed in the unparalysed side, producing the best symmetry. An alternative is to weaken the contralateral normal frontalis muscle by frontal nerve denervation with or without frontalis muscle resection or chemodenervation with Botox. Preservation of the supratrochlear and supraorbital nerves is also achieved by direct visualization endoscopically and incisions are smaller with less sensory disturbance and less alopecia.

Management of the eye

The goal is to achieve complete eye closure, thereby providing corneal protection, with minimal ptosis in the open position. In principle, the upper eyelid needs to be lowered (unopposed action of levator palpebrae superioris) and the lower eyelid needs to be elevated. It is useful to separate the eye into static and dynamic options for the upper and lower lid.

Upper lid options

Lid loading was first described over 50 years ago and is well tolerated and highly efficacious. Gold and platinum have been the most widely used materials for implantation because of their high density and excellent side-effect profile.²⁰ The amount of weight required for complete closure is frequently too large, and a large weight is more likely to detach and erode through the

skin. Test weights attached to the upper eyelid with double-sided tape are used preoperatively to determine the lightest weight that will bring the eyelid within 2–4 mm of the lower lid. The most commonly selected weights are 1.0 g and 1.2 g, though standard sizes range from 0.6 to 1.8 g. For the best results, they should be placed at least 4 mm above the lid margin (to decrease visibility). Care must be taken to avoid damage to the levator insertion during the dissection for placement to avoid ptosis. Patients are advised that eyelid closure is a slow rather than a rapid blink and that they must practise holding the closure. Complications include a visible lump (capsule formation), ulceration and/or extrusion, migration of the weight and irritation of the cornea. More recently, a flexible platinum chain has been described as an alternative to rigid gold weights, with multiple links allowing flexibility to adapt to the shape of the globe. Platinum has a better biocompatibility profile and a higher density compared to gold and can thus be made into a smaller implant with improved cosmesis.

An alternative to lid loading is levator lengthening.²¹ The patient should be examined standing up and asked to close their eyes maximally; the width of sclera still exposed is the amount the levator needs to be lengthened and is typically approximately 5 mm. Through a standard blepharoplasty incision, the levator is exposed and separated from the tarsal plate and medial and lateral horns. A separate incision is made, hidden in behind the hairline, to harvest a deep temporal fascia graft. The size is measured and an additional 2 mm is harvested to allow for suture fixation to the levator and tarsal plate. The graft is inset and eye closure can be assessed once the patient is awake.

Lower lid options

There are fewer options to correct the lower eyelid in isolation as a consequence of the upper lid primarily controlling eye closure. In terms of static support for the lower lid, the use of autologous conchal cartilage grafts can be an effective procedure to improve the function and appearance of the atonic lower eyelid.

A fascial or palmaris longus tendon sling can be used to provide support the lower lid. The sling is fixed at the medial canthal ligament and supraorbital margin laterally. Proper placement of the tendon 1–2 mm below the lid margin is crucial as low placement will exacerbate an ectropion and high placement may result in an entropion.

Combined options

A tarsorrhaphy results in support of the lower eyelid and decreases the amount of corneal exposure by lowering the upper lid. McLaughlin's lateral tarsorrhaphy involves resection of the posterior lamellar of the upper lid (tarsal plate and conjunctiva) as well as an identical area of anterior lamellar of the lower lid (skin and muscle).²² The two denuded areas are overlapped and both tarsal plates are sutured. It is indicated in patients with concomitant senile ectropion but may result in the appearance of a smaller eye and difficulty when looking laterally. If this fails to achieve the desired result, then a fascial or palmaris longus

tendon sling is used as well. Canthoplasties are rarely performed as they tend to stretch over time in patients with facial paralysis.

The palpebral spring implant is a wire shaped by the surgeon in the form of an open safety pin. The lower arm is attached to the lid margin and the upper arm to the superior orbital rim. When the eye is opened by contraction of the levator muscle, the spring is compressed. When the levator is relaxed, the spring expands and forces the eyelid to a closed position. It has an advantage over upper lid weighting in that it will function equally well in the upright or supine position, but the results depend on the experience of the operator and the spring may loosen or fracture with time.²⁰

The transfer of a strip of temporalis muscle was first described by Gilles to provide a dynamic closure of the eyelid using autologous tissue.²³ A 1.5 cm strip of temporalis muscle is detached superiorly, turned anteriorly, and extended with two strips of tendon or fascia, passed through the upper and lower eyelids, and fastened to the medial canthal ligament. Alternatively, a static sling can be placed in the lower lid as previously described and a dynamic temporalis transfer applied to the upper lid as this eyelid requires more movement. The temporalis muscle transfer is activated by closing the jaw and therefore frequent biting throughout the day results in frequent corneal lubrication. It does result in a bulge over the lateral orbital margin and a slit-like appearance to the eye and is technically demanding to perform, but it can provide forceful and full eyelid closure, especially if the tension of the transferred muscle has been set correctly.

In order to restore the blink reflex, FFMT must be considered. This should ideally be performed as part of a two-stage reanimation process where two cross-facial nerve grafts are harvested, one being attached to a functioning buccal branch, the other to a zygomatic branch of the contralateral side. At the second stage, a free platysma flap may be raised with its neurovascular pedicle, tunnelled through the upper and lower lids and attached to the medial canthal ligament medially and temporal fascia laterally. The neurovascular pedicle is then typically joined to the cross-facial nerve graft (CFNG) from the functioning zygomatic branch and the superficial temporal vessels. This provides the patient with true spontaneous eye closure and restoration of the blink reflex.

A combination of lid loading, lateral tarsorrhaphies and lower eyelid slings are the most frequently carried out procedures on the eyelids in facial paralysis patients. In spite of this, treatment of epiphora is frequently unsatisfactory and may persist as a result of reduced inhibition on the lacrimal gland.

Management of the mid- and lower face

The surgical treatment of the mid- and lower face can be divided into static and dynamic options. In elderly patients, or in cases where dynamic options are unavailable, the mainstay of treatment is with autologous grafts or synthetic materials. The use of synthetic grafts offers the benefit of no donor site morbidity at

the risk of increased infection and extrusion of the material. Many examples of autologous grafts exist, ranging from palmaris longus to the Achilles tendon, however tensor fascia lata yields good to excellent results.²⁴ The graft can be easily harvested with two parallel incisions over the lateral thigh and provides sufficient graft length and breadth in comparison to others. The graft is split into three distal slips, one being inserted at the midline of the upper lip, one just beyond the midline in the lower lip and the last being inserted at the modiolus. Overtensioning of the graft is required in order to compensate for stretching over time. Due to the breadth of the graft, excellent support of the cheek is achieved which improves function and aesthetic appearance of the mid-face. An adjunct static procedure, much akin to a lateral canthopexy in the eye, is to close the angle of the mouth. Although this reduces the aperture of the mouth, it helps to lift the lower lip to improve symmetry at rest and improve symptoms.

Dynamic reanimation of the mid-face can be achieved with regional muscle transfers using the temporalis muscle, the masseter muscle or with a free microvascular (functional) muscle transfer.²⁵ A masseter muscle transfer results in a hollow at the angle of the mandible from where the muscle has been removed. In 1934, Gillies folded the temporal muscle over the zygoma to the lip subcutaneously, resulting in significant muscle bulging over the zygoma and a residual hollow in the temporal fossa. McLaughlin developed an intraoral approach to the coronoid process, removing part of the coronoid. The coronoid is then attached to fascia lata slings secured to the modiolus around orbicularis oris, allowing for a dynamic result using the whole of the temporalis.²⁶ Morrison's widely used modification is an extraoral approach to the temporalis tendon and placement of fascia lata grafts. In 1997, Labbé modified this technique further by carrying out an osteotomy of the zygomatic arch,²⁷ detachment at the coronoid process for insertion to the lips and temporalis muscle repositioning/lengthening by detaching the posterior third, resulting in a 'temporalis slide'. This procedure avoids contour problems and the routing provides good excursion but patients have to undergo intense physical therapy to relearn function. This is useful in patients for whom FFMTs are less effective and more unreliable. Using local muscle transfers requires the brain to retrain in order to achieve truly spontaneous results; with age, cortical plasticity diminishes and whether it is possible to achieve such a result remains to be seen.

Lower lip deformity is due to lack of innervation of the depressor labii inferioris by the marginal mandibular nerve. This is more evident during speech when the paralysed side remains elevated and the non-paralysed side moves inferiorly. Non-surgical management includes chemodenervation with Botox of the non-paralysed depressor labii inferioris to improve symmetry. Surgical options include selective myectomy of the non-paralysed depressor labii inferioris through the buccal surface of the lip, sectioning of the marginal branch of the contralateral facial nerve or transfer of the anterior belly of digastric muscle on the paralysed side. The anterior belly of



Figure 23.3 Unilateral facial reanimation. (a) Preoperative photograph showing a right sided facial paralysis. (b) Postoperative photograph after facial reanimation using a pectoralis minor free functional muscle transfer.

digastric is supplied by the mylohyoid branch of the mandibular division of the trigeminal nerve, and is spared in facial paralysis. The muscle can be divided at the tendinous junction separating the two bellies and then transferred over the chin (Conley's transfer).²⁸ It is secured to orbicularis oris at the lower lip and acts to recreate the downwards dynamic motion of the mouth and help symmetrize the position of the mouth, particularly during speech or eating. In more complex congenital facial hypoplastic conditions such as first arch syndrome, or following extensive surgery in the digastric triangle, where the anterior belly of digastric may be absent or damaged, an alternative option is a free EDB transfer or a mini-hypoglossal nerve transfer.

FFMT was popularized by Harii and continues to be the gold standard of treatment for facial paralysis. It allows for a true spontaneous smile to be restored. The use of FFMT in adults has evolved over the years and different treatment modalities are available depending on age groups. When considering muscle choice, the clinician must take into account muscle bulk, pedicle length and the size of the nerve. If a bulky muscle, such as latissimus dorsi, is used then adequate trimming of the muscle must be performed prior to inset. Pedicle length is vital if a one-stage procedure is to be considered.

For patients under the age of 20 years, a two-stage reconstruction is still advocated. Below this age, the ability of a nerve to regenerate along a CFNG is predictable and therefore favourable and the same two-stage procedure as described for the paediatric group is performed (Figure 23.3) utilizing gracilis or pectoralis minor for the FFMT.

In patients between 20 and 40 years, a one-stage procedure may be performed. In this case, a distal branch of the contralateral functioning buccal branch is used to neurotize the FFMT, and is approached via a small incision over the nasolabial fold. A muscle is chosen with a long pedicle length (typically latissimus dorsi) as the recipient nerve has to be tunnelled from one side of the face to the other and coapted

to the donor buccal branch directly. The advantage this has over a two-stage procedure is that the nerve only has to cross over one neurotomy in contrast to two in CFNGs and allows for a sufficient number of axons to reinnervate the muscle. This one-stage procedure also allows for a spontaneous symmetrical smile as it is powered by the contralateral facial nerve.

For patients between the ages of 40 and 60 years, similarly a one-stage procedure is advocated, however the donor nerve of choice is an ipsilateral branch of the masseteric nerve. The masseteric nerve is chosen due to the increased axonal counts, which are sufficient to power a FFMT and compensates for the increased age. In patients over 60 years, FFMT is not suggested as the results become increasingly unpredictable and fewer patients achieve good results.

In situations that require a planned segmental excision of the facial nerve, such as facial nerve involvement with a parotid carcinoma, a nerve graft may be used. A sural nerve can be coapted to the proximal and distal stumps of the divided nerve and a good recovery can be achieved. It is important to note that this is unaffected by radiotherapy treatment, which is a frequent treatment modality in this patient group.^{29,30}

Frequently a facial palsy patient may require multiple operations. This is in part due to the changes of an ageing face but also to improve cosmesis. Static slings may stretch over time and require tightening or an FFMT may require revision due to the patient not being happy with the bulkiness. Although 80% of FFMT obtain either a good or excellent result, 20% gain no benefit and warrant another surgical option.¹⁵

Conclusion

The essential primary strategy when approaching the management of facial paralysis is to assess the patient in detail and to form a tailored plan for each individual. In general, early referral

to a facial reanimation service is recommended, particularly in the paediatric group. A facial reanimation unit should be in a specialized centre which can cater for paediatric services and should also be able to deliver all the options available to a patient.

Currently, there is no standardized outcome measurement for facial palsy surgery, although the surgical community is working towards this. Photographic and video assessment by a specialized panel of judges appears to be the most widely used and transferable.¹⁵ More detailed methods of measuring vectors of movement in the face are incredibly time consuming and almost impossible to perform on small children.

The ultimate aim of facial reanimation is to address all the branches of the facial nerve. The goal is to restore facial symmetry, allow for spontaneous movement and preserve function; this will serve to protect the cornea and improve speech and oral continence, allowing for the normal expression of emotion.

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PART IV

Head and neck reconstruction

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CHAPTER 24

Head and neck malignancy: staging and neck dissections

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Introduction

The majority of malignant tumours of the head and neck region are squamous cell carcinomas (HNSCCs) arising from the mucosa of the upper aerodigestive tract. HNSCC is the fifth most common neoplasm worldwide, with greater than half a million new cases occurring per year globally.¹ The major tumour sites include the oral cavity, the nasopharynx, the oropharynx, the hypopharynx, the larynx, the nose and the paranasal sinuses. Tumours occur most commonly in the oral cavity, which collectively accounts for 60% of all oral cancer cases in the UK (2010).²

Oral cancer is the 15th most common cancer in the UK, accounting for 2% of new cases (6539 new cases in 2010) and its incidence has increased since the 1970s.² It is more common in males than females (incidence: 2 : 1, male : female), and with increasing age, with approximately 50% of cases occurring in patients aged between 45 and 64 years and 44% of cases occurring in those over 65 years of age.²

The increasing incidence of oral cancer in the UK is probably due to changes in the prevalence of major risk factors for oral cancer, such as heavy alcohol consumption, smoking and infection with the human papilloma virus (HPV).² Cumulative changes in DNA caused by the synergistic effects of cigarette smoking and alcohol are thought to underlie the majority of oral cancer cases. This may explain the variations in geographical incidence of oral cancer within the UK, with the highest incidence rates occurring in Scotland, Northern Ireland and the North of England.³

Assessment of the patient with head and neck cancer

The assessment of the patient with head and neck cancer involves a multidisciplinary team (MDT) approach, comprising history and clinical examination, radiological and histopathological evaluation. The clinical information is collated and

discussed at a head and neck MDT meeting to formulate a management plan. In our unit, the core MDT comprises a head and neck surgeon, a plastic and reconstructive surgeon, an oncologist, a radiologist, a pathologist, a clinical nurse specialist, a dietician, a prosthetist and a speech therapist. Important points considered in the decision-making process for treatment include: patient age, tumour factors (site and extent), comorbidity, social circumstances and the patient's wishes.

History

The history can provide useful information regarding the primary tumour, any secondary metastases, and the patient's general health/fitness for surgery. Age is an important consideration as elderly patients generally have more comorbidity, less functional reserve and may be more difficult to successfully rehabilitate for speech and swallowing than younger ones. We do not, however, have a cut-off age for microvascular transfer and reconstruction and would not exclude a patient based on age.

Tumour symptoms are frequently related to the anatomical location of the neoplasia. Rapidly progressive symptoms suggest aggressive disease in contrast to slow progression, which suggests less aggressive disease. The past medical history is important as comorbidities can increase the risks of surgery and of general anaesthesia and ultimately affect prognosis. Tumours developing in the immunocompromised generally have a poor prognosis. The social history is of particular importance in planning physical and psychological rehabilitation following treatment. Furthermore, the history should include an assessment of the risk factors for development of head and neck cancer such as alcohol and tobacco use.

Clinical examination

A general examination of cardiovascular, respiratory and abdominal systems to assess the patient's fitness for surgery and also to detect signs of metastasis is performed. This is combined with an examination of the primary tumour and of the cervical lymph node groups. The primary tumour is inspected with a good light source (e.g. headlight) and palpated to determine:

position, borders and functional effect. Palpation of the tongue may reveal tumour that is not obvious on inspection alone. Fibreoptic endoscopy is useful for the examination of nasal cavity, nasopharynx and the larynx. The piriform fossa can be exposed by asking the patient to exhale against a closed mouth, which causes the cheeks to blow outwards.⁴

The cervical lymph node groups are examined by standing behind the patient, with the patient's neck slightly flexed to relax the strap muscles. Palpation of all cervical lymph node groups is performed in a systematic manner to ascertain the position, size, number of enlarged nodes and fixation of lymph nodes to adjacent anatomical structures or skin. Fine needle aspiration cytology (FNAC) is the initial investigation of choice for patients with cervical lymphadenopathy. The diagnostic accuracy is very high and there are no reports of seeding of head and neck tumours following FNAC.⁴ The adequacy of FNAC is improved if performed under ultrasound guidance.

The clinical assessment should also include an assessment of nutritional status as many of these patients are prone to malnutrition due to difficulties with eating and swallowing caused by the tumour, and also because they are often heavy smokers and drinkers. Malnutrition can compromise wound healing and increase susceptibility to infection and preoperative feeding may be required to optimize these patients for surgery. In patients who are likely to require a long postoperative rehabilitation before swallowing, a radiologically inserted gastrostomy (RIG) tube is organized. Presurgical dental assessment is also vital in this group of patients. Many have high levels of dental neglect and periodontal disease and may need dental extractions at the time of ablative surgery, particularly if postoperative radiotherapy is likely. Some patients will also benefit from psychological assessment and counselling.

Haematological tests including full blood count, urea and electrolytes, liver function tests, clotting studies and a group and save are routinely performed. Many patients with head and neck cancer have synchronous tumours (e.g. carcinoma of the bronchus) and so chest X-ray or chest CT scan are routine before treatment. Other tests such as thyroid function tests, electrocardiograms, echocardiograms and respiratory function tests are performed when clinically indicated. Anaesthetic review is frequently sought to establish patient fitness for general anaesthesia.

Radiological examination

Radiological examination is a vital part of the assessment of the patient with head and neck cancer. It provides information about the site and extent of primary tumour, neck node involvement (number of nodes, size, position), distant metastases and synchronous primary tumours, which is ultimately used in staging. CT scans are fast and provide excellent bony detail but expose the patient to relatively large doses of ionizing radiation. CT scans are the staging investigation of choice in our unit. MRI can also be used but scan times are significantly longer than those for CT. MRI may therefore not be suitable for all patients (many patients with head and neck cancer have difficulty with

breathing, swallowing, lying flat and keeping still). Positron emission tomography (PET) has a role in evaluating the patient with an unknown primary and will detect the primary in approximately one third of cases.⁴ Other imaging modalities have specific indications: barium swallow is useful in assessment of some hypopharyngeal tumours and some tumours of the cervical oesophagus; an orthopantomogram can show tumour invasion of the mandible and dental disease, and may be useful in planning surgery for tumours of the oral cavity and oropharynx.

Endoscopy

The majority of patients with head and neck cancer undergo rigid endoscopy under general anaesthesia to establish the position and extent of the primary tumour and to obtain tissue biopsies. Radiological imaging should be performed before biopsy in order to avoid the effect of upstaging from oedema caused by biopsy. Biopsies should be taken from the viable part of the tumour (i.e. not from the centre (may be necrotic), and not from the edge (may only show dysplasia)).

Panendoscopy or triple endoscopy (laryngoscopy combined with oesophagoscopy and bronchoscopy) is performed to exclude a synchronous primary tumour in the upper aerodigestive tract although its routine use is controversial.⁴ Arguments for routine panendoscopy include: it is quick, has low morbidity and is easily performed at the time of endoscopy for the primary site tumour. Arguments against routine panendoscopy include: it creates extra risks and expense, and detection rates are similar to those with symptom-directed investigations combined with routine chest radiography. Panendoscopy is therefore only recommended for symptomatic patients, patients with primary tumours at high risk of a synchronous primary tumour (e.g. oropharynx), and patients who have suspicious abnormalities on radiological studies. Panendoscopy also has a role in the investigation of the patient with an unknown primary tumour where high-risk sites (ipsilateral tonsil, nasopharynx, ipsilateral tongue base, piriform fossa) are biopsied.⁴

Staging

The tumour, node, metastasis (TNM) staging system developed by the American Joint Committee on Cancer (AJCC) in 2002 is the most widely used system to establish disease stage and prognosis, and also to direct management. The clinical information used in TNM staging comes from the physical examination, radiographic, intraoperative and pathologic findings. The TNM system is composed of three categories: T – the characteristics of the primary tumour (may be based on size, location or both); N – the degree of regional lymph node involvement; and M – the absence or presence of distant metastases. The given TNM status translates to the disease stage (I–IV). Early-stage disease is denoted as stage I or II disease, and advanced-stage disease as stage III or IV disease. Of note, any metastatic neck disease will classify the disease as advanced (except in selected nasopharynx or thyroid tumours).^{5,6}

Although the definitions for Tx, T0 and Tis are the same for all primary tumours of the head and neck region, the definitions for T1–4 vary according to anatomical site of primary tumour (see Tables 24.2, 24.3, 24.7, 24.9 and 24.10). Tx indicates that the primary tumour cannot be assessed; T0 indicates that there is no evidence of primary tumour and Tis is defined as carcinoma *in situ*.

Anatomical sites

The upper aerodigestive tract (UADT) begins where the skin meets the mucosa at the nasal vestibule and the vermilion borders of the lips and continues to the junction of the cricoid cartilage where the hypopharynx meets the cervical oesophagus. This chapter focuses on sites with the greatest incidence of pathology and on subsites with specific reconstructive challenges. Tumours of the thyroid gland and salivary glands are beyond the scope of this chapter but interested readers are referred to other comprehensive texts for detailed discussion.⁷

Table 24.1 Primary tumour site locations of the oral cavity

Lip	Skin – vermilion junction to oral mucosa
Buccal mucosa	Mucosal lining extending from pterygomandibular raphe anteriorly
Alveolar ridge	Gingival mucosa anterior to retromolar trigone
Retromolar trigone	Mucosa behind the last mandibular molar tooth extending superiorly to the maxillary tuberosity; overlies the anterior aspect of the ascending ramus of the mandible
Hard palate	Bounded by the superior alveolar ridge and the junction of the soft palate
Floor of mouth	Bounded by the inferior alveolar ridges and the tongue
Anterior tongue (2/3)	Extends anteriorly from the circumvallate papillae and bounded by floor of mouth

Source: Saadeh and Delacure, 2007.⁸ Reproduced with permission of Lippincott, Williams and Wilkins.

Oral cavity^{5,8}

The anterior border is the junction of the skin and vermilion border of the lip. The posterior border is formed by the junction of the hard and soft palates superiorly, the circumvallate papillae inferiorly, and the anterior tonsillar pillars laterally. Primary tumour site locations within the oral cavity are summarized in Table 24.1. T-staging for the oral cavity is summarized in Table 24.2.

Nasopharynx^{5,8}

The nasopharynx includes the vault, the lateral walls, the posterior walls, and the superior surface of the soft palate. Table 24.3 summarizes the T-staging for tumours of the nasopharynx.

Oropharynx^{5,8}

The oropharynx begins where the oral cavity ends at the junction of the hard and soft palates superiorly and the circumvallate papillae inferiorly. It extends from the level of the soft palate superiorly (which separates it from the nasopharynx) to the level of the hyoid inferiorly (where the hypopharynx begins). Table 24.4 defines primary tumour site locations within the oropharynx. Table 24.2 summarizes the T-staging for the oropharynx.

Hypopharynx and cervical oesophagus^{5,8}

The hypopharynx extends from the hyoid bone superiorly, where it is contiguous with the oropharynx, and extends inferiorly to the cricopharyngeus muscle, where it meets the cervical oesophagus. Table 24.5 defines the primary tumour site locations of the hypopharynx and cervical oesophagus. Table 24.2 summarizes the T-staging for tumours of the oropharynx.

Larynx^{5,8}

The larynx is bordered by the oropharynx superiorly, the trachea inferiorly, and the hypopharynx laterally and posteriorly. The larynx is subdivided by the vocal cords into the supraglottic,

Table 24.2 T-staging for primary tumours of oral cavity, oropharynx and hypopharynx^{5,6}

	Oral cavity	Oropharynx	Hypopharynx
T1	<2 cm	<2 cm	One subsite and is <2 cm
T2	2–4 cm	2–4 cm	More than one subsite or 2–4 cm with NO fixation of hemilarynx
T3	>4 cm	>4 cm	>4 cm or fixation of hemilarynx
T4a	Lip – Tumour invades through cortical bone, inferior alveolar nerve, floor of mouth, or skin of face (i.e. chin or nose)	Invades the larynx, deep/extrinsic muscle of the tongue, medial pterygoid, hard palate, or mandible	Invades thyroid/cricoid/hyoid/oesophagus/central compartment soft tissues
Moderately advanced local disease	Tongue – Extrinsic muscles, fixed tongue or mandible invaded		
T4b	Invades masticator space, pterygoid plates or skull base or encases the internal carotid artery	Tumour invades the lateral pterygoid muscle, pterygoid plates, lateral nasopharynx, or skull base or encases the carotid artery	Invades prevertebral fascia, mediastinum or encases carotid artery
Very advanced local disease	[Note: Superficial erosion alone of bone/tooth socket by gingival primary is not sufficient to classify a tumour as T4]		

Source: Shah, 2003.⁷ Reproduced with permission of Elsevier.

Table 24.3 T-staging for tumours of the nasopharynx^{5,6}

T1	Tumour confined to the nasopharynx, or extends to the oropharynx and/or nasal cavity, without parapharyngeal extension
T2	Tumour extends into the parapharyngeal space
T3	Tumour involves bony structures and/or paranasal sinuses
T4	Tumour with intracranial extension and/or involvement of cranial nerves, infratemporal fossa, hypopharynx, orbit, or masticator space

Table 24.4 Primary site locations within the oropharynx

Soft palate	Extends from the hard palate junction to the uvula posteriorly and anterior tonsillar pillars laterally
Tonsil	Bounded by the tonsillar pillars
Lateral pharyngeal wall	Extends from posterior tonsillar pillar to posterior pharyngeal wall
Posterior pharyngeal wall	Bounded by the lateral pharyngeal walls and extending from the level of the hard palate superiorly and hyoid bone inferiorly
Posterior tongue (1/3; base of tongue)	From circumvallate papillae to epiglottis

Source: Shah, 2003.⁷ Reproduced with permission of Elsevier.

Table 24.5 Primary tumour site locations within the hypopharynx and cervical oesophagus

Postcricoid region	Extends from the arytenoids superiorly to the cricoids cartilage inferiorly
Posterior pharyngeal wall	Anterior to retropharyngeal space and extends from the level of the epiglottis to the level of the cricoids cartilage
Piriform sinus	Pyramidal in shape with base superiorly and apex inferiorly. Extends from the oropharynx superiorly to the laryngeal ventricles inferiorly. Medial border is the lateral cricoids cartilage. Lateral border is the medial thyroid cartilage. The posterior border is bounded by both the lateral and posterior pharyngeal walls
Cervical oesophagus	Extends from the cricoids level to the sternal notch

Source: Saadeh and Delacure, 2007.⁸ Reproduced with permission of Lippincott, Williams and Wilkins.

glottic and subglottic subsites. Table 24.6 defines the primary tumour site locations of the larynx. Table 24.7 summarizes the T-staging for tumours of the larynx.

Nasal cavity and paranasal sinuses

Primary tumour sites within the nasal cavity and paranasal sinuses are summarized in Table 24.8. Table 24.9 summarizes T-staging for tumours of the maxillary sinus. Table 24.10 summarizes T-staging for tumours of the nasal cavity and ethmoid sinus.

Table 24.6 Primary tumour site locations within the larynx

Supraglottis	Epiglottis, arytenoids, aryepiglottic folds, and false cords
Glottis	True vocal cords, the anterior and posterior commissures
Subglottis	Below the glottis extending to the inferior margin of the cricoid cartilage

Source: Saadeh and Delacure, 2007.⁸ Reproduced with permission of Lippincott, Williams and Wilkins.

Table 24.7 T-staging for the supraglottis, glottis and subglottis respectively^{5,6}

	Supraglottis	Glottis	Subglottis
T1	One subsite of the supraglottis, normal vocal cord movement	T1a one vocal cord involved, with normal mobility T1b both cords involved with normal mobility	Limited to subglottis
T2	Invades mucosa of other subsite of supraglottis but no laryngeal fixation	Invades into sub or supraglottis or impaired mobility of the cords	Extends to vocal cords
T3	Limited to larynx with vocal cord fixation or invasion of post cricoid area, periglottic spaces or thyroid cartilage	Limited to larynx with cord fixation or invasion of paraglottic space or inner cortex thyroid cartilage	Limited to larynx with vocal cord fixation
T4a	Invades through thyroid cartilage or beyond larynx	Invades through thyroid cartilage or beyond larynx	Invades cricoid or thyroid cartilage or beyond larynx
T4b	Invades prevertebral space or mediastinum or encases carotid artery	Invades prevertebral space or mediastinum or encases carotid artery	Invades prevertebral space or mediastinum or encases carotid artery

T4a, moderately advanced local disease; T4b, very advanced local disease.

Neck staging under the TNM staging system for head and neck tumours^{5,6,8}

Table 24.11 summarizes the neck staging system for primary tumours of the head and neck region (excluding nasopharynx). Table 24.12 summarizes the neck staging system for primary tumours of the nasopharynx.

Distant metastasis under the TNM staging system for head and neck tumours^{5,6,8}

Table 24.13 summarizes distant metastasis under the TNM staging system.

TNM stage grouping

Tables 24.14 and 24.15 summarize the TNM stage groupings for tumours of the head and neck region.

Table 24.8 Primary tumour site locations within the nasal cavity and paranasal sinuses⁵

Nasal cavity	Extends superiorly from the walls of the ethmoid sinus anteriorly and the sphenoid sinus posteriorly down to the hard palate anteriorly and nasopharynx posteriorly. Lateral margins are the medial walls of the maxillary sinus and the nasal cavity is bisected sagittally by the septum
Maxillary sinus	Bounded superiorly by the orbital floor, inferiorly by the hard palate, posteriorly by the pterygoid plates and pterygopalatine fossa, and laterally by the pterygoid muscles and mandibular ramus
Frontal sinus	Above and along the anterior aspect of the ethmoid sinus
Ethmoid sinus	Between medial orbits, superior to the nasal cavity
Sphenoid sinus	Skull base, posterior to the ethmoid sinus

Source: Saadeh and Delacure, 2007.⁸ Reproduced with permission of Lippincott, Williams and Wilkins.

Table 24.9 T-staging for primary tumours of the maxillary sinus^{5,6}

T1	Tumour is limited to the maxillary sinus mucosa, with no erosion or destruction of bone
T2	Tumour is causing bone erosion or destruction, including extension into the hard palate and/or middle nasal meatus, except extension to the posterior wall of the maxillary sinus and pterygoid plates
T3	Tumour invades any of the following: bone of the posterior wall of the maxillary sinus, subcutaneous tissues, floor, or medial wall of the orbit, pterygoid fossa, or ethmoid sinuses
T4a	Tumour invades anterior orbital contents, skin of cheek, pterygoid plates, infratemporal fossa, cribriform plate, sphenoid or frontal sinuses
T4b	Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve (V2), nasopharynx, or clivus

T4a, moderately advanced local disease; T4b, very advanced local disease.

Table 24.10 T-staging for primary tumours of the nasal cavity and ethmoid sinus⁵

T1	Tumour is confined to the ethmoid sinus with or without bone erosion
T2	Tumour invades two subsites in a single region or extends to involve an adjacent region within the nasoethmoidal complex, with or without bony invasion
T3	Tumour extends to invade the medial wall or floor of the orbit, maxillary sinus, palate, or cribriform plate
T4a	Tumour invades any of the following: anterior orbital contents, skin of nose or cheek, minimal extension to anterior cranial fossa, pterygoid plates, sphenoid or frontal sinuses
T4b	Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than (V2), nasopharynx or clivus

T4a, moderately advanced local disease; T4b, very advanced local disease.

Table 24.11 Neck staging for primary tumours of the head and neck region (excluding nasopharynx)^{5,6}

Nx	Regional lymph nodes cannot be assessed
N0	There is no regional nodes metastasis
N1	Single ipsilateral lymph node metastasis ≤ 3 cm in greatest dimension
N2a	Single ipsilateral lymph node metastasis ≥ 3 cm but < 6 cm in greatest dimension
N2b	Multiple ipsilateral lymph node metastases, all < 6 cm in greatest dimension
N2c	Bilateral or contralateral lymph node metastases, all < 6 cm in greatest dimension
N3	Lymph node metastasis > 6 cm in greatest dimension

U, L, A designation of 'U' or 'L' may be given in addition to indicate the level of metastasis above the lower border of the cricoid cartilage (U) or below the lower border of the cricoid cartilage (L).

Table 24.12 Neck staging for primary tumours of the nasopharynx^{5,6}

Nx	Regional lymph nodes cannot be assessed
N0	There is no regional lymph node metastasis
N1	Unilateral lymph node metastasis(es), ≤ 6 cm in greatest dimension, above the supraclavicular fossa
N2	Bilateral lymph node metastasis(es), ≤ 6 cm in greatest dimension, above the supraclavicular fossa
N3	Lymph node metastasis/es is ≥ 6 cm and/or to the supraclavicular fossa
N3a	Tumour is ≥ 6 cm in dimension
N3b	Tumour extends to the supraclavicular fossa

Table 24.13 Distant metastasis under the TNM staging system^{5,6}

M0	No distant metastasis
M1	Distant metastasis

Neck dissection

The lymph node levels of the neck

The Memorial Sloan Kettering level system, which divides the lymph nodes of the neck into six distinct groups (Figure 24.1) is the widely accepted system for describing the location of lymph node disease in the neck. Levels I, II and V are subdivided into sublevels (Figure 24.2).⁹ Table 24.16 summarizes the six lymph node groups and six sublevels. Table 24.17 summarizes the anatomical boundaries of the levels and sublevels of lymph nodes in the neck.

Neck dissection

'Neck dissection' is the general term used to describe the surgical removal of the fibrofatty contents of the neck, for the treatment or assessment of the cervical lymph nodes. Neck dissections are classified as comprehensive (radical, modified radical, extended radical) or selective, based on the nodal levels dissected and the structures preserved (Table 24.18). Radical neck dissection (RND) was historically the standard procedure for the management of both clinically positive and occult metastases in the cervical lymph

Table 24.14 Stage grouping for primary tumours of the oral cavity, oropharynx, larynx, and hypopharynx^{5,6}

	T	N	M
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T0, T1, T2 or T3	N1	M0
Stage Iva	T4a	N0 or N1	M0
	T1, T2 or T3	N2	M0
Stage Ivb	T4b	Any N	M0
	Any T	N3	M0
Stage Ivc	Any T	Any N	M1

Stage IVa, moderately advanced local/regional disease; stage IVb, very advanced local/regional disease; stage IVc, distant metastatic disease.

Table 24.15 Stage grouping for primary tumours of the nasopharynx^{5,6}

	T	N	M
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T1	N1	M0
	T2	N0	M0
	T2	N1	M0
Stage III	T1	N2	M0
	T2	N2	M0
	T3	N0	M0
	T3	N1	M0
	T3	N2	M0
Stage IVA	T4	N0	M0
	T4	N1	M0
	T4	N2	M0
Stage IVB	Any T	N3	M0
Stage IVC	Any T	Any N	M1

nodes. However, over the last two decades there has been a shift towards more conservative surgical procedures as neck dissection with conservation of non-lymphatic structures gives equivalent oncologic outcomes with improved functional results.^{10,11}

Radical neck dissection

RND was first described by Crile in 1906 and involves unilateral removal of lymph node levels I–V, the spinal accessory nerve (SAN), the internal jugular vein (IJV) and the sternocleidomas-toid muscle (SCM). RND is now reserved for patients with advanced neck disease, recurrent disease after chemoradiation or gross extracapsular spread to the SAN, IJV and SCM.

Modified radical neck dissection

Modified radical neck dissection (MRND) comprises removal of lymph node levels I–V with preservation of one or more of the non-lymphatic structures which are normally resected in RND (i.e. the SAN, IJV and SCM). The structure or structures preserved should be indicated in the name of the procedure (e.g. MRND

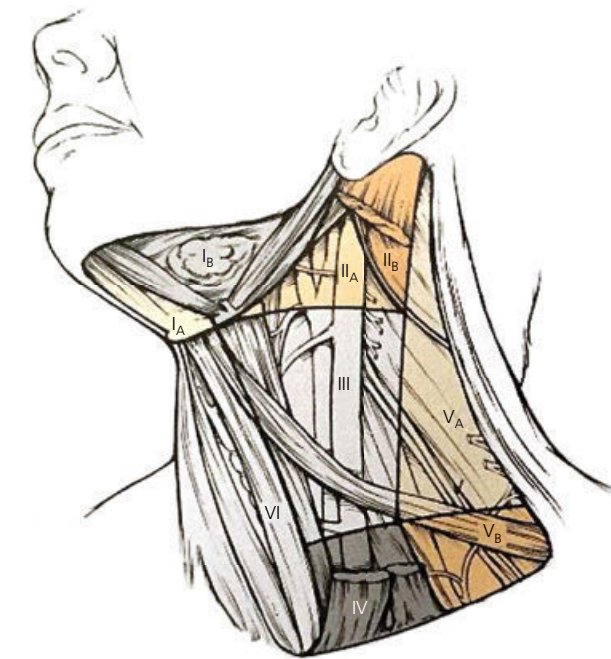


Figure 24.1 The six levels of the neck. Source: Cummings *Otolaryngology-Head and Neck Surgery*, 5e, Paul Flint et al., 2010. Reproduced with permission of Elsevier.

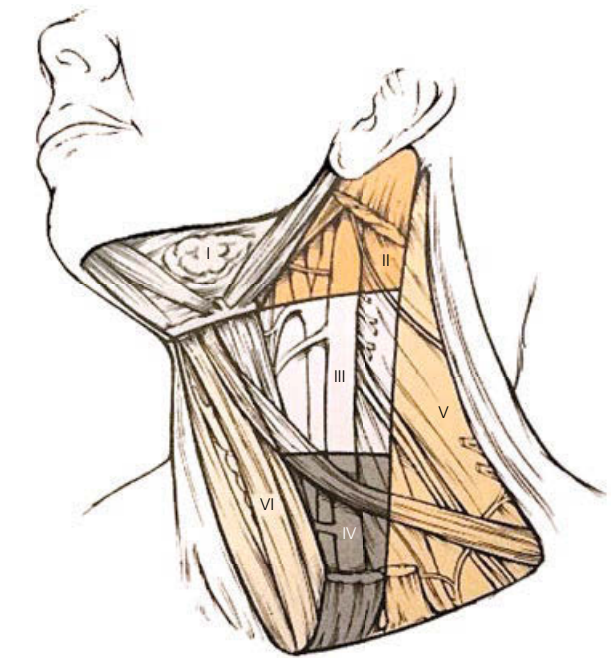


Figure 24.2 The six sublevels of the neck. Source: Cummings *Otolaryngology-Head and Neck Surgery*, 5e, Paul Flint et al., 2010. Reproduced with permission of Elsevier.

with preservation of accessory nerve and IJV), however the following nomenclature has gained widespread acceptance;

- MRND I – Preservation of SAN
- MRND II – Preservation of SAN, SCM
- MRND III – Preservation of SAN, SCM and IJV

Extended neck dissection

Extended neck dissection (END) is the combination of RND with removal of additional structures such as additional lymph node groups or non-lymphatic structures (e.g. carotid artery, cranial nerves, muscles or the parotid gland) that are not routinely removed in RND.

Selective neck dissection

Selective neck dissection (SND) describes a neck dissection where there is preservation of one or more lymph node levels that are routinely removed in RND (Table 24.16). SND has been developed based on the observation that cervical metastases in previously untreated patients proceed in a predictable fashion depending on the site of the primary tumour.¹² Hence, the lymph node groups most likely at risk of involvement can be removed while those at low risk of involvement may be safely preserved.^{10,11} For oral cavity cancers, the lymph nodes at greatest risk are located in levels I–III. The lymph nodes at greatest risk for oropharyngeal, hypopharyngeal and laryngeal cancers are located in levels II–IV, and for thyroid cancer are in level VI.

Indications for neck dissection

As previously mentioned, there is a trend away from performing RND towards MRND/SND. MRND is indicated for clinically palpable metastatic neck disease but conversion to RND sometimes becomes necessary upon gross involvement of the SAN, IJV, SCM. RND may also be indicated in recurrent disease. Bilateral MRND is indicated when there is contralateral lymph node involvement.

SND is generally performed in patients who have N0 disease but who have a high probability of occult disease and in all cases that require microsurgical reconstruction to gain access to the vessels. SND may also sometimes be performed for N1 disease in levels I or II. SND (I–III) is performed for oral cavity cancer. Some authorities recommend SND (I–IV) for tongue cancer as level IV is also at risk.⁹ Bilateral SND (I–III) is indicated in patients who have tumours of the floor of mouth/anterior tongue where lymph nodes on both sides of the neck are at risk. SND (II–IV) is performed for oropharyngeal, hypopharyngeal and laryngeal cancers while SND (VI) is performed for thyroid cancer. Posterolateral neck dissection (= SND II–V + postauricular and suboccipital nodes) is performed most commonly to remove nodal disease from cutaneous malignancies of the posterior scalp and neck. END is performed in patients with

Table 24.16 The lymph node levels and sublevels of the neck⁹

Level (nodal group)	Anatomy	Likely primary site
Level I	Submental (sublevel 1A): nodal tissue superior to the hyoid bone and between the anterior bellies of the digastric muscles. Submandibular (sublevel 1B): nodal tissue between the mandibular border, the anterior bellies of the digastric muscles and the stylohyoid muscle, includes the submandibular gland	Floor of mouth, anterior oral tongue, anterior mandibular alveolar ridge, lower lip Submandibular gland, oral cavity, anterior nasal cavity, midfacial soft tissues
Level II Upper jugular	Nodal tissue along the upper third of the internal jugular vein extending from the skull base to the inferior border of the hyoid bone. Anterior border is lateral border of stylohyoid muscle, posterior border is posterior border of sternocleidomastoid muscle Sublevel IIA: tissue anterior to the spinal accessory nerve Sublevel IIB: tissue posterior to the spinal accessory nerve	Oral/nasal cavity, nasopharynx, oropharynx, hypopharynx, larynx, parotid gland
Level III Middle jugular	Nodal tissue along the middle third of the internal jugular vein extending from the inferior border of the hyoid bone to the inferior border of the cricoid cartilage. Anterior border is lateral border of sternohyoid muscle, posterior border is posterior border of sternocleidomastoid muscle	Oral cavity, nasopharynx, oropharynx, hypopharynx, larynx
Level IV Lower jugular	Nodal tissue along the lower third of the internal jugular vein extending from the inferior border of the cricoid cartilage to the clavicle. Anterior border is lateral border of sternohyoid muscle, posterior border is posterior border of sternocleidomastoid muscle	Hypopharynx, cervical oesophagus, larynx, thyroid gland
Level V Posterior triangle	Nodal tissue along the lower half of the spinal accessory nerve and the transverse cervical artery. Triangle is defined by the posterior border of the sternocleidomastoid muscle, the anterior border of the trapezius muscle, and the clavicle. Also includes the supraclavicular lymph nodes Sublevel VA: tissue above the horizontal plane marking the inferior border of the anterior cricoid arch Sublevel VB: tissue below the horizontal plane marking the inferior border of the anterior cricoid arch	Oropharynx, nasopharynx, posterior scalp and neck
Level VI Anterior compartment	Nodal tissues delimited by the hyoid bone superiorly, sternal notch inferiorly, and common carotid arteries laterally. Includes the pretracheal and paratracheal nodes, precricoid (Delphian node), and the perithyroidal nodes	Thyroid, glottic/subglottic larynx, piriform sinus apex, cervical oesophagus

Table 24.17 Anatomical structures defining the levels and sublevels of the neck⁹

Level (nodal group)	Boundary
Level IA	Superior: symphysis of mandible Inferior: body of hyoid Anterior (medial): anterior belly of contralateral digastric muscle Posterior (lateral): anterior belly of ipsilateral digastric muscle
Level IB	Superior: body of mandible Inferior: posterior belly of digastric muscle Anterior (medial): anterior belly of digastric muscle Posterior (lateral): stylohyoid muscle
Level IIA	Superior: skull base Inferior: horizontal plane defined by the inferior body of the hyoid bone Anterior (medial): stylohyoid muscle Posterior (lateral): vertical plane defined by the spinal accessory nerve
Level IIB	Superior: skull base Inferior: horizontal plane defined by the inferior body of the hyoid bone Anterior (medial): vertical plane defined by the spinal accessory nerve Posterior (lateral): lateral border of sternocleidomastoid muscle
Level III	Superior: horizontal plane defined by inferior body of hyoid Inferior: horizontal plane defined by inferior border of cricoid cartilage Anterior (medial): lateral border of sternohyoid muscle Posterior (lateral): lateral border of the sternocleidomastoid or sensory branches of cervical plexus
Level IV	Superior: horizontal plane defined by inferior border of cricoid cartilage Inferior: clavicle Anterior (medial): lateral border of sternohyoid muscle Posterior (lateral): lateral border of the sternocleidomastoid or sensory branches of cervical plexus
Level VA	Superior: apex of the convergence of the sternocleidomastoid and trapezius muscles Inferior: horizontal plane defined by the lower border of the cricoid cartilage Anterior (medial): posterior border of the sternocleidomastoid muscle or sensory branches of cervical plexus Posterior (lateral): anterior border of the trapezius muscle
Level VB	Superior: horizontal plane defined by the lower border of the cricoid cartilage Inferior: clavicle Anterior (medial): posterior border of the sternocleidomastoid muscle or sensory branches of cervical plexus Posterior (lateral): anterior border of the trapezius muscle
Level VI	Superior: hyoid bone Inferior: suprasternal Anterior (medial): common carotid artery Posterior (lateral): common carotid artery

Table 24.18 Types of neck dissection

Type	Levels dissected	Preserved structures	Primary tumour
RND	I–V	None	
SND (I–III)	I–III	XI+STM+IJV	Oral cavity
SND (II–IV)	II–IV	XI+STM+IJV	Oro/hypopharynx, larynx
SND (I–IV)	I–IV	XI+STM+IJV	Tongue
SND (VI)	VI	XI+STM+IJV	Thyroid
SND (II–V + occipital and postauricular nodes) (Posterolateral)	II–V + occipital and postauricular nodes	XI+STM+IJV	Posterior scalp and neck

N+ necks who would otherwise be candidates for RND/MRND when there is evidence of invasion into other surrounding structures (e.g. carotid artery).

Complications of neck dissection

The complications of neck dissection can broadly be classified into: intraoperative complications (e.g. bleeding, air embolus,

pneumothorax, nerve injury); complications in the early postoperative phase (e.g. skin flap necrosis, carotid artery blowout, chyle leak); and those that occur in the late postoperative phase (e.g. scar contracture, neuroma, shoulder pain syndrome, cellulitis and facial/cerebral oedema).

Chyle leak is rare and typically presents with drainage of milky fluid, although the fluid may be clear if the patient is

fasting or on a fat-free diet. The medical management of chyle leak includes leaving drains on continuous suction, application of a pressure dressing and bed rest. Dietician input is important and an elemental feed supplemented with medium-chain triglycerides may be given enterally. Total parenteral nutrition is used in refractory cases. Surgical intervention has been advocated for greater than 500 mL drainage per day persisting after one week of medical treatment, persistent low-output drainage occurring over a prolonged period, and if complications occur.¹³

Synchronous bilateral radical neck dissections may result in the development of facial oedema, cerebral oedema, or both. The oedema, which is more common in patients who have had previous radiation to the head and neck, is thought to be due to inadequate venous drainage and usually improves as collateral circulation develops. Preserving at least one external jugular vein in patients undergoing bilateral radical neck dissection can reduce postoperative facial oedema.

Indications for radiotherapy

External beam radiotherapy alone or in conjunction with chemotherapy has a well-established role in the treatment of head and neck cancer as definitive therapy or as an adjuvant to primary surgical treatment.⁸ Radiotherapy is also used in metastatic disease to relieve symptoms and to achieve local control. Adjuvant radiotherapy should ideally begin within 4–6 weeks following primary surgical resection and neck dissection, unless postoperative complications significantly delay wound healing. Delaying adjuvant therapy has been shown to significantly reduce locoregional control. Of recent note, inhibition of the epidermal growth factor receptor (EGFR) with the monoclonal antibody cetuximab has been shown to improve both disease-free survival and overall survival when combined with radiotherapy for stage 3/4 disease.¹⁴

In the presence of limited disease, the factors that influence the selection of treatment modality (surgery versus external beam radiotherapy versus brachytherapy) include primary tumour site, age, comorbidity, occupation, preference and quality of life following treatment. Surgery is the treatment of choice for early-stage oral cavity and paranasal sinus tumours. Surgery is preferred to radiotherapy in the oral cavity due to the risk of osteoradionecrosis from radiotherapy and because oral cavity SCCs may be less sensitive to radiotherapy than oropharyngeal or laryngeal cancers. Positive surgical margins, multiple involved lymph nodes, and/or extracapsular tumour spread may indicate postoperative radiotherapy ($\pm$ chemotherapy) to reduce local recurrence.⁸ In oropharyngeal, hypopharyngeal and laryngeal cancers, primary radiotherapy is generally preferred, as surgery is associated with greater levels of functional impairment and reduced quality of life.¹⁵ The treatment of laryngeal cancers varies widely, and for early-stage lesions, radiotherapy or transoral endoscopic excision are the most common treatment

options.⁸ The treatment of more advanced laryngeal tumours is controversial. Although total laryngectomy has historically been considered the gold standard for treating advanced laryngeal cancers, chemoradiotherapy has been shown to be quite effective in achieving local regional control, survival and voice preservation.⁸

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CHAPTER 25

Oral tongue reconstruction

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The oral tongue: anatomic defect and functional deficit-guided reconstruction

The oral tongue, which occupies most of the oral cavity, has diverse and important functions, and is also a common site for intraoral malignancy.^{1,2} Most oral tongue cancers require surgical treatment, with reconstruction according to underlying principles. Given that resections and reconstructions of the oral tongue can have wide-ranging functional consequences, it is best that a team approach with the ablative surgeon, the reconstructive surgeon and early input from the speech and language and swallowing therapists be developed.³ Because functional restoration is the main goal of reconstruction, in this chapter we present the principles that guide the selection of a reconstructive option to achieve this objective.

Normal and postresection altered anatomy and physiology

The anatomy and physiology of the oral cavity are complex. This overview concentrates on the oral tongue anatomy and physiology relevant to speech and swallowing, both in the normal person and in the patient after oral (mobile) tongue cancer resection and reconstruction.

The motor nerve to the tongue (hypoglossal, XII) is under voluntary control. It gives branches to the extrinsic tongue muscles and then ramifies to supply the intrinsic tongue muscles, supplying each side separately. The intrinsic tongue muscles (superior longitudinal, transverse, and inferior longitudinal and vertical), which interdigitate with the hyoglossus muscle, provide the oral tongue mobility, the change in shape, tissue volume and the intraoral position of the tongue, all so necessary for the coordinated oral preparatory and then transport phases of swallowing.⁴⁻⁷ The intrinsic muscles are also essential for speech. For instance, for the vowel sounds, the vertical height of the tongue and the shape of its dorsal and lateral aspects must be varied for

each vowel. For the consonants, the anterior/tip of tongue is required for the anterior 'stops' (e.g. 't', 'd', where the tongue must touch the anterior teeth or hard palate), the posterior oral tongue is required in tongue-palate contact for the posterior 'stops' (e.g. 'k', 'g'), the entire oral tongue is needed to change the airflow to a funnel as in 'laterals' (e.g. 'l') or to a constricted pattern as in 'fricatives' (e.g. 's', 'sh', 'th').⁸ All of these motor activities require a high degree of fine motor coordination, and depend on intact sensation to function normally.

The extrinsic muscles with the genioglossus, and chondroglossus attaching anteriorly and working with the geniohyoid, mylohyoid muscle and anterior digastric muscles, and the styloglossus attaching posteriorly and working with the palatoglossus and posterior digastric muscle,⁹ function to give a stable but moveable foundation for the tongue to work against in the oral preparatory and transport phases of swallowing. Many of these same muscles work to elevate the laryngo-hyoid complex in the pharyngeal phase of swallowing.

Thus a deep resection of the oral tongue can affect both oral stages of swallowing as well as the pharyngeal phase.

The main requirements of oral tongue function for speech and swallowing are:

- motility of appropriate portions for the task required;
- adequate tissue bulk plus ability to selectively alter that volume;
- positioning of the tongue.

Because different regions of the oral tongue predominate in different tongue actions, with the anterior, middle and posterior thirds variably involved, we consider the regions separately in the resections and reconstructions to follow, realizing, however, that many resections, although centred on one region, usually involve more than one region on the affected side, and may extend to the opposite side. Defects which extend to several regions take on the functional deficits of those regions also. In the simplest case, a functional deficit of a tip defect may affect the anterior 'stops' in speech, may limit the ability to move food within the mouth in the oral preparatory stage in swallowing

and, if tethered anteriorly, will impair the oral transport stage, compounding that with a tendency to drool. Each region of the oral tongue can be similarly profiled.

Classification systems of anatomic defects

No widely accepted classification system of defects has been developed. The proposals vary considerably in their categories and detail.^{10–15} The difficulties relate to the complexity of the oral cavity and its functions, the three-dimensional nature of these resections, with deeper resections, particularly those involving deep muscles and nerves, giving very different functional changes for the same surface area, and the ability of individual patients to adapt differently to what appears to be the same defect. Generally the extent of surface resected broadly correlates with the deficit of function.¹⁶ A suitable classification system must find the balance between complexity and simplicity so that only essential information is collected, must be easy to use, must be reproducible, must be reportable in a meaningful easily understood way, and must achieve the following objectives:

- accurately define the surface resected of the tongue, floor of mouth, alveolar process and mandible, buccal region and oropharynx;
- accurately define the deeper structures resected as to bone, muscle, nerve(s); and
- accurately determine the tissue volume resected.

A classification system is not an end in itself. For applicability, it must be linked to the reconstructive option(s) and procedural manoeuvres used, and then scored as to specific outcomes, for instance in speech and swallowing. Until such a coherent system is developed, only partial and approximate answers can be given to: What is the precise defect? What are the functional problems from it? What reconstruction manoeuvres and tissue give the optimal result? How does this 'optimal' result remain deficient? What deficiencies attend alternative reconstruction choices? How are the outcomes to be measured in a standardized fashion^{10,17} and ranked? The answers to these questions will only be found in well-designed multicentre studies.¹⁷

Goals of surgical reconstruction

The overall goal is to preserve and/or restore optimum speech, dental hygiene capability and eating functions, including those of the oral preparatory (mastication and bolus formation) and oral transport stages of swallowing.¹⁸ Often, the defect in a specific case can only be estimated preoperatively. The ideal assessment for a particular patient occurs after the resection, when the surgeon can estimate the functional deficits based on what has been resected and what remains, and then draw a defect template. Then, the various reconstruction approaches can be considered. For this reason, decisions regarding reconstructions

are best done after the defect has been fully created, especially for the less-common or complex defects. In practice, for the common scenarios and to facilitate the logistical arrangements required for a two-team surgical approach, a preoperative decision is made which limits the reconstructive options based on what the most likely defect and its associated functional deficit will be, and only modifications to one of the preselected options are performed.

The persistent factors affecting speech intelligibility are age, tumour localization, resection volume, method of reconstruction and administration of postoperative radiation therapy.^{19, 20} Loss of tongue mobility, loss of tissue bulk, loss of control of appropriate intraoral position of the tongue and lack of propulsive power are the greatest hindrances to proper swallowing.²¹ Therefore, the surgeon will strive to:

- retain as much viable, sensate, and motile native tissue as possible without violating sound oncologic principles;
- reconstruct the tongue epithelial surface primarily if possible, especially if postoperative radiation therapy is contemplated;
- maintain the separation between the oral cavity and neck (this is particularly relevant when the resection includes the adjacent floor of mouth and an ipsilateral neck dissection);
- preserve extrinsic tongue muscles and pharyngeal muscles as much as possible in the resection (where one or more of these has to be sacrificed, manoeuvres such as laryngeal suspension, cricopharyngeus myotomy and reconstruction of the oral diaphragm between the mandibular mylohyoid lines should be considered);
- preserve some tongue tip, or create a 'neo-tip,' possibly with a flap;
- preserve tongue mobility by avoiding tethering of the remaining tongue tissue;
- minimize the intraoral insensate area (consider sensory neurotization of the flap; unresected contralateral tongue, even if small as in some subtotal oral glossectomies, should be sensate and motile if at all possible);
- restore volume of lost tissue (anteriorly this will prevent depressed excavations which allow secretions and food to collect and pool; laterally–posteriorly this promotes the movement of an intraoral food bolus posteriorly and to the opposite functioning side and promotes the acquisition of good articulation);
- replace and stabilize resected mandible with vascularized bone secured by internal fixation, and preserve dentition according to dental goals (if osseointegration is planned, then the bone needs to be robust and of suitable vertical height; loss of vertical height anteriorly changes facial proportions).

Surgical options

The first decision that the surgeon must make after resecting oral tongue cancer is whether to close the defect or part of it primarily, or whether to use a flap for some or all of the reconstruction. In the pre-flap era, primary closure, closure by secondary

intention, or use of a tongue flap were all employed in the closure of sizeable defects. This resulted in significant impairment of swallowing and speech, even with small defects.⁶ The development of flaps opened up the possibilities of improved oral function in speech and swallowing,^{13, 22–26} and better cosmesis. Speech and swallowing may vary as to the advantage that each gains from flap reconstruction.^{27–29}

Decision making in surgery is based on the defect and resultant functional deficits being reconstructed, the flaps available, the technical facilities available, and the surgical team's experience. The final result is additionally strongly affected by the patient's age and motivation, the additional treatment given (for example radiation therapy) and the rehabilitation team's expertise.

Primary closure

Primary closure is usually possible when the defect allows the remaining dorsal tongue tissue to be coapted to remaining ventral tongue tissue. Loss of tongue volume of up to one third of the oral tongue in many cases will not give serious permanent swallowing problems.^{20, 30} Tongue loss can be overcome to a certain extent by compensatory mechanisms. When the resection involves ventral tongue to the extent that primary closure would result in tethering of the dorsal tongue tissue to the floor of the mouth mucosa, as when less than 1 cm of mucosa remains following a mid-third oral tongue resection between the resection margin and the gingiva, then an alternative method should be used.¹⁶

Healing by secondary intention

When a defect, in whole or in part, cannot be entirely closed primarily (or when a primarily closed defect opens in the postoperative period), healing by secondary intention follows. Such healing takes time, delaying subsequent treatment such as radiation therapy. During the healing, the patient has more local discomfort and must work diligently at oral hygiene. In some cases there is a greater chance of neck contamination. Once healing is complete, these sites can be harder to assess for early local recurrence. Nevertheless, in the appropriate patient this approach is well tolerated and the best option, and is frequently employed following laser resections.²⁰ Generally, swallowing is less impaired than is articulation with primary closure or healing by secondary intention.

Skin grafting, either by full thickness or split thickness

This is seldom the ideal method of reconstruction. Most small defects do not require this approach, and most larger defects are better managed otherwise, as restoration of bulk becomes increasingly important. Skin grafts may be applied to small defects which have a good recipient bed of tongue muscle, and where restoration of tissue volume is not necessary. In these cases the graft should be larger than the maximum dimensions of the defect to allow for some graft shrinkage.¹⁶ The use of a bolster dressing to maximize the chances of graft 'take' may be poorly tolerated

and some patients may require an otherwise-unnecessary tracheostomy. Furthermore, many such grafts simply function initially as biological dressings for a wound which then heals by secondary intention when the graft is lost, following which the grafted area progressively shrinks. 'Quilting' of the graft may avoid some of the above problems, but the shrinkage of the grafted site with a tendency for tethering remains a drawback. However, in a select few (usually edentulous) patients who are poor candidates for a flap option, and who have a shallow, uniform vascularized resection bed, this can be a useful technique. Mucosal grafts, which are of necessity small, do not offer any advantage over primary closure or skin grafts.

Intraoral local flaps

The attraction of these flaps is the ability to replace lost mucosa with mucosa. The limitations are the area that can be resurfaced, the lack of bulk, and with buccal donor sites, potential for contracture whether closed primarily or by a skin graft.

The first candidate for this approach is the tongue flap. Despite replacing 'like' with 'like,' it is almost never justifiable, because the surgery takes tissue from the remaining tongue with unnecessary additional functional compromise, and there are now many better reconstruction options. The second possibility is the facial artery myo-mucosal (FAMM) flap,^{31, 32} which is anterior to Stensen's duct and is supplied by the facial artery and based either superiorly or inferiorly.³³ The third option is the buccinator myomucosal island flap.^{34–36} Such flaps may be posteriorly based on the buccal artery from the internal maxillary artery, or anteriorly based from branches of the facial artery,^{37, 38} giving a good range of reach. In edentulous non-irradiated patients with a modest-sized defect where bulk is not a critical issue, this flap can be an excellent choice. The donor site morbidity is low if the flap is carefully planned and oriented.

Pedicled extraoral locoregional flaps

These options are useful when a free flap is not available, or not required, or not likely to be well tolerated by the patient.

The submental artery (SMA) flap was first described by Martin *et al.*³⁹ and has become a preferred option in some centres.⁴⁰ These flaps are robust pedicled flaps of good thickness and surface area which will reach all areas of the ipsilateral tongue, floor of mouth and oropharynx. Raising the flap can be moderately difficult, and the venous drainage is not always reliable. Flap viability in larger series is over 90%. However, their elevation requires dissection where nodal metastases from the oral tongue are prone to occur. Therefore, this flap must be used with caution, and only in cases where the neck, being easy to examine clinically and radiologically, is staged as N0. Some patients who are poor candidates for an alternative flap, and for whom postoperative radiation therapy is planned, are well served by this flap.

Infra-hyoid myocutaneous flaps⁴¹ have seen limited use in oral reconstruction as yet. In the presence of a tracheostomy

there is an increased risk of wound infection. The flap is fairly robust and mostly hairless, and is adequate to reconstruct modest-sized defects. It may be susceptible to venous insufficiency unless a modified approach is taken,⁴² and also requires careful dissection along the superior thyroid artery to preserve its blood supply. Raising this flap does not carry the same risk of violating the principles of a sound oncologic dissection as may be the case in the SMA flap, but caution is advised nevertheless.

The supraclavicular artery island flap (SCAIF) is a recently developed flap based on the supraclavicular branch of the transverse cervical artery, supplying the fasciocutaneous island over the shoulder tip and extending down over the mid-lateral deltoid muscle.⁴² It will reach the oral cavity for floor of mouth and tongue reconstructions. It offers pliable hairless skin of good size. Experience is still limited with it, thus its place still has to be determined. If its early promise is sustained, it may well displace some of the more demanding, time-consuming and expensive free flap options.

Pectoralis major myocutaneous (PMC) flap, first described by Ariyan⁴³ is presently more used in the salvage situation. It is easy to raise and fairly reliable. The main problems with it is that it suffers from a bulky pedicle relative to the usual tunnel between the neck and the oral cavity beneath an intact mandible, it creates an unsightly bulge in the neck, and its skin paddle is from the random part of the flap, all of which challenge viability. The skin does not regain useful sensation.⁴⁴ The flap recipient site will sag with time creating intraoral pooling, although technical refinements can minimize this problem.⁴⁵ There is significant donor site morbidity with this flap, with a cumulative effect on shoulder dysfunction in cases where there has been a neck dissection around the spinal accessory nerve.

The delto-pectoral flap, described by Bakamjian⁴⁶ became the first widely used axial pattern flap for major head and neck reconstruction. It is very occasionally useful in the intraoral reconstruction-salvage situation, but otherwise is mainly of historical significance. These flaps are easy to raise, and are usually reliable provided the dissection of the proximal pedicle does not endanger the supplying internal mammary artery perforators by coming too close to the midline, and the flap is made long enough to avoid tension when reaching the resection site. This gives the desired distal part of the flap a random arterial supply which increases risk of necrosis. The skin does not regain sensation unless reinnervated.⁴⁷ If the flap survives, it is often too thick when a thin flap is desired, or alternatively too thin to provide needed bulk. The pedicle is subject to compression as it enters the oral cavity, and conventionally it must be divided in a second stage. The reliability is lower than that of the PMC flap.

Naso-labial flaps, whether interpolated or not, offer a small area of tissue with limited reach, and find little application for tongue reconstruction.

Platysmal flaps are generally unreliable, thin and difficult to orientate for tongue reconstruction. As a result, they have found little use in these situations.

Free flaps

Free flaps are the most reliable reconstructive method, with over 95% viability common. They provide a non-mucosal desquamating surface of skin or skin graft which means that 'like' has not been replaced with 'like'. However, the skin surface becomes thinner in the intraoral environment. Although free flaps using jejunal wall to give a mucosal surface have been tried, a major abdominal procedure is not warranted except in the very rare case of incapacitating oral dryness. Free flaps can be neurotized, or allowed to reinnervate spontaneously from peripheral or resection bed nerve endings,⁴⁸ with some evidence that function of the reconstructed tongue is improved by surgical reinnervation.⁴⁹ A single free flap is used most often, sometimes with a multilobed skin island,⁵⁰ but multiple free flaps in extensive and complex defects can be employed, or a free flap can be combined with a non-free flap option. Free flaps most commonly used in oral tongue reconstruction are discussed below.

Radial forearm free flap

This venerable free flap⁵¹ is the commonest one used in intraoral reconstruction, and large series of cases have been reported.^{52, 53} It may be raised as a fasciocutaneous, or osteocutaneous flap. The skin paddle can be specifically designed for the defect²⁶ or a bilobed or multilobe design which allows the oral tongue and floor of mouth portions to be somewhat separate.¹⁶ A proximal fascial component (the 'beavertail' modification⁵⁴) can provide a random part which can then be shaped to provide bulk. The radial forearm free flap (RFFF) can be raised with two or more discreet skin paddles. The appeal of this flap in oral tongue defect reconstructions is its reliability, its long vascular pedicle which opens up a range of possible recipient vessels without the need for vein grafts, and most importantly its thin pliable versatile skin paddle of adequate size which makes this the best flap to prevent tongue tethering. Good speech and swallowing are reported after using this flap.⁵⁵ Modifications to the skin paddle giving extra bulk will facilitate tongue-palate contact and secondarily aid glosso-pharyngeal closure. The flap can be neurotized using the lateral antebrachial cutaneous nerve,⁵⁶⁻⁵⁸ but if not, some spontaneous return of sensation occurs over time.^{43, 59} Although there is no unequivocal evidence that neurotization itself gives better oral function than spontaneous reinnervation,²¹ perhaps only hastening the recovery of protective sensation, it can be recommended as a simple step in the procedure, which can only be of benefit or of no use, and cannot be harmful to the patient. When harvested as an osteocutaneous flap, the bone is unsubstantial, fragile and poorly tolerant of osteotomies, thus best limited to situations where a straight bony reconstruction which will not be expected to provide osseointegrated implants is acceptable. The radius bone at the donor site has a significant risk of distal fracture, and prophylactic plating in high-risk situations should be considered. The flap donor site which is usually closed with a split-thickness skin graft may be conspicuous. The graft may not always 'take', especially over exposed tendons even though the

paratenon layer is preserved. In selected cases, the flap donor site can be closed using a proximal advancement flap. Intraoral hair growth on the skin island can be a nuisance, but becomes less problematic over time, and is eliminated by postoperative radiation. Despite its limitations, this flap remains a frequent choice for oral tongue defect reconstructions where thin pliable skin to maintain tongue motility is the primary objective, and bulk is a secondary issue.

Anterolateral thigh flap

The anterolateral thigh flap (ALT), more recently popularized,^{60–62} provides a large skin paddle, although the skin is less pliable than the RFFF. It is reliable, provided that the perforating vessels are carefully identified and preserved, and it can have a moderately long pedicle. It can be reinnervated.⁶³ Harvesting can take place during the ablation procedure, adding to the efficiency of the operation. Donor site morbidity is minimal. The major limitations with it are the thickness of the subcutaneous tissue, which if too thick make it unsuitable for some oral tongue reconstructions. The flap can be raised in a suprafascial plane which makes it thinner overall. A variation, where a portion is thinned *in situ* prior to division of the pedicle to preserve the subdermal vascular plexus, has been used to give a thin part of the flap for the floor of mouth or to give tissue laterally over the defect from a marginal mandibular resection. The risk of flap failure is slightly higher than that of the RFFF as the perforator vessels are small, and the pedicle is more susceptible to compression in the tunnel between the oral cavity and the recipient neck vessels.

Lateral arm free flap

The lateral arm free flap (LAFF), first described by Song *et al.*,⁶⁴ has become popular for tongue reconstruction where more bulk is required.^{12,65} Being fasciocutaneous, it does not atrophy to any extent. The skin provided is more pliable in most patients than that of the anterior thigh region and it can be innervated using the posterior cutaneous nerve of the arm, which may enhance tongue function.⁶⁶ The donor site can be closed primarily. It can be modified by a proximal forearm extension,⁶⁷ and it has been used as a musculocutaneous flap, harvesting some triceps muscle with the skin flap.⁶⁶ The major limitation of this flap is its short pedicle with small-diameter vessels, making its revascularization more technically demanding, and consequently its failure rate somewhat higher. It is awkward to harvest this flap while the ablation is in process.

Brachioradialis musculocutaneous flap

The ability to offer innervated muscle and a reliable skin paddle which can be innervated make this a possible, although rarely selected free flap.⁶⁸ It is more challenging to raise, and imposes a small donor site penalty with about a 20% reduction in the strength of elbow flexion. The harvested brachioradialis muscle in some muscular patients can give considerable bulk which is mostly retained or restored with successful neurotization. The pedicle of this flap is shorter than the RFFF. Probably the most

telling strike against this flap and others involving reinnervated muscle, however, is the lack of high-quality evidence as to how significant neurotization of the transferred muscle is in improving reconstructed tongue function.

Rectus abdominis musculocutaneous free flap

Although this flap is used in head and neck reconstructions mainly for skull base, mid-face, and scalp defects, it can be used to reconstruct major (total or subtotal) defects of the oral tongue,⁶⁹ or more commonly of the combined mobile and posterior tongue. It is a reliable flap, whose main advantage is the provision of soft tissue bulk, which aids in the tongue–palate and/or tongue–pharyngeal wall apposition during the late oral and the pharyngeal phases of swallowing. It can be reinnervated, but spontaneous reinnervation from axons from remaining tongue tissue also occurs.⁷⁰ Of course a unidirectional muscle cannot duplicate the complexity of the extrinsic–intrinsic muscles, but some contraction may help with tongue–palate contact and bolus propulsion. It can have a long pedicle. It can be raised without the rectus muscle component, and only the anterior rectus sheath with the overlying fasciocutaneous island sustained by a single perforator. In this configuration, the anterior rectus sheath can be secured laterally in the oral defect to provide support for the remaining intraoral flap tissue.

Defects and reconstruction selection

Often, in oral tongue and related oral defects, the best results come from the cumulative benefit of several reconstructive manoeuvres. Nevertheless, these defects often carry significant functional disability in speech and swallowing which even the best reconstruction cannot correct. It is important that patients and the treatment team are well aware of this from the outset.

The following categorization of oral tongue regions is adopted because defects of each region cause different functional changes,⁷¹ which influence the reconstruction options.

Anterior third

Tip only

A midline tip defect is best closed primarily with remaining sensate tissue from each side. Although the tongue is thereby shortened, this is not usually a problem with eating, and gives minor articulation changes. Where the defect is unilateral, the remaining tip is best closed to itself, as this give a sensate although narrow tip. No significant eating difficulties result, and the articulation change is small, although it may be slightly more than in the primarily closed central defect.

Anterior third of oral tongue only

The remaining tip is ideally closed, giving a sensate residual tongue tip, and the more posterior portion of the defect can be closed primarily if no tethering would occur, or otherwise with a local intraoral flap or skin graft in selected cases. A free flap is

not usually required unless more than the anterior third of the unilateral oral tongue is resected.

Anterior third of oral tongue with floor of mouth, mandible preserved

The lesions requiring this type of resection may be primaries in the floor of mouth which spread to the tongue, or primaries in the tongue which spread to the anterior floor of mouth. In either case, the resection may be hindered by intact anterior mandibular teeth, and if so, the teeth should be extracted to guarantee adequacy of resection and/or to facilitate reconstruction. The main problems resulting from non-flap reconstructions, such as primary or secondary closures and skin grafts, are the significant tethering of the tongue with serious speech impairment and problems with the oral phase of swallowing, and pooling of saliva and fluids which predisposes to drooling. However, some of these cases can be adequately managed using an anteriorly based buccinator myomucosal island flap. The remainder are often best reconstructed using an RFFF.

Some lesions are deceptively small on the oral surface, but have a larger and deeply infiltrative extension. This situation can usually be anticipated by adequate clinical and imaging examination preoperatively, and confirmed by careful bimanual examination under anaesthesia before the resection commences. If the resection is deep into the the genioglossus/geniohyoid/hyoglossus muscles, sometimes including the mylohyoid and anterior digastric muscle, the surgeon must be aware of two additional dangers. The first is the possibility of extension to the inner table of the mentum, as this requires bone resection. Second, even if no bone needs to be resected, the additional problem created by the deep anterior muscle resection changes the nature of the defect and consequently the reconstruction. Restoration of the hyoid to mandible complex should be provided, and the deeper defect will need to be filled by some bulk, for which the RFFF may still be the best option, possibly using one of the modifications to the skin/fascial island.

Anterior third of oral tongue with floor of mouth and anterior mandible

It makes a difference if the anterior mandible resection is marginal or segmental. In the former case, restoration of the bony height is a lower priority than in the latter. Sensation to the lower lip should be preserved, if necessary by releasing the inferior alveolar nerve on at least one side from its bony canal. If the decision is to restore lost bony height, then this may be achieved to a degree by an onlay of bone from either the RFFF, which still gives the pliable skin for soft tissue reconstruction, or the fibula free flap (FFF), whose skin and soft tissue is less reliable and thicker. If the decision is made not to correct for the lost bone height after a marginal resection and only to reconstruct the soft tissue, then an RFFF can be used, and even in some cases (for example, in the salvage situation after flap failure) a split-thickness skin graft provided that the tongue tip is not tethered. When a full segmental resection is done, then the bone must be

reconstructed, and the flap usually used is the FFF, on which the bone can be 'double-barrelled' to give better bone height. Alternatives include a free iliac crest osteocutaneous flap.

Mid-third of oral tongue

This part of the oral tongue provides important tongue mobility and bulk in bolus propulsion, as well as central depression in bolus formation. The reconstruction cannot provide all of these functions. Bulk for bolus transport takes precedence over central depression in bolus preparation, and tissue volume restoration gives better articulation results than does leaving a central third of oral tongue volume deficit. The adjacent floor of mouth covers the lingual nerve as it courses to the anterior third of the oral tongue. When the nerve is segmentally resected, no restoration of sensation to the anterior third of the tongue is usually provided, as the unilateral sensory deficit is well tolerated.

Middle third of oral tongue only

The defects of the middle third of the oral tongue that can be closed primarily are those where the tongue mobility will not be compromised by that approach. As the medial resection approaches the midline furrow, increasing the vertical thickness of the tongue resected, there is greater tendency for tethering with primary closure, causing significant speech impairment. An RFFF will preserve mobility, but if more bulk is needed, either a modified RFFF or an LAFF will be useful. The ALT is usually too large unless adjacent portions of the oral tongue are also resected as in a true oral tongue hemi-glossectomy.⁷²

Mid-third oral tongue with floor of mouth

As the floor of mouth becomes part of the defect, then an RFFF or LAFF are useful options, or in some cases an SMA flap where there is no oncologic concern in the neck as noted above.

Mid-third oral tongue with floor of mouth and lateral mandible

If the mandible is segmentally resected, then bony replacement as part of the reconstruction is preferred, as the use of a reconstructive plate alone frequently results in eventual plate exposure. Since the defect is lateral, there may be no need for bony osteotomy. In that case, an osteocutaneous RFFF may be a good option especially if no dental implants are planned. Where the bony defect is more extensive, or where osteotomies are required, or when osseointegrated implants are planned, then an FFF is most commonly employed.

Posterior third of oral tongue

This portion of the tongue is required in propulsion of the oral bolus posteriorly, and in apposition against the palate superiorly and towards the pharyngeal wall posteriorly for both swallowing and speech. It works in concert with the extrinsic tongue muscles and the pharyngeal constrictors in a coordinated fashion. The lingual nerve is usually resected, and frequently also the hypoglossal nerve.

Posterior third oral tongue with/without lateral soft tissue

When the defect is of this part of the tongue only, in many cases it can be closed primarily or allowed to heal by secondary intention. When properly selected, the resultant narrowing of the entry into the oropharynx compensates for the reduction in tissue volume. Such isolated defects are fairly rare, as resections more commonly involve one or more neighbouring regions such as the adjacent oral tongue anteriorly, base of tongue posteriorly, the oropharynx superolaterally, or the mandible laterally, in which case a flap reconstruction should be performed to restore missing volume, retain mobility and restore mandible continuity (see below). Nerve grafting to bridge the lingual nerve gap is done if possible. The hypoglossal nerve is not reconstructed, but if the branch to the base of the tongue is saved then the pharyngeal phase of swallowing will be better.

Posterior third oral tongue and lateral soft tissue plus mandible

These defects are common, and involve sacrifice of the inferior alveolar nerve, the lingual nerve and usually a segment of the jaw, although a marginal resection is sometime possible, sparing the need for bony reconstruction. The best reconstructions involve nerve grafting for the inferior alveolar and lingual nerves, neurotization of the free flap skin from the proximal lingual stump, soft tissue replacement with fasciocutaneous component of the free flap and bone reconstruction as indicated. The commonest flap in these situations is the FFF, although if no bone is required, an LAFF or even an RFFF can be good options. The skin island should not reconstruct a large area of the base of tongue portion of an extended defect to avoid insensate skin being just above the larynx, a situation which favours aspiration; rather, the base of tongue part of the defect should be closed with sensate base of tongue remnant as is usually possible when the defect is less than half of the base of tongue.

Bilateral oral tongue

Bilateral oral tongue resections almost always proceed from anterior to posterior in terms of tissue resected; it is unusual to spare anterior portion(s) of the tongue and yet have bilateral oral tongue resected posteriorly. Preservation of motility and sensation to the unresected tongue is paramount for best results.

Anterior thirds of oral tongue

Reconstruction of the anterior tongue using a flap designed to give enough bulk for a 'tip', and with enough deep support of the reconstruction to preserve ability to contact anterior upper palate/teeth is the goal here. Sometimes, when the defect is asymmetrical, the side with the most tongue tissue remaining can be formed into a neo-tip of more use than a flap-constructed tip. The defect is limited when tongue alone is involved, in which case an RFFF will usually give enough bulk using one of the skin paddle modifications. Sometimes an LAFF is appropriate, although its short pedicle in this position can be

problematic. Neurotization of the flap is used. Where the defect includes the deep anterior extrinsic tongue muscles then a laryngeal suspension⁷³ and possible reconstruction of the oral platform between the mylohyoid lines will convert a deep defect to the more superficial one, allowing the same reconstruction as noted above with an RFFF or LAFF. Where bone is included in the resection, the reconstructive approach will follow that of 'Anterior third of oral tongue with floor of mouth and anterior mandible' above.

Anterior two-thirds of oral tongue/subtotal oral tongue glossectomy

In these defects, the vertical height and bulk of the reconstruction is even more important to maximize speech and swallowing, with mobility derived from the movement of the surviving tongue tissues and extrinsic tongue muscles. As above, where the deep tissues are resected, a laryngeal suspension and reconstruction of the oral platform are very helpful. The flap is usually either an ALT or rectus abdominis musculocutaneous free flap (RAMFF), but an RFFF or LAFF may be acceptable in some patients with adequate flap subcutaneous fat. The volume of soft tissue resected should be slightly overcorrected by the reconstruction. An FFF is commonly used if mandibular reconstruction is required. Various attempts have been proposed to make the reconstructed tongue assume the specific shape of the normal tongue.^{21, 27, 74, 75}

Total oral tongue glossectomy (may be termed a 'subtotal glossectomy')

Major defects such as this often include extrinsic muscles also, and are very difficult to reconstruct without obvious disability in eating and speech. Preservation of as much sensate and motile tongue base is essential to prevent chronic aspiration, and laryngeal suspension is used routinely. It is important to provide support for the intraoral reconstruction, whether by bone as with the iliac crest⁷⁶ or with soft tissue such as muscle.¹⁷ The commonest flaps employed are the ALT, the RAMFF or the gracilis muscle free flap,^{77, 78} taking care to overcorrect for lost volume. A strong case can be made for reinnervated muscle when such a flap is used. An occasional case where extrinsic tongue muscle loss is minimal can be reconstructed with an RFFF modified for volume augmentation, or with a brachioradialis musculocutaneous free flap. The reconstruction must ensure support for the intraoral tissue.

Conclusion

The oral tongue is difficult to reconstruct because of its sophisticated anatomy and coordinated movements in speech and swallowing. With attention to the defect created and the resultant functional deficits, the reconstructive surgeon should be able to select a reconstructive option which minimizes the impact of surgical treatment on these unfortunate patients.

Selected cases

Submental artery flap

This 55-year-old woman had a T2N0 squamous cell carcinoma (SCC) mainly of left middle third of oral tongue resected with posterior third, and coming within 1 cm of the midline, along with a selective ipsilateral neck dissection (Figure 25.1). Reconstruction was by a pedicled SMA flap. Postoperatively she received radiation therapy as there was a 7 mm node involved with cancer showing microscopic extracapsular spread (pN1). Now 7 years without recurrence, she has nearly normal speech and eats a full oral diet.

Lateral arm free flap

A 65-year-old man with a T3N1 SCC of the posterior third, and small amounts of adjacent middle third and base of tongue, underwent resection which spared the anterior third of oral tongue, crossing the midline furrow by 1 cm in the mid and posterior thirds (Figure 25.2). The base of tongue defect was closed primarily. An LAFF was used to reconstruct the soft tissue. He received postoperative radiation therapy. At eight months, he remains free of disease, and his speech is entirely intelligible with only some minor dysarthria. He eats a full diet.

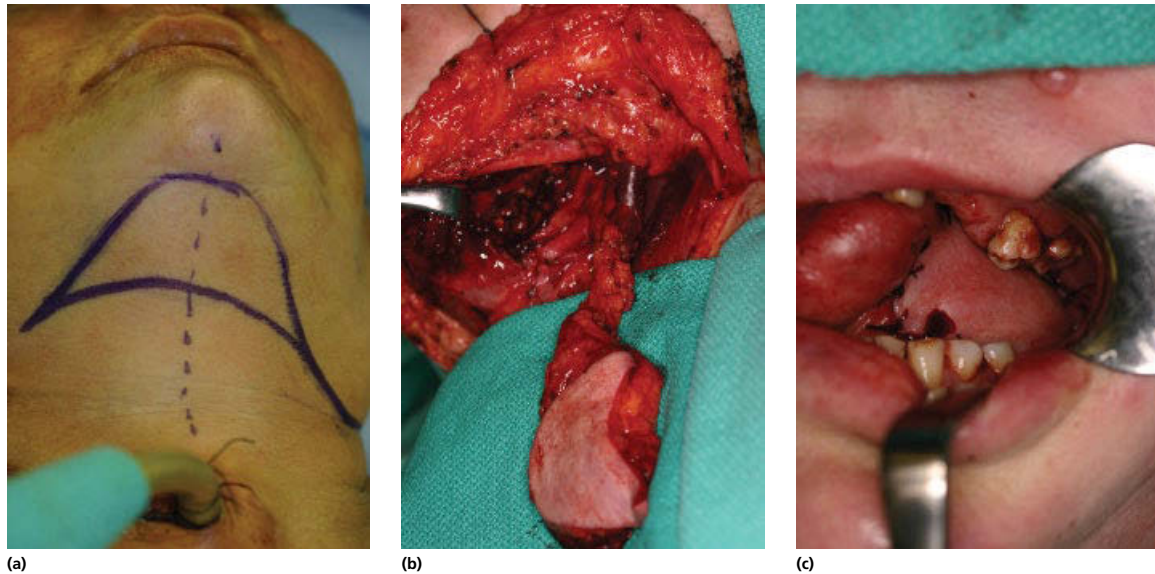


Figure 25.1 Submental artery flap. (a) Outline of left-sided pedicled SMA flap. (b) Left pedicled SMA flap raised. (c) SMA flap inset into left tongue defect.

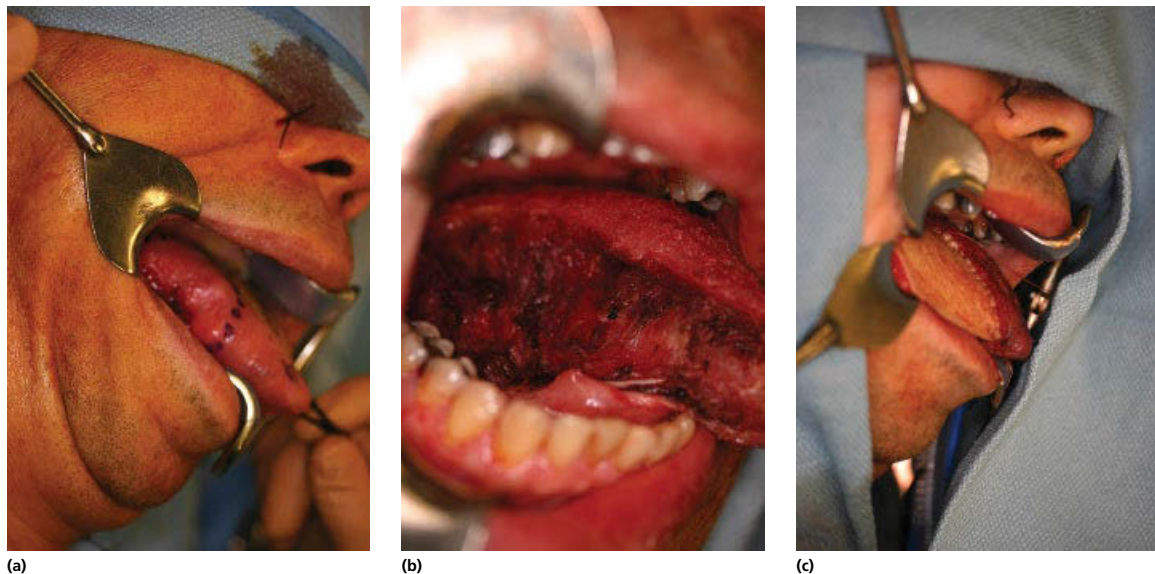


Figure 25.2 Lateral arm free flap. (a) View of T3 SCC of right oral tongue. (b) Defect following resection of SCC of right oral tongue. (c) Lateral arm free flap inset into right tongue defect, providing good bulk.

Radial forearm flap

The resection on this 75-year-old man whose large T2N0 SCC straddled the middle and posterior thirds of the oral tongue included the posterior aspect of the anterior third and all of the middle and posterior thirds of the oral tongue, with a uniform fairly shallow resection (Figure 25.3). Reconstruction was by an RFFF. There was no radiation therapy. At over 4 years, he is free of disease, his speech is fully intelligible, and he eats a full diet.

Anterolateral thigh free flap, neurotized, with laryngeal suspension

A 55-year-old man, with a T4 left oral tongue cancer involving almost all of the left oral tongue, including the root of tongue, spreading across the floor of mouth to within 1.0 cm of the gingiva, crossing to within 1.5 cm of the right lateral tongue lateral border in the middle third, extending into the base of tongue and involving the lower half of the lateral oropharyngeal wall (Figure 25.4). Resection by a marginal mandibulectomy plus anterior

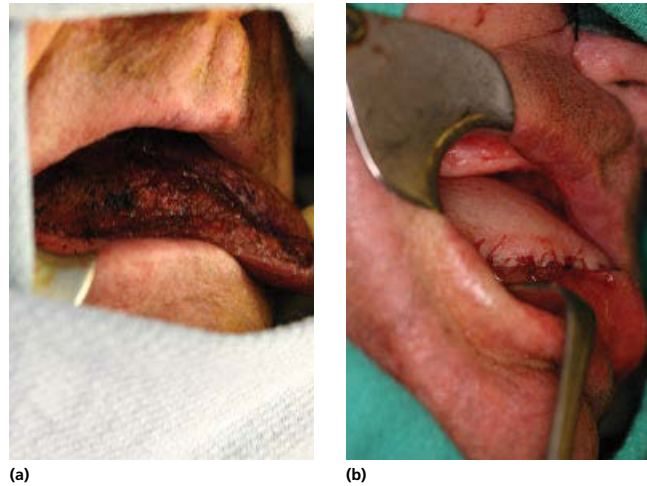


Figure 25.3 Radial forearm flap. (a) Shallow defect created by resection of T2 SCC right oral tongue. (b) Inset RFFF skin paddle into right tongue defect.

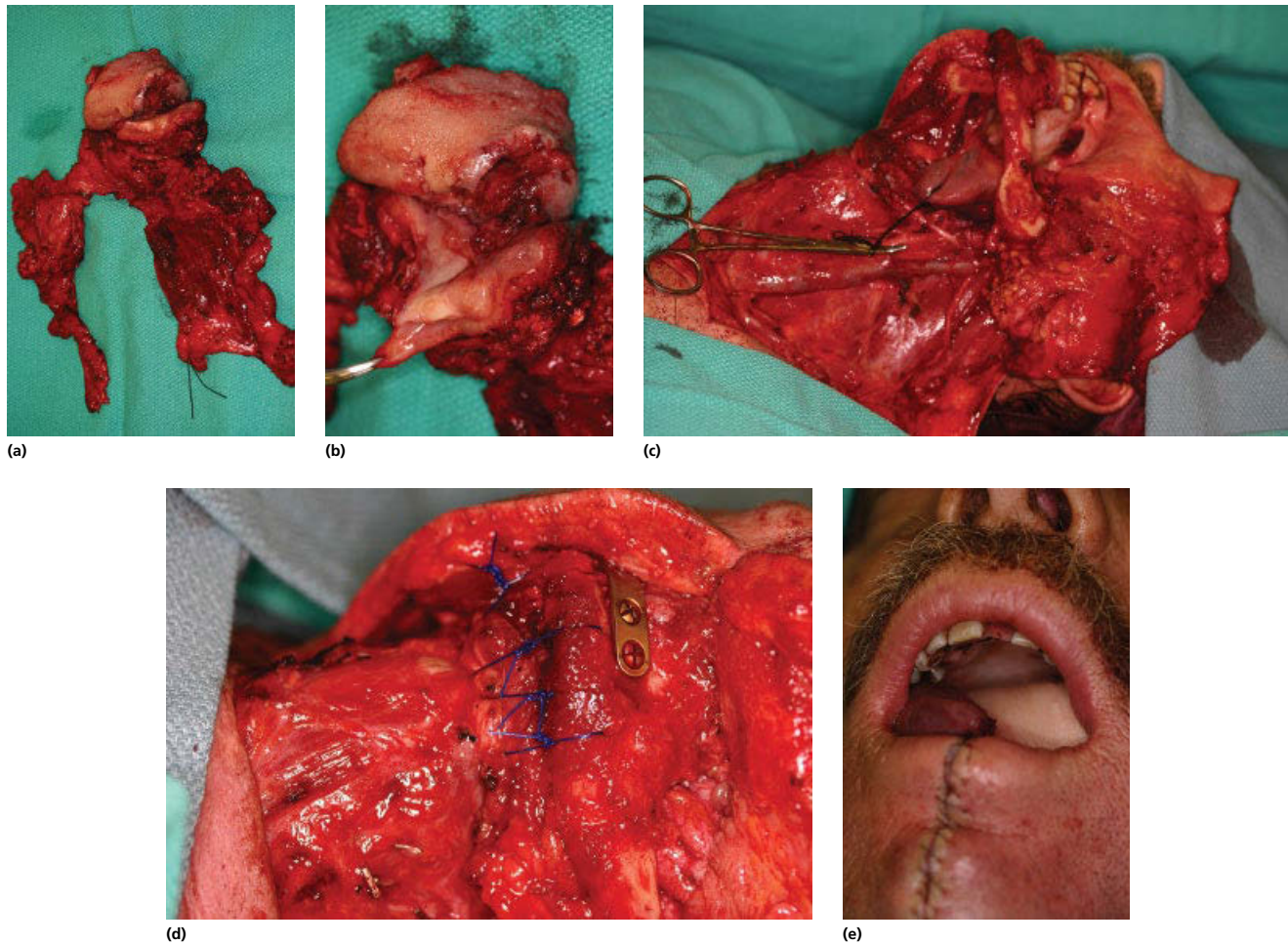


Figure 25.4 Anterolateral thigh free flap, neurotized, with laryngeal suspension. (a) Resected specimen of T4 tongue cancer. (b) Extensive involvement of almost all of the left oral tongue extending to left floor of mouth, with resection including marginal mandibulectomy. (c) Small remnant of contralateral oral tongue and root of left tongue remaining; a mandibulotomy to assist with exposure has been done.



Figure 25.5 Free fibula osteocutaneous free flap. Insufficient soft tissue from the skin paddle of a free osteo-cutaneous fibula flap has severely compromised this patient's speech, and rendered him feeding-tube dependent.



Figure 25.6 Radial forearm flap, with mesh to reconstruct oral diaphragm. The radial forearm fascio-cutaneous free flap has been supported by replacing the resected oral "diaphragm" with synthetic mesh, and the hyo-mandibular complex further restored by a laryngeal suspension, giving good speech and swallowing of pureed foods.

mandibulotomy, left only the anterior third, some root and extrinsic muscles, a lateral middle third margin and some posterior third and the base, all on the contralateral side. Reconstruction involved partial base of tongue primary closure, some primary closure of the residual contralateral tongue tip, and soft tissue replacement with a neurotized ALT flap. The reconstruction gave incomplete palato-glossal contact, with a prolonged oral transit stage, and abnormal oropharyngeal stage. He regained swallowing of fluids and some oral soft foods without on-going gastrostomy tube feeds, but his speech was markedly impaired. He had postoperative radiation therapy complicated by osteoradionecrosis requiring a second flap (FFF), and then developed local recurrence at 12 months. This proved highly responsive to three cycles of chemotherapy and he survived for the next 3 years.

Free fibula osteocutaneous free flap

This man shows a suboptimal reconstruction from 18 years previously using an osteocutaneous FFF after a subtotal oral glossectomy and mandibulectomy (Figure 25.5). The patient received postoperative radiation therapy. The soft tissue was inadequate and almost immobile, severely compromising his oral and pharyngeal phases of swallowing, and rendering his speech barely intelligible. He remains feeding-tube dependent. He has refused all offers of further reconstructions.

Radial forearm flap, with mesh to reconstruct oral diaphragm

This man, now 12 years post operative after a subtotal oral glossectomy and marginal mandibulectomy reconstructed with laryngeal suspension, restoration of the oral mandible 'diaphragm' with mesh to allow the RFFF to have adequate oral height, has intelligible speech and problem-free swallowing of fluids and pureed food (Figure 25.6). He has been fully employed since his treatment.

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CHAPTER 26

Mandibular reconstruction

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Introduction

The mandible and its attached musculature play an important role during many social interactions, including eating, drinking, talking and maintenance of oral continence. Aesthetically, its inferior margin defines the lower third of the face, its dimensions contribute to facial height, width and profile, and its dentition highlights a smile. A functionally asymmetric mandible can lead to long-term problems, including dental malocclusion, articulation difficulties, painful temporomandibular joint pathology, and unattractive static and dynamic facial disproportions. Accordingly, a suboptimal mandibular reconstruction can impact multifactorially on overall psychosocial wellbeing and postoperative societal reintegration. An ideal mandibular reconstruction should restore functional and aesthetic harmony not only for the skeletal deficit but also for the adjacent soft tissues and dentition, preferably with minimal donor site morbidity.

The mandibular reconstruction itself is just one aspect of a multidisciplinary, holistic treatment approach for the patient. The treating team should include experienced and subspecialty-trained reconstructive and ablative surgeons working alongside medical oncologists, radiographers, pathologists, radiotherapists, anaesthetists, psychologists and various dental, prosthodontic and rehabilitation specialists. These patients should therefore be managed in a specialist centre.

Mandibular anatomy

The mandible is constructed embryologically of two symmetric halves that are each formed from an individual ossification centre by intramembranous ossification. At birth, they are united at the midline by a fibrous symphysis; ossification is completed by the age of 2 years.^{1,2} It is the largest, strongest and only mobile bone of the facial skeleton. It articulates at the temporomandibular joints, each of which provides 6 degrees of freedom. This remarkable freedom of motion benefits from mechanical redundancy of the muscles of mastication.³

The mandible can be anatomically divided into one horizontal component (the symphysis and the body on each side), two vertical components (the angle, ramus, coronoid process and condylar process make up one vertical component on each side) and three processes (coronoid, condylar and alveolar). The mandible is elegantly designed to withstand the enormous forces of mastication. Thick cortical bone presides in areas that withstand unidirectional forces, such as at the condylar neck, and the bone possesses trabeculae where it needs to endure multidirectional forces, such as the alveolar process and condylar head.⁴ Pressure on the dentition is transmitted to underlying trabecular bone, which remodels throughout life according to dental loss, movement and the individual's masticatory habits. Adults possess 16 mandibular teeth: two central incisors, two lateral incisors, two canines, two first premolars, two second premolars and six molars. These occlude against their 16 maxillary counterparts.

The mandible transmits the bilateral inferior alveolar neurovascular bundles that deliver sensation from the dentition and related mucosa, and the chin and lower lip via the mental nerve. This inferior alveolar neurovascular bundle is the source of axial blood supply for the mandible; however significant blood supply is also derived from its periosteum and attached muscles. These muscles are involved in mastication, deglutition, oral continence, speech and, to a lesser extent, facial expression.

Mandibular defects

The most common indication for mandibular reconstruction is segmental bone loss. The vast majority of such defects result from soft tissue tumour ablations that require segmental bony resection for local control. Neoplasms of bone origin may also require segmental resection, but these are less common. Primary traumatic segmental loss is also uncommon; when it occurs it is usually due to a gunshot wound. Non-neoplastic conditions such as osteoradionecrosis, osteomyelitis and drug-related osteonecrosis (classically due to bisphosphonates) may also require

segmental resection for cure. Marginal mandibulectomies are generally not reconstructed as they do not inflict the same detrimental impact, since the mandible remains in continuity. Marginal mandibulectomies are, however, amenable to height augmentation using non-vascularized bone grafts or, in specific circumstances, vascularized bone flaps for dental rehabilitation.^{5,6}

Many systems have been proposed for classifying segmental mandibular defects. In our experience, the easiest and clearest is the scheme devised by Daniel.⁷ We have adopted its use routinely for audit, research and communication for ablation/reconstruction planning. This system always recognized the importance of the soft tissue deficit, as follows:

- 'isolated bone',
- 'compound' (bone and oral lining or skin),
- 'composite' (bone, oral lining and skin), or
- 'extensive composite' (bone, oral lining, skin and intervening soft tissues).⁷

The segment of bone that is missing is simply described according to the region affected (e.g. 'left angle to right parasymphysis'). Some additionally describe the defect in terms of the teeth that are resected with the segmental defect (e.g. 'left canine, right second premolar'); however, this is limited to dentulous patients and to the region between the mandibular angles.

Another popular system was proposed by Jewer *et al.*, who divided the mandible into 'H', 'C' and 'L' regions.⁸ A lateral defect of any length is described as an 'H' defect when the condyle is affected, or an 'L' defect when it is not. A 'C' defect is one that involves the central segment between the canines. For example, an angle-to-angle defect would be described as 'LCL' and a mandibular body defect as 'L'. The original classification did not consider the soft tissue deficit and was therefore later modified by adding the letters *o* for osseous defect only, *m* for mucosa, *s* for external skin and *ms* for combined surface defects.⁹ The loss of intervening soft tissue bulk is not accounted for by this system so it does not differentiate between *composite* and *extensive composite* defects.

Segmental mandibular reconstruction: principles and options

The reconstructive surgeon and multidisciplinary team will be faced with important decisions during patient selection. Some will be better served overall by a downgraded reconstruction whereas for others the best possible reconstruction should be offered.^{10,11} This decision will be affected by the patient's wishes, expectations and medical comorbidities, as well as the outcome of preoperative staging investigations. The intent of treatments (curative or palliative), and the planned ablative defect (dimensions and components), must be clearly communicated as these will all affect the choice of donor site(s) for tissue transfer. It should be noted that it is not unusual for resectional plans to change with intraoperative findings; patients should be made aware of this when providing informed consent to the overall procedure.

Segmental mandibular defects, however small, should not be collapsed for primary fixation. Even minimal malocclusion, which would be almost inevitable with this approach, causes chronic frustration for patients due to the lack of sensory feedback from unopposed teeth and symptomatic alterations in masticatory function. Likewise, no segmental mandibular defect should be left floating, as muscle actions become unopposed, the incongruence worsens, and the soft tissues contract and shorten, exacerbating the problem and complicating secondary reconstruction.

Segmental mandibular reconstruction should be completed within the same anaesthetic as an ablation, and primary healing achieved expeditiously so that adjuvant treatments are not delayed. Options include microsurgical and non-microsurgical reconstructions. Attention should also be directed at inferior alveolar nerve reconstruction, especially in benign cases, since lower lip numbness and/or dysaesthesiae are bothersome morbidities for patients, such as when sipping or testing the temperature of a drink, kissing, or when trying to maintain oral continence.¹²

Microsurgical free tissue transfer: choosing a single free flap for reconstruction

For patients who can undergo curative resection and microsurgery, the reconstructive procedure of choice for almost any ablative segmental mandibular defect is a free vascularized osseous flap that incorporates adequate soft tissues and inflicts minimal donor site morbidity. Desirable characteristics include high-quality bone stock, lengthy availability of donor bone, versatility in tailoring of the bony contour, flexibility in tridimensional inset of pliable and thin soft tissues, ability of the bone to support osseointegrated dental implants, and the option for a two-team ablative/reconstructive simultaneous approach. All of these features, and others, are fully provided by the fibula osteoseptocutaneous free flap, which has increasingly been accepted internationally as the best donor for segmental mandibular reconstruction.^{10,11,13–17}

A single fibula osteoseptocutaneous free flap will provide a good reconstruction for *isolated bone* and *compound* defects, and for many *composite* defects. Usually it cannot provide adequate soft tissues for *extensive composite* defects without inflicting too much donor site morbidity from extensive muscular harvest.^{10,17–19} The anatomy and harvest of this flap have been extensively described, but there are some key points that are important to stress.

First, the fibula should always be harvested with a skin paddle, even for *isolated bone* defects (when it should be harvested thin enough to allow comfortable primary closure of the donor site), as it will serve as an invaluable monitor of vascularity for the underlying bone.¹⁵ The skin paddle also serves to facilitate tension-free soft tissue closure at the recipient site and augment volume or contour if needed. Second, angiography of the donor leg is not a routine requirement; it should be considered if the pedal pulses are abnormal on clinical assessment or if there is a history of peripheral vascular disease or significant leg trauma.²⁰ Third, the vascularity of the skin paddle is reliable when correctly

harvested.¹⁵ Fourth, it is important not to harvest the proximal or distal 6 cm of fibula in order to preserve ankle and knee stability. Fifth, primary donor site closure should only be employed if the surgeon is confident that there is no risk of compartment syndrome from excessive tension; increasing postoperative leg pain and/or swelling, in particular, should be monitored for and a low threshold maintained for releasing the donor site for skin grafting if encountered. Sixth, it is important to exclude arteria peronea magna directly before harvest of the peroneal vessels; this is performed by inspecting the peroneal vessels for excessive size and by placing a microvascular clamp across the distal peroneal vessels early in the flap harvest to ensure maintenance of pedal pulses and absence of foot ischaemia. These last three measures remove the risk for the rarer but most significant potential donor site morbidities from fibula harvest. Other reported donor site morbidities from fibula harvest are also generally avoidable. For example, preservation of the neurovascular supply to the flexor hallucis longus muscle during fibula harvest and meticulous reapproximation of muscle origins to the remaining interosseous membrane under correct tension are important points of technique that effectively prevent toe flexion contracture and weakness. Additionally, suprafascial dissection of the skin paddle anterior to the posterior crural septum is important for preserving the superficial peroneal nerve branches. The preservation of paratenon is important to skin graft take over the peroneal tendons. Ling and Peng recently performed a literature-wide systematic review and provided level II evidence that the price of fibula harvest is low in terms of donor site morbidity as long as harvesting technique and donor site care are sound.²¹

Specific advantages of the fibula osteoseptocutaneous flap that make it ideal for mandibular reconstruction include its: (a) unmatched supply of strong, straight, lengthy bone of nearly uniform thickness and biomechanically sturdy triangular cross-section; (b) reliable acceptance of osseointegrated dental implants; (c) sizeable (2–3 mm) and lengthy pedicle; (d) thin, pliable, sizeable skin paddle (up to 22–25 cm in length and 10–14 cm in breadth) that can be inset into almost any configuration with respect to the bone; (e) potential usefulness as a chimeric or flow-through flap; (f) donor site that is an ideal nerve graft donor site for inferior alveolar nerve reconstruction if indicated; (g) distance away from the head and neck region, which invites a two-team approach to ablation and flap harvest; (h) tolerability of multiple osteotomies for bone shaping.¹¹

For these reasons, our first choice source of vascularized bone for segmental mandibular reconstruction is the left fibula osteoseptocutaneous flap and our second choice is the right fibula. It is therefore unusual for us to resort to any of the numerous alternative osseous flaps. These include harvests from the iliac crest, scapula, radius, rib, humerus and metatarsals. Although some prefer to use the vascularized iliac crest, the skin paddle is unsuitably bulky (even in slim patients) for lining the oral cavity and has limited mobility with respect to the bone.²² In addition, the pedicle is short, the deep circumflex iliac artery

supply is tenuous to the skin paddle,²³ and although the bone provides excellent bone stock it does not tolerate shaping osteotomies well. Donor site morbidity for the iliac crest includes protracted gait pain and/or disturbance, lateral femoral cutaneous nerve dysaesthesiae, abdominal wall structural problems such as hernia formation, and pelvic fracture, although, like for the fibula harvest, particular points of technique allow donor site morbidity for this flap to be significantly reduced.^{24–29}

Although the radial forearm provides a pliable, thin, hairless and reliable cutaneous or fasciocutaneous flap, the underlying radius should be regarded as a poor choice of donor vascularized bone for the mandible due to its intolerance of tailoring osteotomies, lack of bone stock, poor ability to support osseointegrated implants and the high risk for subsequent radius fracture. Some have resorted to bone grafting (using iliac crest bone graft) and/or plating the radius following its harvest in order to reduce the risk of secondary radius fracture, but this approach inflicts donor site morbidity in an additional location that is itself fraught with donor site morbidities, and prophylactic plating is not cost-effective.^{30,31}

The scapula donor site provides versatility regarding the soft tissue reconstruction, but the quality of the donor bone does not approach that of the fibula, and its location on the back is a major drawback since it precludes a two-team approach, prolonging the operation.

Technique of reconstruction: fibula osteoseptocutaneous free flap

The ablative and reconstructive teams should communicate clearly preoperatively to agree the likely defect and reconstructive requirements; this allows them to work simultaneously to reduce operative time. Once the ablative operation is completed, the premorbid occlusal relationship should be defined by maxillo-mandibular temporary fixation if dentition allows. This facilitates accurate shaping and fixation of the reconstruction plate. Alternatively, the reconstruction plate can be shaped against preoperatively fashioned mandibular templates or purchased pre-bent to standardized mandibular anatomy (available in different sizes). Maxillo-mandibular fixation can be released once each end of the reconstruction plate has been secured to the native mandible; tandem and full range of movement of the temporomandibular joints can thus be verified. Mini-plates may alternatively be chosen for fixation.

The recipient vessels are then prepared (usually branches of the ipsilateral external carotid and internal/external jugular systems, but may be contralateral or from the transverse cervical vessels if necessary) and the flap devascularized for shaping on a side table. Osteocytes are known not to be sensitive to a moderate period of ischaemia,³² which is usually well within 4 h for this flap, so we recommend that all osteotomies of the fibula should be performed at a side table. Unequalled surgical freedom is granted by freeing the fibula osteoseptocutaneous flap from the leg. It allows checking of planned osteotomy sites and angulations directly within the mandibular defect; rechecking of

completed osteotomies one-by-one against the defect before proceeding to the next osteotomy in case fine adjustments are required; draping of the flap skin paddle within the soft tissue defect to assess the site of the first osteotomy (which defines the length of fibula bone that will be discarded); freedom to rotate and manipulate the flap in hand to gain the most accurate appreciation of angulations in any plane; and trial inset(s) with temporary sutures to check and recheck any bony or soft tissue adjustments made on the flap.

The position of the first osteotomy should account for four main factors: first, the location of the blood vessels branching off the peroneal pedicle to supply the skin paddle; second, the required pedicle length (since this can be increased by dissecting it subperiosteally off the proximal fibula); third, there must be enough bone distal to the first osteotomy to complete the segmental defect reconstruction; fourth, it needs to be appreciated that every osteotomy will reduce available fibula bone length for reconstruction (this is particularly the case for wedge osteotomies, or if burring of osteotomy sites is required to refine accuracy of angulations). A fifth consideration is important when double-barrelling the fibula (useful for restoring either/both alveolar ridge height and facial height), which is that a segment of fibula no less than 3 cm in length should be removed subperiosteally at the folding hinge point and discarded.^{11,33} This is to allow double-barrelling without causing any kink, twist or stretch of the pedicle/periosteum between the barrels. All of these assessments are easier if the flap is in hand rather than still attached to the leg.

Osteotomies must be accurate and safe. They are best performed by the lead reconstructive microsurgeon with one assistant surgeon and one scrub nurse. The lead surgeon plans, marks, executes and checks the accuracy of all osteotomies. The lead surgeon holds one end of the fibula with a sharp-tipped bone holder (these do not crush the periosteum) using the non-dominant hand, and holds the oscillating saw with the dominant hand. The assistant surgeon stabilizes the other end of the fibula with another sharp-tipped bone holder and protects the pedicle (this is achieved by passing a flat instrument, such as a McDonald retractor, subperiosteally between the fibula and the pedicle in order to prevent the saw from damaging the periosteum/pedicle). The scrub nurse (or second assistant surgeon) is responsible for irrigation during osteotomies to prevent any bone heating, and irrigation of the flap tissues to prevent desiccation. Additionally, we recommend that osteotomies produce segments of fibula no less than 3 cm in length. Any shorter, and the blood supply to that segment will likely be threatened. A careful and sensible balance needs to be struck between exactly mimicking the curvature of the mandible and maintaining the blood supply to each fibula segment.

Insetting generally proceeds with the oral lining reconstruction first, followed by the bone. The soft tissue reconstruction deserves meticulous attention, as it is of at least equal importance to the bony reconstruction. Soft tissue deficiency is poorly tolerated in the head and neck region, especially when cavities

are left unfilled as this invites fluid accumulation and infection. It is important that the oral lining reconstruction is water-tight so as to avoid salivary leakage around the mandible reconstruction and into the neck. Additionally, soft tissue tethering places a check-rein on the patient's functional potential. Poor tongue mobility, for example, may result from insufficient tissue redundancy or pliability. Conversely, an overly bulky flap may hinder a functional residual hemi-tongue and yet may be necessary for a subtotal glossectomy reconstruction to achieve occlusion against the palate. Likewise, postreconstruction trismus may be the result of contracted or inadequate buccal or retromolar trigone lining, whereas an overly bulky reconstruction in these subsites will lead to dental trauma to the flap. Accordingly, untethered soft tissues with appropriate redundancy and yet without excessive bulk, is the desired soft tissue reconstruction goal.¹⁰ Long-term soft tissue shrinkage should be expected and accounted for when designing the soft tissue reconstruction, particularly in patients who will receive adjuvant radiotherapy. Additional mobility of the skin paddle with respect to the bone is possible by selectively incising the septum either side of its supplying vessels. For *isolated bone* defects, the skin paddle can be concealed intraorally or submentally within an incision used during the ablation and excised at a later date if necessary.

Bony fixation needs to be rigid in order to allow postoperative rehabilitation of the temporomandibular joints. Monocortical screws are recommended to safeguard blood supply; they can gain tip purchase of the second cortex for additional stability but must not encroach upon the overlying periosteum. Microsurgical anastomoses are performed after all inset(s) is complete (Figure 26.1).

Microsurgical free tissue transfer: performing a double free flap reconstruction

Although a fibula osteoseptocutaneous free flap can import a sizeable fasciocutaneous paddle and, if absolutely necessary, some muscle (e.g. soleus and/or flexor hallucis longus) with the bone, *extensive composite* mandibular defects generally require even more volume. Recruitment of additional muscles from the leg to maximize the soft tissue volume available with the fibula increases the donor site morbidity. In such circumstances, a double free flap can provide a superior reconstruction.^{10,17}

Some may argue that the morbidity of having one donor site is better than having two. However, when one donor site is large, has significant muscular harvest for volume and cannot be primarily closed, then its morbidity could actually be more severe than using two smaller donor sites that each preserves donor site function. In effect, the volume and morbidity requirements for a large complex defect are shared by two donor sites rather than inflicted on only one.¹⁰

By performing a double free flap, the best donor osseous and soft tissue characteristics can be selected independently to satisfy the requirements of the defect. This improves inset(s), manoeuvrability and conformability, thus avoiding critical flap



Figure 26.1 Compound segmental mandibular defect reconstructed with a fibula osteoseptocutaneous flap. (a) Squamous cell carcinoma of the left lower gum. (b) Compound segmental mandibular defect stabilized with a reconstruction plate (note the associated extensive intraoral mucosa defect). (c) Skin island of the fibula osteoseptocutaneous flap used for intraoral mucosa lining reconstruction. (d) Redundant portion of the skin island de-epithelialised and used for soft tissue augmentation over the fibula–reconstruction plate construct. (e) Appearance immediately after revascularization of the fibula osteoseptocutaneous flap at the reconstruction site. (f, g) Appearance at 6 years follow-up.

folding even with large volumes of tissues. Our usual choices for a double free flap mandibular reconstruction are the fibula osteoseptocutaneous flap for mandible with oral lining and the anterolateral thigh flap for cheek volume and external facial coverage. Both flaps are far away from the head and neck region, and therefore a two-team approach is possible throughout the double flap procedure.

The main deterrent to performing a double free flap in most centres is probably the length of surgery. However, this can readily be reduced with efficient team work; indeed it has been demonstrated in many centres, including our own, that the difference in operating times between single and double free flap procedures can be negligible.^{10,17} To achieve this, two operating teams should work simultaneously throughout the operation. First, the ablative and the first reconstructive

team work together; second, the second reconstructive team works alongside the first reconstructive team until the end of the operation. Accordingly, the operation will begin with the first reconstructive team raising the flap that will be used to line the oral cavity (usually this would be the fibula osteoseptocutaneous flap) while the ablation is underway. When ablation is complete, the second reconstructive team raises the second flap (usually the soft tissue free flap that will be used to fill the cheek and reconstruct the facial skin defect) while the first free flap is osteotomized, inset and revascularized by the first reconstructive team. By the time the first free flap is inset and revascularized, the second free flap should be ready for inset and revascularization by the first reconstructive team while the second reconstructive team closes both donor sites.

Osseointegration of dental implants

The bone stock and dimensions of the fibula osteoseptocutaneous free flap are consistently adequate for implant placement.³⁴ Whether performed primarily or secondarily, osseointegration of dental implants involves three distinct stages. At the first stage, the implant fixture is set into the bone and the internal thread is protected with cover screws. The first stage can be performed in select patients as part of the microsurgical mandibular reconstruction (primary osseointegration); in others it can be offered as part of the postoperative rehabilitation program (secondary osseointegration). We have utilized primary osseointegration into

free osseous flaps for many years and have published our outcomes in single and double-barrelled fibula osteoseptocutaneous free flaps.^{11,33,35–38} Double-barrelled fibula osteoseptocutaneous flaps are useful for simultaneously achieving the facial and alveolar process heights for aesthetics as well as dental rehabilitation. Vertical distraction osteogenesis is another method that is used for this purpose, but requires secondary osseointegration instead and we have found that it generally has a higher complication rate.³⁷ At the second stage, the cover screws are removed, healing abutments are screwed into the fixtures, a palatal mucosal graft is secured around the abutments and the graft is protected with a removable stent. At the third stage, the prosthesis is fitted (Figure 26.2).

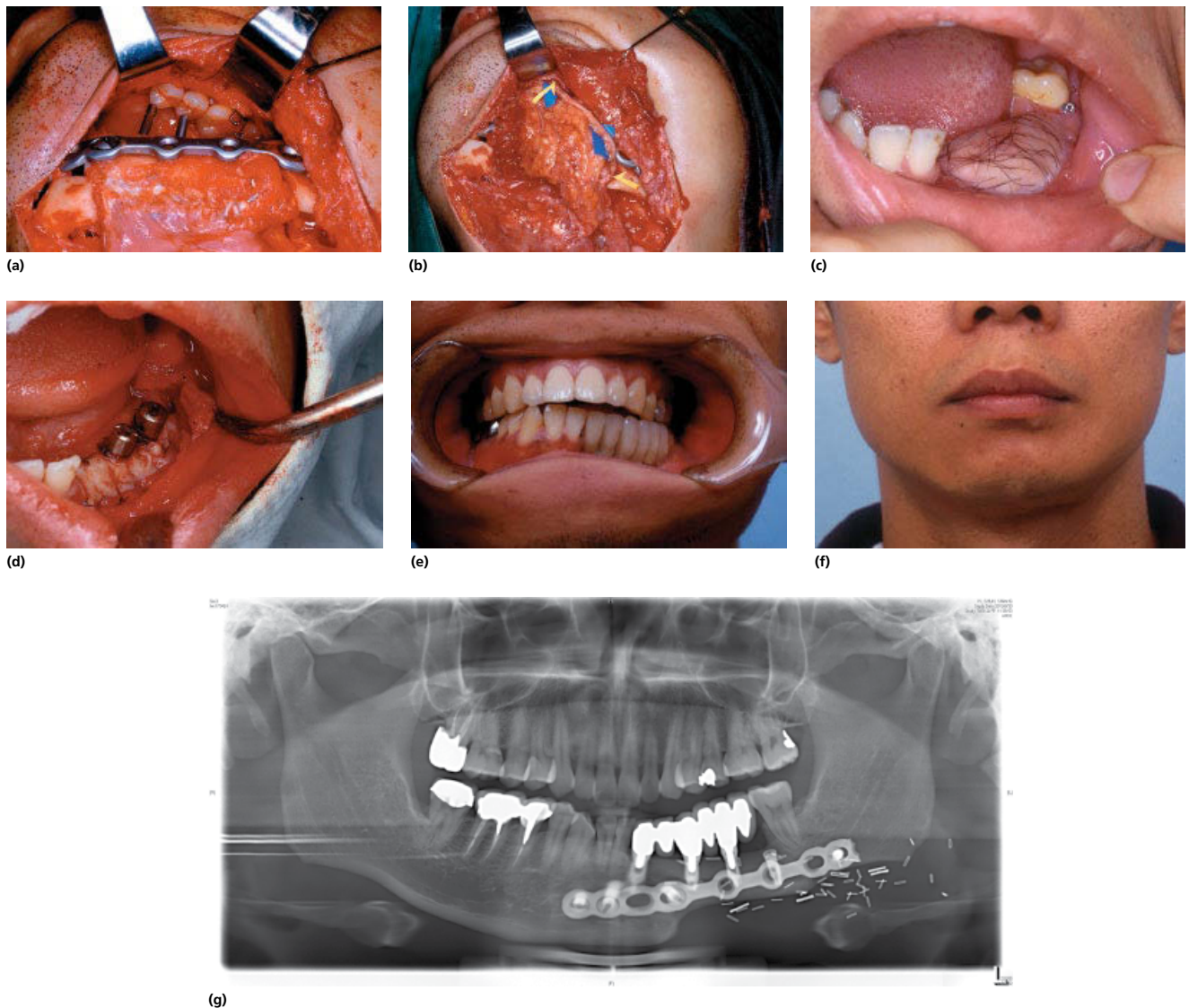


Figure 26.2 Recurrent ameloblastoma of the mandible segmentally resected and reconstructed with a fibula osteoseptocutaneous flap with simultaneous osseointegrated dental implantation for dental rehabilitation and sural nerve graft for restoration of lip and chin sensation. (a) Resultant mandibular defect reconstructed with a fibula osteoseptocutaneous flap and simultaneous osseointegrated dental implantation. Proper occlusion confirmed with guiding screws. (b) Mental nerve defect reconstructed with a sural nerve graft. (c) Appearance after initial reconstruction. (d) Palatal mucosa graft to replace the skin island around the abutments. (e, f) External and occlusal appearance 3 years after surgery. (g) Panorex radiograph 15 years after surgery.

Non-microsurgical techniques

These include grafts, prosthetic materials and pedicled flaps in various combinations and should be mentioned for completeness. They generally offer downgraded reconstructions in comparison to what can be achieved with microsurgical free tissue transfer, but may be appropriate primary reconstruction choices in patients with particularly poor oncological prognoses and/or an array of significant medical comorbidities.¹¹

Non-vascularized bone grafts still have a place in modern segmental mandibular reconstruction for small *isolated bone* defects in biomechanically favourable locations (body or ramus), which have not undergone and will not undergo radiotherapy, which have minimal risk of infection, and are surrounded by healthy, well-vascularized soft tissues. This combination of requirements is rare for non-traumatic segmental defects, but more common for marginal defects, such as those requiring alveolar ridge height augmentations for dental restoration.⁵

The most common prosthetic reconstruction is a substantial bridging mandibular reconstruction plate. This usually would require additional soft tissue coverage, such as with a pectoralis major myocutaneous flap. Alternatively, microvascular free tissue transfer from a location distant to the head, such as from the anterolateral thigh, allows a two-team approach and a total operating time that is similar to using a regional pedicled flap. Nevertheless, whatever method of soft tissue coverage is chosen, plate reconstructions are non-biological and therefore have major disadvantages, including plate/screw fatigue, failure and extrusion, especially amidst infection or radiotherapy.³⁹ Regional vascularized bone can be imported from the ribs, clavicle or scapula, with the pectoralis major, sternocleidomastoid or trapezius flaps, respectively, but have significant limitations in terms of poor bone quality, fragile osseous blood supply, inadequate flexibility in bone inseting, and significant donor site morbidity. Nevertheless, these may provide 'lifeboat' options such as following multiple free flap failures.

Complications and their management

A free flap success rate in excess of 95% for the head and neck region should be the benchmark. But still, despite best efforts, some free flaps will fail. The reader is directed to an in-depth study that addressed the management of the failed free flap in the head and neck region.⁴⁰ Our overall recommendation is that, usually, a failed free flap in the head and neck region should be replaced by another similar free flap. This applies particularly for a failed fibula osteoseptocutaneous free flap used for mandibular reconstruction. Once local wound infection has been controlled and the patient has been reoptimized from the anaesthetic perspective, it is recommended that the contralateral fibula be used for segmental mandibular reconstruction as a second attempt.⁴⁰ Performing a downgraded reconstruction in a candidate who had been deemed suitable for a high-quality

fibula osteoseptocutaneous free flap reconstruction is a mediocre alternative. Interestingly, failed free flaps in the head and neck region that were then managed by regional flaps had a higher subsequent complication rate than those that were replaced by a second free flap. The success rate for a second free flap was 94.1% in this series.⁴⁰

The accumulation of any fluid in the neck, such as saliva, haematoma, abscess or lymph, needs to be dealt with expediently. The wound must be opened, irrigated and debrided as necessary in the operating room to prevent flap failure and still allow timely wound healing so that adjuvant treatments are not delayed. Carotid artery blow-out is a feared complication that mandates healthy coverage of the major neck vessels following any regional reconstruction.

Later complications include metalwork exposure/fatigue/fracture and radiation-induced soft tissue shrinkage/contractures, osteoradionecrosis, osteomyelitis and the various specific complications related to osseointegrated implants, including implant loosening, malpositioning and associated soft tissue problems.

Conclusions

The requirements for a high-quality segmental mandibular reconstruction are clear. The anatomy, including the bone, soft tissues and joints, needs to be restored as closely to its normal state as possible, in the fewest surgical stages and as quickly as possible, in order that musculoskeletal functional, aesthetic, dental and psychosocial impacts are minimal to the patient. In addition, wound healing must be completed rapidly, which means without complications, so that life-saving adjuvant treatments are not delayed. This is a challenge, but when armed with the above techniques and principles, one can provide a high-quality reconstruction for many patients.

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CHAPTER 27

Maxillary reconstruction

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Introduction

Indications

Reconstruction of the maxilla (or midface) is necessary due to defects caused by tumours (malignant or benign), trauma or congenital defects. By far the most common of these are those caused by cancer and hence this chapter will focus on this aetiology.

Comprehensive reconstruction of maxillary defects requires restoration of lost function and form. Functional impairment may involve speech, eating and oral hygiene, but may extend to breathing and vision. Form loss can further alter facial proportions, symmetry and motion. Reconstructions need to take into account which functional losses must be corrected and in what priority, and which alterations in form can and should be corrected. The bones of the midface provide the underlying shape of the face, over which lie the muscles of facial expression and integument that give us each our unique facial appearance. These bones separate the contents of the orbits, nasal cavity, sinus and oral cavities, and provide support for the maxillary dental arch inferiorly, the orbits and sinuses laterally, and the skull base superiorly. In general terms, any reconstruction is aimed at restoring the compromised separations and the lost supports. The reconstructions span the spectrum from prosthetics to free tissue transfers to combinations of these, and therefore can range from relatively simple to extremely complex. Most cases require a 'team' approach. In this chapter we attempt to set out the principles that guide such reconstructions, and to provide some examples.

Historical evolution of maxillary reconstructions

The advent of microsurgery in the late 1970s and early 1980s can be considered a seminal time period in which a quantum leap occurred in our ability to reconstruct the challenging deformities left after maxillectomy. Until this time period, the mainstay of reconstruction in most maxillary defects was the lining of the defects with skin grafts and the use of obturators or some type of maxillary prosthesis. For the smaller defects the obturators

provided separation of the oral and sinonasal cavities and allowed 'normal speech'. The provision of prosthetic teeth in conjunction with the obturators provided the ability to eat. With the small defects, there is role for obturators even today. Obturators may obviate complex reconstructions, a distinct advantage in frail patients. When faced with larger defects, however, the obturators were not as useful, primarily related to poor fitting and loosening.¹

Historically, multistaged flaps² led to pedicled, axial-pattern, fasciocutaneous flaps³⁻⁵ and their application to maxillectomy defects.⁶

The next evolutionary step in the maxillectomy reconstruction was the introduction of the pectoralis major myocutaneous flap.⁷ This, however, had limited use due to the high reach needed and the random nature of the circulation of the distal portion of this flap, and the downward drag of these flaps, causing the reconstruction to change over time.

The use of pedicled flaps in the premicrosurgery era (and in some cases overlapping with it), was not as successful due to the restrictions imposed by the base of the flap, the reach of the flap to its target area and the cosmetic appearance in this anatomically sensitive area.

It is in the massive defects that the introduction of microsurgery was a major advance in the rehabilitation of these patients. The ability to transfer autogenous tissue, including bone and soft tissue, allowed us to reconstruct what is missing.⁸⁻¹³

This chapter attempts to cover the state of the art in maxillary reconstruction. We are cognizant though that with the few recent composite tissue allotransplantations (CTAs) reported to the midface globally we may be at the dawn of a new era in maxillary reconstruction.¹⁴⁻¹⁹ Presently midface CTAs have not been performed in postcancer reconstructions to our knowledge. The few reported cases in trauma situations show amazing results that will definitely dwarf the autogenous tissue microsurgical reconstructions from both the functional and cosmetic point of view. As soon as the issue of 'safe immunosuppression' is achieved it is expected that CTA will supplant any other reconstructive method in the extensive maxillary defects.

It is to be understood that up to now, only lower level evidence exists in the literature in guiding reconstructive surgeons in the selection of appropriate techniques. This chapter is written based on this lower level evidence. Presently, no single cancer centre will have many midface reconstruction cases to allow investigators to produce high-quality level of evidence.

Anatomic considerations

To understand the intricacies of maxillary reconstruction one needs to understand the three-dimensional anatomy of the maxilla. The maxilla has been described as a hexahedron with a box with six walls.^{13,20,21} The roof of this box is formed by the floor of the orbit and its floor is the hard palate and alveolar ridge. Its medial wall is made by the lateral wall of the nasal cavity. The anterior, posterior and lateral walls are made by the vertical and horizontal bony buttresses. These walls contain in their centre the maxillary sinus. The maxillary bones are reinforced by the two horizontal buttresses (inferior orbital rim and the maxillary arch) and the three vertical buttresses (nasomaxillary, zygomaticomaxillary and pterygomaxillary). The muscles of facial expression and mastication minimally overlie the maxilla.

Tumours arising in the midface and maxilla can extend and involve tissues beyond its boundaries. In addition to the maxilla and overlying soft tissues they may include the contiguous areas of the orbit, skull base, nose, mandible and parotid gland.²² Consequently in a maxillectomy procedure contiguous structures that may be resected include: soft palate, lips, nose, cheek and overlying skin, orbital contents, zygoma and pterygoid plates.

Maxillectomy therefore represents a group of diverse defects ranging from a minor oro-antral and/or oronasal defect of the palate to a major defect bounded superiorly by the anterior skull base and inferiorly by the tongue.²³

The morbidity associated with maxillectomy is variable depending on the extent of the resection and can affect deglutition, speech, vision, maintenance of hygiene from debris accumulation and equally important facial disfigurement.

Reconstruction methods

General principles

The three main goals of maxillary reconstruction can be listed as follows:

- separate intracranial, orbital, nasal, and oral spaces;
- restore function; and
- restore a reasonable cosmetic appearance.

The most important goal is to provide safe separation between a contaminated 'nasal space' and the 'clean' anterior cranial fossa. Failure to accomplish this successfully will lead to meningitis and ultimately the death of the patient. There is a need to separate the orbital contents from the wide open sinonasal space.

Under the rubric 'function', there are a number of objectives. The key ones are: (a) to stabilize the floor of the orbit and avoid dystopia with subsequent diplopia; (b) to leave a functional nasal airway for breathing; (c) to separate the oral from the nasal cavities to allow proper eating and intelligible speech; (d) to provide a stable bony maxillary arch to permit acceptance of osseointegrated implants or soft tissue-borne prosthesis for chewing and eating.

The cosmetic appearance will be determined to a great extent by the choice of a suitable flap that takes into consideration the colour match, texture and volume of the autologous tissue transferred. It is in this domain that CTA hopefully will provide the next quantum leap in maxillary reconstruction.

In complex defects the reconstruction may apply to multiple subunits.²⁴ The ranking of reconstruction goals for complex defects can be found in Table 27.1.

Classification systems

Because of the various permutations of defects that can be produced after extirpation of cancer, which may include bone, bone and soft tissues, bone and orbital contents, bone and palate, bone and nasal lining, etc.) different methods of reconstructions are necessary. Over the years a number of surgeons and prosthodontists have introduced various classification systems of these, depending on their experience, technical skills or specialty training, without achieving consensus.^{10,13,22,23,25–34} The classification systems were based on the procedure performed,^{13,22} the tissue loss,^{25,28,29,31} the prosthodontist perspective post surgery^{25,26,31} and the surgical resection.^{10,13,22,27–30,32–34}

As a result of the different perspectives taken in dealing with the maxillectomy defects (i.e. surgical versus prosthodontic) and the failure to address all anatomical issues of the defect, no universal classification has been accepted so far.

In a recent systematic review of the classification systems Bidra *et al.*¹ identified these weaknesses and suggested six criteria, with the acronym of DOC-SAM, that are necessary for universal description of maxillectomy defect (Table 27.2).

These criteria take into consideration the importance of 3D reconstruction of the defects and the future prosthodontist rehabilitation. These criteria are: (a) dental status, (b) oro-antral/nasal communication status, (c) contiguous structure involvement, (d) superior–inferior extent, (e) anterior–posterior extent and (f) medial–lateral extent.

These criteria, if adopted, should definitely improve the communication among healthcare professionals dealing with the maxillectomy defects and add clarity to the reporting of defects and results of reconstruction.

Algorithms for maxillary reconstructions have been published but again these are prone to selection biases based on the authors' subspecialty, interest and experience.^{21,29,35}

As the focus of this chapter is maxillary reconstruction and directed towards plastic surgeons, we selected the Cordeiro classification in this chapter. We are aware that this classification system does not include a localized lateral hard palate resection

Table 27.1 Ranking of reconstruction goals for complex defects

Priority	Subunit	Goal	Solution
1	Upper	Prevent meningitis or brain abscess	Restore lost barrier between sino-nasal complex and anterior cranial fossa
2	Lower	Restore oral functions of eating and speech (in some cases, goal no. 6 is included here)	Replace the lost barrier between sinonasal complex and mouth, either with soft tissue alone, with both soft tissue and bone, or with a prosthesis secured in one of two ways
3	Central	Restore central midface surface contour (with restoration of sensation and motion very secondary goals). In some cases, goals nos 5, 6, or both are best included here	Restore the external soft tissue with tissue of sufficient subcutaneous thickness, and skin with acceptable texture and colour match
4	Upper	Prevent diplopia	Restore support to the orbital contents with a soft tissue layer only (e.g. sling, fascia patch, skin graft) or with antral space obliteration (cf. no. 5)
5	Central	Eliminate the nasal and sinus cavities as a common chamber above an intact or reconstructed palate, to avoid foul-smelling, inaccessible crusting	Replace the lost lateral nasal wall and/or obliterate the antral space with soft tissue
6	Central and lower	Restore anterior projection of the maxillary arch and vertical height of the midface (nasomaxillary, zygomatico-maxillary and pterygo-maxillary buttresses). Decide on goals of dental rehabilitation (none, tissue-borne prosthesis, or osseous integrated implants)	Depending on dental rehabilitation goals, may choose either to restore the lost dimensions with soft tissue only or to replace the anterior or posterior buttresses of the maxillary arch with suitably robust bone. This is accurately positioned, firmly secured, and covered with a skin paddle
7	Upper	Provide shape and support to the orbit and symmetry to the peri-orbita, including canthal ligaments. If orbit exenterated, decide how to deal with the cavity	Restore zygomatic buttress and/or horizontal buttress of the infraorbital rim. Consider whether flap is to obliterate/ cover the orbit or whether a prosthesis is to cover the orbit, if so, should prosthesis be bone-anchored or soft tissue borne? ³³

Source: Archibald *et al.*, 2005.¹² Reproduced with permission from Elsevier.

Table 27.2 Six different criteria (acronym DOC-SAM) and their corollaries that are necessary for universal description of maxillectomy defect

Criterion	Outcome
Dentition	Teeth absent or present; ^a right posterior, right anterior, left anterior, left posterior regions
Oro-antral/nasal communication status	Absent or present
Contiguous structure involvement	Soft palate, lip, cheek, nose, orbital contents, zygoma, pterygoid process or none
Superior–inferior extent	Anterior base of skull level, orbital level, nasal/sinus level, palatal level or alveolar level
Anterior–posterior extent	Right anterior, left anterior, right posterior or left posterior regions
Medial–lateral extent	Isolated, unilateral or bilateral defect

^aAnterior teeth include central incisors, lateral incisors, and canines. Posterior teeth include all premolars and molars.

Source: Bidra *et al.*, 2012.¹ Reproduced with permission of Elsevier.

for what is primarily an oral cancer, yet involves the lower maxillary infrastructure. We are also aware that the classification system does not include central or bilateral resections. Nevertheless although this system does not meet all of Bidra's criteria, until a better system is introduced it will suffice. In any case it is the most widely used as it is based on a relatively large volume of cases and it encompasses the clinically important types of

defects that we are likely to encounter in our surgical practices. In addition the authors provide an accompanying algorithm for flap selection per type of defect.²¹ This classification system is shown in Figure 27.1. It includes type I, which is a limited maxillectomy in which the anterior and medial walls of the maxilla with associated skin and soft tissues are resected; type II, which is a subtotal maxillectomy defect in which the lower five walls of the maxilla including the palate are resected; type IIIa, in which all six walls of the maxilla, including the floor of the orbit and hard palate, are resected; type IIIb, in which all six walls of the maxilla, including the floor of the orbit and orbital contents, are resected; and type IV which is an orbitomaxillary defect. This is the same as type IIIb with the exception that the palate remains intact.

The Cordeiro algorithm is shown in Figure 27.2. This algorithm shows the choice of only two free flaps for the reconstruction of these defects. Although in general we agree with this simplification¹³ (Cordeiro *et al.* only use two free flaps; radial forearm or free rectus abdominis), there are occasions where other free flaps may need to be chosen. Archibald *et al.*¹² provided the ranking of various free flaps chosen for midfacial reconstruction by their reconstructive microsurgeons in their institution (Table 27.3).

With careful consideration of the missing tissues and their meticulous reconstruction one can attain reasonable functional results. In a large series of 246 maxillary reconstructions, 95% of patients achieved greater than 80% speech intelligibility (with some of them requiring obturation in addition to free flap) and 70.8% were able to feed themselves with unrestricted diet.³⁵

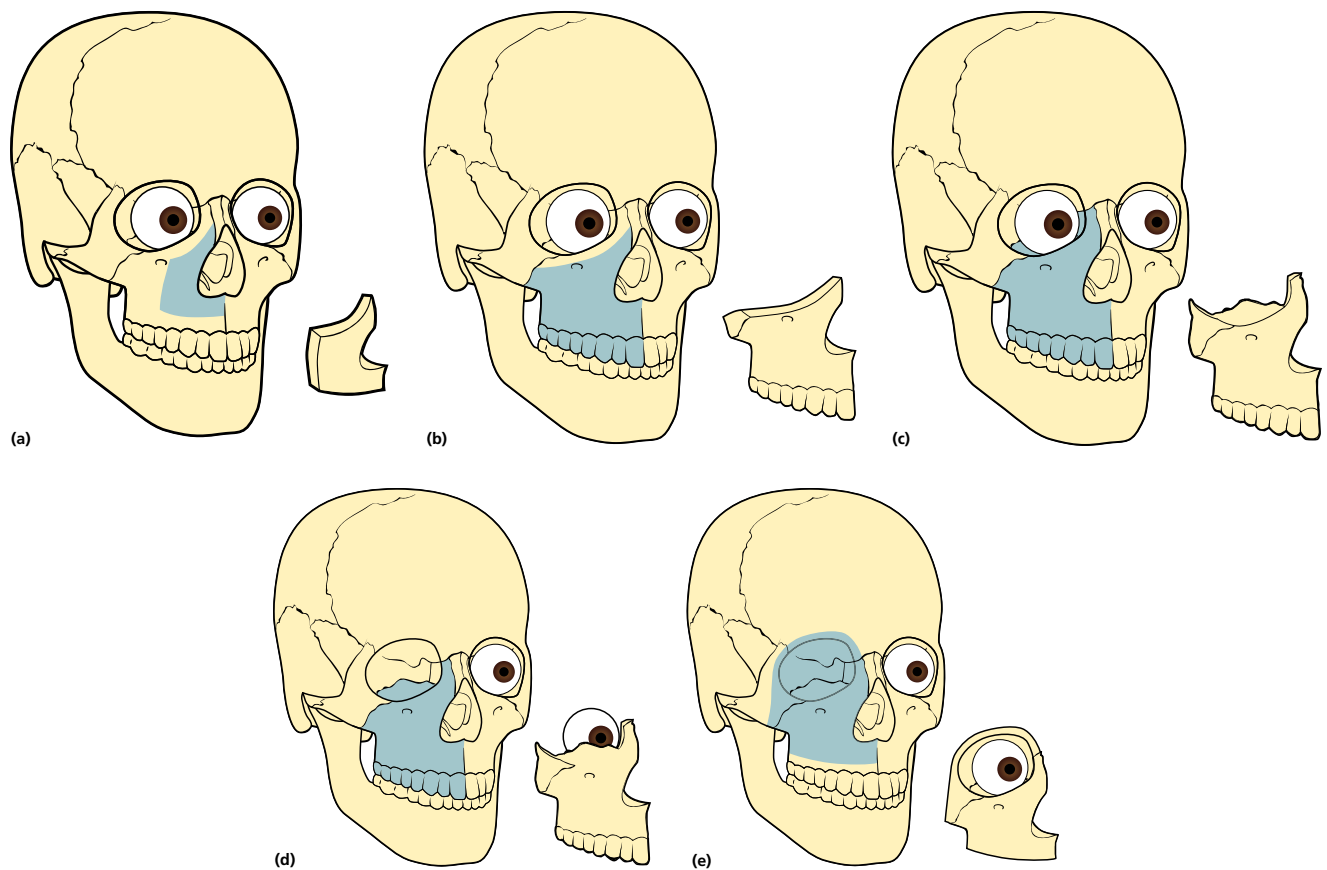


Figure 27.1 Corderio classification system for the maxillectomy defect. (a) Type I, (b) type II, (c) type IIIa, (d) type IIIb, (e) type IV.

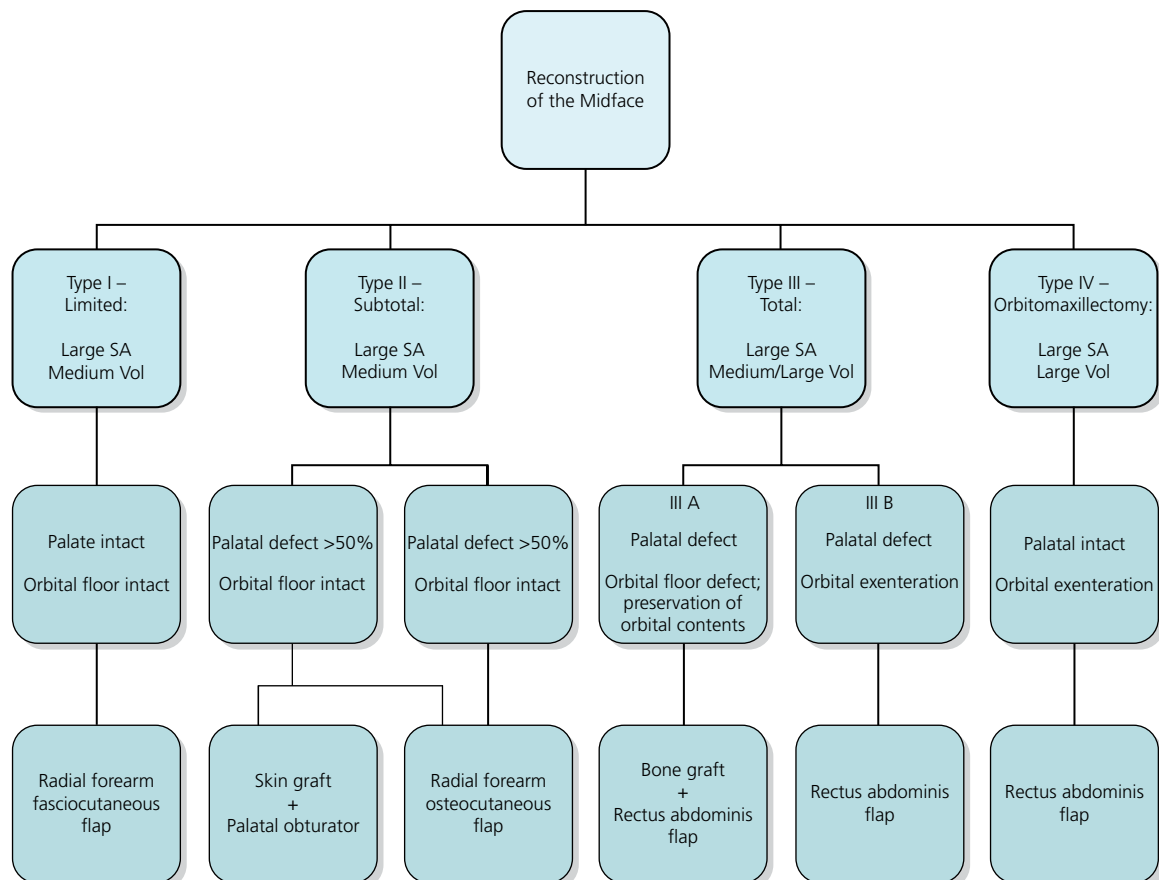


Figure 27.2 Diagrammatic representation of algorithm for reconstruction of the midface. SA, surface area. Source: McCarthy and Cordeiro, 2010.²¹ Reproduced with permission of Lippincott Williams & Wilkins.

Table 27.3 Characteristics of free flaps used in midface reconstruction

Part	Characteristic	RFFF ^{37,38}	Lateral arm	Scapula ³⁹⁻⁴¹	Fibula ⁴²⁻⁴⁴	Rectus ⁴⁵	Iliac crest ^{46,47}	Latissimus dorsi ⁴⁸	Anterior lateral thigh ¹⁶
Skin	Size adequacy	3.0	2.3	4.0	2.3	4.0	3.5 (2)	4.0	3.0 (2)
	No. of paddles	2.3	1.7	4.0	1.7	3.7	2.0 (2)	4.0	2.0 (2)
	Colour match ^a	1.7	2.7	3.7	1.7	2.0	1.0 (2)	2.7	1.0 (2)
	Texture ^a	2.3	3.0	3.7	2.3	2.3	1.5 (2)	2.7	3.0 (2)
	Pliability	3.3	3.3	2.7	3.3	2.7	2.5 (2)	3.0	3.0 (2)
	Reliability	4.0	3.0	3.7	2.0	3.3	3.0 (2)	3.7	3.0 (2)
Subcut tissue (thicker = better)	Neurotization	4.0	3.5 (2)	1.0	3.0 (2)	1.0	1.0 (2)	1.0 (2)	1.5 (2)
	Thickness/pliability	2.0	3.0	3.3	3.0	2.3	2.5 (2)	2.3	2.5 (2)
Skin–bone mobility	Translation/rotation	2.5 (2)	2.5 (2)	4.0 (2)	2.5 (2)	N/A	2.5 (2)	N/A	N/A
Bone	Length	2.7	2.5 (2)	2.7	4.0	N/A	2.5 (2)	N/A	N/A
	Thickness	2.0	1.5 (2)	2.0	3.7	N/A	4.0 (2)	N/A	N/A
	Osteotomization	3.0	1.5 (2)	2.0 (2)	4.0	N/A	3.0 (3)	N/A	N/A
	Reliability	3.3	2.0 (2)	3.0 (2)	4.0	N/A	4.0 (2)	N/A	N/A
	Osseointegration	1.7	1.0 (2)	1.5 (2)	3.7	N/A	4.0 (2)	N/A	N/A
Pedicle	Length	4.0	1.7	3.3	2.7	3.0	2.0 (2)	3.3	2.0 (2)
	Vessel quality	4.0	1.5	3.3	3.0 (2)	3.0 (2)	2.0 (1)	3.5 (2)	1.0 (1)
Donor site	Cosmesis	1.3	3.3	3.0	3.0	3.7	3.5 (2)	3.0	2.5 (2)
	Morbidity	3.0	4.0	3.0	3.3	1.7	1.0 (2)	3.3	3.5 (2)
	Risk of complications	2.7	3.7	3.3	3.3	2.0	1.5 (2)	3.3	3.5 (2)

Values are the means of the scores of the microsurgeons at the authors' centre. Where less than three of the surgeons recorded a value, the number reporting is given in parentheses. 1 = lowest/worst rating; 4 = highest/best rating.

N/A, not applicable; RFFF, radial free forearm flap.

^aWith reference to use on the face.

Source: Archibald *et al.*, 2005.¹² Reproduced with permission from Elsevier.

Complications and management

There are many complications that can occur in the course of maxillary reconstruction. In general terms they can be classified into medical or surgical complications.

As the oncologic patients are in general elderly they have associated medical problems that can complicate their recovery. Large recent studies have found as many as 37.8% of patients suffered some perioperative complication after maxillectomy reconstruction.³⁵ Consequently, it is important that a patient's medical condition is optimized prior to surgery and followed carefully post operatively. Consultations from internists, anaesthesia or other subspecialists may be important in both preoperative and postoperative period.

Of the surgery-related complications, free flap failure is among the most dreaded. In a recent large series total flap failure was reported to be 3.3%.³⁵ As free flap failure is a serious complication, every effort should be made to achieve success. This is a case where the most expert microsurgeon should perform the microvascular anastomoses to ensure vessel patency. When the reconstructive plan is first developed, a back-up flap should be identified in the unlikely event of total free flap failure. During surgery care should be taken to protect the donor site of the back-up flap.

Fistula (oronasal, oro-antral, orocutaneous, nasocutaneous) is another potential complication. A rate of 7.5% has been reported in the largest series of maxillary reconstructions

(*N* = 246) reported recently.³⁵ If a flap has been used for the maxillectomy defect, and tissue redundancy exists from the primary flap, this tissue can be used to close the fistula.

Midface projection can be lost if traditional bone graft has been used and this was not adequately covered with vascularized tissue. This problem can be avoided by using vascularized bone graft with robust circulation. One must ensure that the maxillary arch is established by using templates and the proper 'carpentry' of the vascularized bone graft intraoperatively. Recent innovations with computer-assisted facial transplantation may help minimize this problem in the future.²⁹

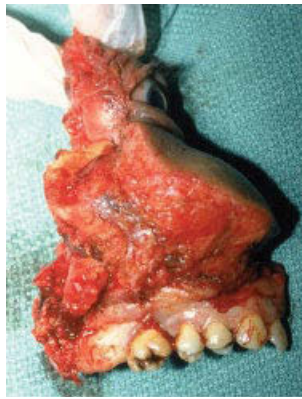
Malocclusion can be a problem if the maxillary arch is not properly aligned. Preplating of the maxilla before the tumour resection, if possible, should be contemplated to maintain the 3D spatial orientation of the future bone graft.

CSF leak can be a serious complication, leading to meningitis. This is reported to occur in 1.2% of cases,³⁵ and is more likely to occur if the defects include the skull base. In such cases a neurosurgeon should be part of the team from the beginning to ensure dural seal. From the reconstructive point of view, the dural seal can be reinforced from below by choosing a myocutaneous flap (e.g. rectus abdominis). Muscle flaps have the added advantage of sealing irregular surfaces and deliver antibiotics to critical areas such as the skull base.

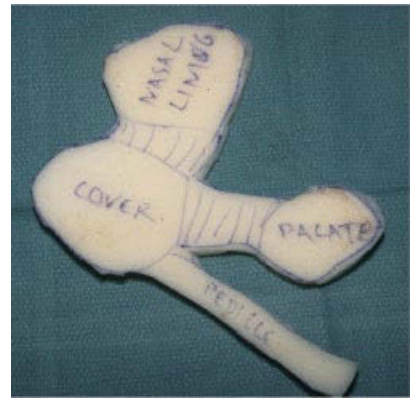
Diplopia usually is the result of failure to adequately reconstruct the floor of the orbit. Non-vascularized bone grafts can be



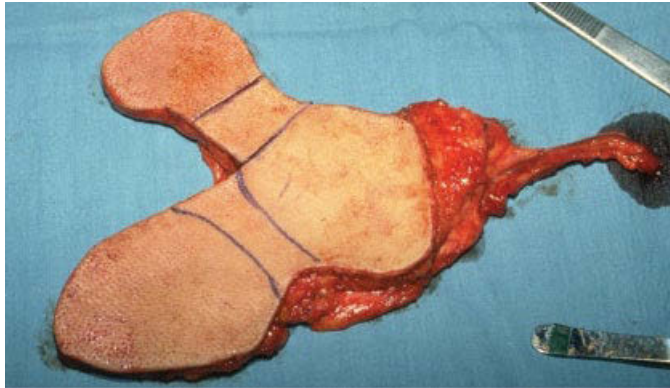
(a)



(b)



(c)



(d)



(e)



(f)

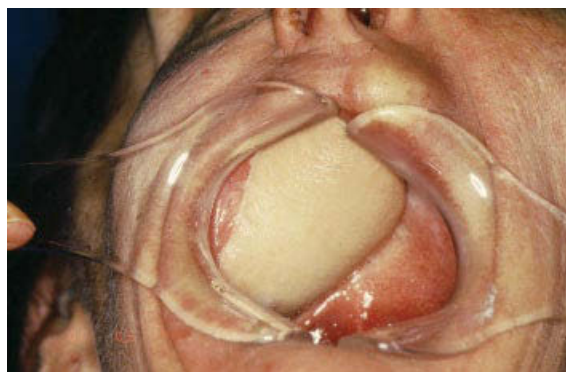
Figure 27.3 Case 1: A 39-year-old female with squamous cell carcinoma. See text for details.



(a)



(b)



(c)



(d)

Figure 27.4 Case 2: A 68-year-old female with poorly differentiated squamous cell carcinoma of the right maxilla. See text for details.

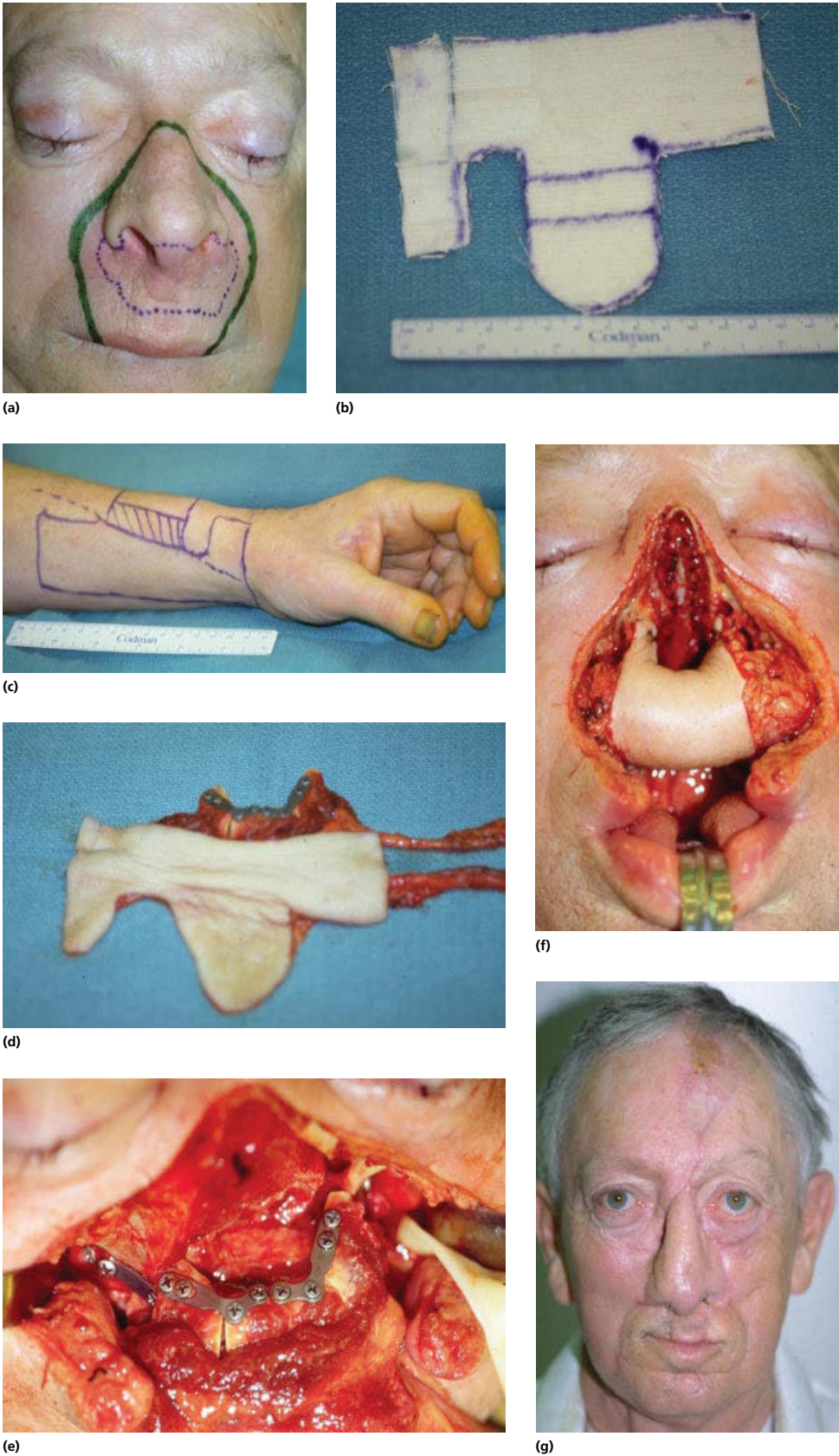
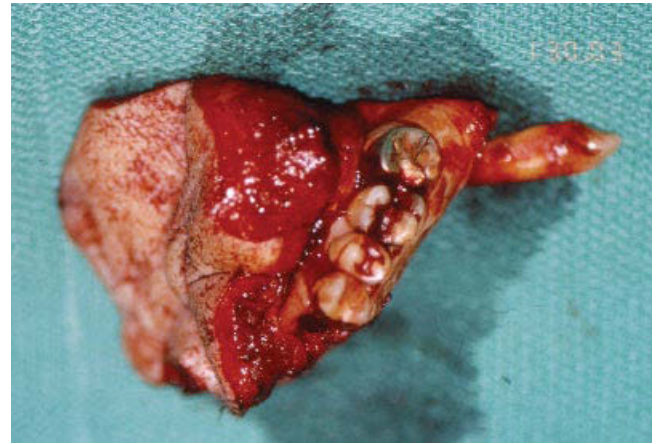


Figure 27.5 Case 3: A 63-year-old male with squamous cell carcinoma. See text for details.



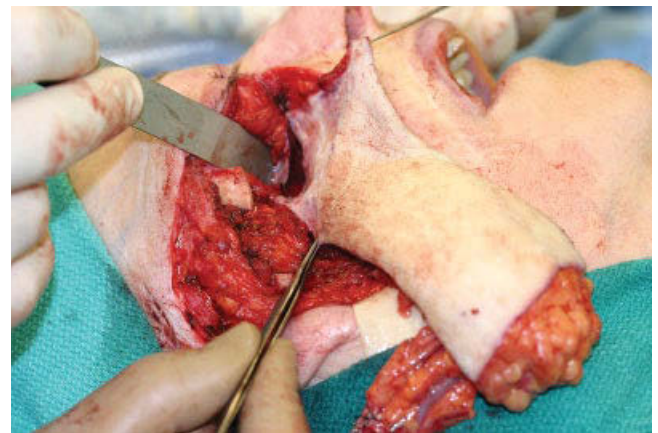
(a)



(b)



(c)



(d)



(e)



(f)

Figure 27.6 Case 4: A 71-year-old woman with recurrent basal cell carcinoma. See text for details.

used provided vascularized muscle is introduced to cover the bone. Titanium mesh can also be used. The rate of diplopia/dystopia as a complication of reconstruction was found to be the same for those patients who had bone or titanium mesh recon-

struction at 1.7%.³⁵ Alternatively, a vascularized bone graft can be used for this purpose.

Social isolation and seclusion can be another serious problem if the reconstruction is aesthetically inadequate. Attention to

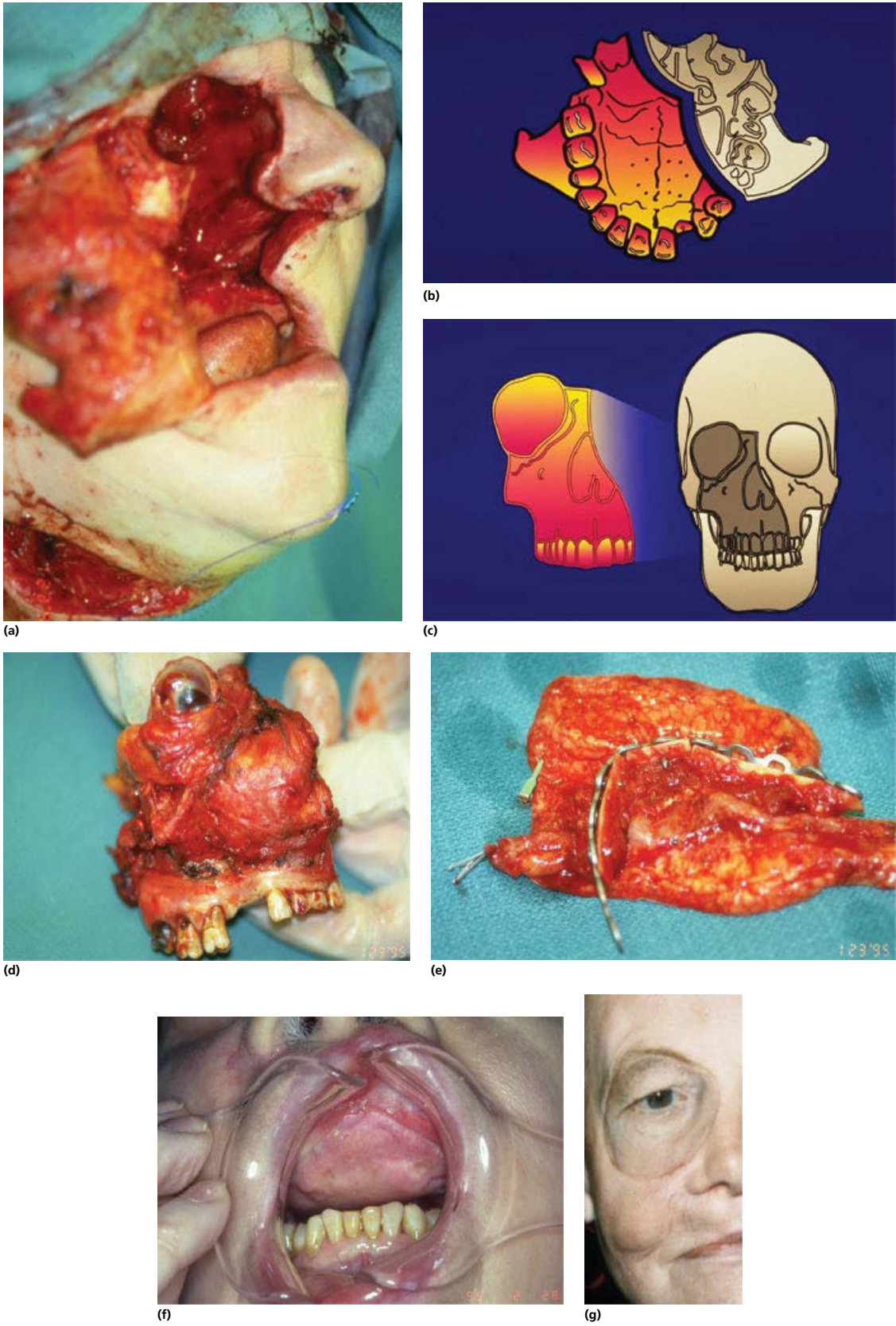


Figure 27.7 Case 5: A 74-year-old female patient with adenoid cystic carcinoma of the right maxilla. See text for details.

details and proper selection of flaps can go a long way to preventing this problem. Patients should be alerted to the need of future revisions.

Case studies

Below we show some cases of maxillary reconstruction from our centre.

Case 1

A 39-year-old female presented with squamous cell carcinoma (Figure 27.3a) of the maxillary sinus which did not respond to radiotherapy. Radiation changes are evident on facial skin over the right maxilla. Following right maxillectomy and orbital extenteration (Figure 27.3b) (Cordeiro defect IIIb) she was reconstructed with a three-panel scapular flap to provide separation of two cavities (i.e. nasal, oronasal) and to provide coverage for the orbital defect (Figure 27.3c,d). Figure 27.3e shows final result. The bulk and stiffness of the flap eliminated the dead space of the maxillectomy defect. Figure 27.3f shows the intraoral panel of the scapular flap.

Case 2

A 68-year-old female with poorly differentiated squamous cell carcinoma of the right maxilla extending to overlying skin, orbit and oral cavity (Figure 27.4a). After bi-frontal craniotomy and supraorbital exploration, right maxillectomy and orbital exenteration was performed (Cordeiro IIIb defect) (Figure 27.4b). Duroplasty was performed with temporal fascia. Reconstruction was performed with a rectus abdominis myocutaneous flap from which two separate skin panels were used: one to separate the sinonasal cavity from the oral cavity (Figure 27.4c) and the second to provide external coverage to the facial orbital defect (Figure 27.4d). The bulk of the muscle provided dural seal superiorly.

Case 3

A 63-year-old male with squamous cell carcinoma required resection of the maxillary alveolus, portion of the hard palate and nasal floor, complete rhinectomy (Cordeiro defect II) (Figure 27.5a). Figure 27.5b shows a template of the defect and Figure 27.5c the outline of the template on the forearm. The hatched area was eventually de-epithelialized to separate the oral from the nasal panels of the flap. Figure 27.5d shows the doubly osteotomized radial forearm osteocutaneous flap to reconstruct the alveolus. The alveolus was reconstructed using mini plates (Figure 27.5e) and the forearm skin wrapped around the bone graft to reconstruct the floor of the nose and palate (Figure 27.5f). The lip defect was reconstructed with twin Karapandzic flaps (not shown here). The nasal defect was reconstructed with a forehead flap at the same operation. Figure 27.5g shows the final result of this difficult defect.

Case 4

A 71-year-old woman with recurrent basal cell carcinoma required partial right maxillectomy (Cordeiro defect II). Reconstruction was achieved with a three-panel scapular fasciocutaneous flap (Figure 27.6a,b). One panel was used for the lateral nasal wall (Figure 27.6c, d showing inseting), a second panel was used for the intraoral defect (Figure 27.6e) and a third panel was used for the external coverage (Figure 27.6f). No bony reconstruction was used in this case.

Case 5

A 74-year-old female patient with adenoid cystic carcinoma of the right maxilla required orbitomaxillectomy proceeded by craniotomy to establish the extent of the tumour superiorly (Cordeiro defect IIIb). Figure 27.7a shows the defect immediately after the resection. Figures 27.7b and c show the anterior-posterior and vertical extent of resection, respectively. Figure 27.7d shows the resected specimen. Reconstruction was achieved with a radial forearm osteocutaneous flap (Figure 27.7e). Figure 27.7f shows the alveolar and palatal reconstruction. Figure 27.7g shows the final result with external eye prosthesis.

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CHAPTER 28

Pharyngeal reconstruction

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Introduction

The upper aerodigestive (UAD) tract is an intricate maze of alleyways, muscular tubes and cords that allows humans to have a common functional pathway for oral intake, breathing, as well as harmonizing vibrations into speech. Articulation of speech is dependent on the symphony of functions of these passages, influenced by the larynx, epiglottis, pharyngeal walls, soft and hard palate, the tongue musculature, salivation, lip seal or oral competence and dentition as a tightly ordered orchestra. Many of these functional elements involve the pharynx.

The posterior reaches and ramifications of the UAD tract coalesce into a continuous 15-cm-long muscular collecting tube, the pharynx, which begins in continuity with the nasopharynx superiorly and connects to the oesophagus inferiorly. It extends from the skull base to the oesophagus and comprises three parts: upper or nasopharynx behind the nose, middle or oropharynx behind the mouth, and lower or hypo- or laryngopharynx behind the epiglottis and larynx.

Pathology

Pharyngeal cancers are predominately squamous cell carcinoma (SCC) and are of two types: HPV+ related to human papilloma virus (better prognosis) and HPV– usually linked to alcohol and tobacco. Risk factors include alcohol and tobacco, exposure to Epstein–Barr virus and Chinese ethnicity, especially for nasopharyngeal carcinoma. Organ preservation has become the main aim in the management of UAD tract malignancy. Fortunately, early stage disease can be effectively treated conservatively with radiotherapy or chemo-radiation combination so that function can largely be preserved. Given the morbidity of surgery in this most sophisticated functional zone, its place is increasingly

reserved for advanced stage disease or in salvage treatment after failure of non-surgical protocols. Sadly, most patients present at advanced stages and are associated with poor overall survival. Between 70 and 85% have stage 3 or stage 4 disease at presentation and 5-year survivals are reported around 15–35%.^{1,2} The reality is that most surgery is palliative and although the prime focus here is inevitably on removal of the cancer and reduction in the risk of local recurrence, the overall picture must be on the quality of survival. By contrast, nasopharyngeal cancers have a better prognosis as they are mostly responsive to radiotherapy and rarely require surgical resection or reconstruction.

Anatomy

The nasopharynx is open to the nasal cavities anteriorly and extends from the base of the skull superiorly to the upper surface of the soft palate where it becomes the oropharynx. The oropharynx communicates with the mouth anteriorly and includes the soft palate, the tonsils and the tongue base posterior to the circumvallate papillae. The oropharyngeal tube extends from the soft palate above to the hyoid below. The hypopharynx is continuous below the oropharynx, extending from the level of the hyoid bone to the oesophageal inlet. The larynx lies in close anterior proximity. The thyrohyoid membrane through which the internal branch of the superior laryngeal nerve gains access to the larynx is located supero-laterally. The detailed anatomy of the UAD tract can be found in specialized texts. Anatomically, the area has a rich lymphatic drainage to the jugular chains as well as the retropharyngeal nodes and the node of Rouvière^{1,3} with frequent crossing of the midline. The pharyngeal muscular tube, which spans from the base of the skull to the C6 vertebra, comprises circular fibres that form constrictions to propel contents into the oesophagus and longitudinal fibres that lift the walls of the pharynx during swallowing.

Indications for surgery and reconstruction

Most commonly, major UAD tract defects result from surgical ablation of malignancies. It is accepted that X-ray therapy is just as effective as surgery for eradication of early disease, and because X-ray therapy is less destructive to function, it is the preferred modality of treatment. Surgery is therefore reserved for advanced stage or recurrent cancers and will be inevitably destructive. The principles of wide oncological resection, the proximity of the vital structures and the protection of the upper airway patency, speech and deglutition dictate the indications for reconstruction of any significant UAD tract defects. Many pharyngectomies for advanced disease are associated with combined laryngectomy. Thus most advanced neoplasms involving the oropharynx, hypopharynx or cervical oesophagus will result in a pharyngeal defect requiring reconstruction.

Classification of pharyngeal defects

A useful classification of pharyngeal defects was devised by Disa *et al.*⁴

- Type I: Partial defects involving less than 50% of the pharyngeal circumference.
- Type II: Circumferential defects or those greater than 50% of pharyngeal circumference.
- Type III: Extensive non-circumferential defects involving multiple other anatomical sites

As with all classifications, it has shortcomings. It does not distinguish whether partial defects involve the larynx or base of tongue. It also assumes that any defect greater than 50% of the circumference should be considered a circumferential defect and this has implications for the algorithm-directed surgeon.

Historical perspectives

The first attempts at pharyngo-oesophageal reconstruction date back to the nineteenth century. These proved notoriously unreliable, with chronic aspiration, haemorrhage and early death in the postoperative course followed by stricture formation in those who survived the immediate aftermath of the surgery. This led to a loss of enthusiasm for reconstruction until Wookey rekindled interest in 1942 with a random pattern anterior neck skin flap to re-establish the UAD tract.¹ His two-stage reconstruction included using the cervical skin raised as a random flap sutured to the upper and lower ends of the defect, but left open anteriorly thereby exteriorizing the pharynx and oesophagus. The sulcus thus created was closed five weeks later at a second-stage operation. The next surgical leap came when Bakamjian described the more reliable axial deltopectoral flap, based medially on what has subsequently been shown to be the upper 2nd and 3rd internal thoracic artery perforating branches.⁵ It was, however, still a two- or three-stage treatment and was not uniformly reliable.

In 1979, Ariyan's description of a tubed myocutaneous pectoralis major flap finally made possible a single-stage ablative reconstruction with a more predictable outcome.⁶ Subsequently, both the trapezius and latissimus dorsi myocutaneous flaps were described as alternatives for a single-stage reconstruction, but not surprisingly did not achieve significant popularity.^{2,3}

Parallel with these skin tube developments, more reliable pedicled interposition gastrointestinal reconstructions were being attempted as far back as the 1930s, their primary role, however, was in total oesophagectomies. Adaptation of the Ivor-Lewis gastric pull-up to the cervical oesophagus and pharynx was one such procedure, which has survived as a mostly reliable option into the modern era. In addition to the gastric pull-up, the spectrum of these pedicled flap reconstructions included the left colon and the seldom used pedicled jejunum.^{7–10}

In 1959, Seidenberg¹¹ reported the first successful harvesting and transferring of a jejunal free flap using microsurgical techniques, reportedly with threaded 6-0 silk sutures under loupes magnification. His patient died 5 days postoperatively from a massive cerebrovascular accident, but the first successful human free flap was found to be viable at the time. The following year Hiebert and Cummings¹² performed an uneventful gastro-antral free graft joining 2-mm-diameter gastro-epiploic vessels to the superior thyroid artery end to side and facial vein end to end. They noted the report of Jacobson and Suarez, the acknowledged founders of microvascular technique that occurred subsequent to their procedure, recommending using a microscope. These feats were performed at least a decade before the tidal wave of microsurgery began, with the work of Buncke *et al.*,¹³ Daniel and Taylor,¹⁴ O'Brien *et al.*¹⁵ and others who used other tissues, including omentum and skin. Soon, microsurgical free tissue transfer was to become a first-line mainstream reconstructive option.

Microvascular technique brought with it the promise of a reconstruction not bound by restraints of pedicle length or random proportions. The reliability of an axial flap transfer into the defect revolutionized the approach to major reconstructive challenges. Gastro-omental, gastric antral, ileocolic as well as colon free flaps have been described; however, by far the most common visceral reconstruction is a free jejunal reconstruction.^{12,16–25}

The earliest significant clinical series of microsurgical jejunal reconstructions were those reported by Jurkiewicz.^{26,27} But his daring and technical skill were not widely adopted for almost a further 20 years. The free jejunal flap, whether as a tube or patch, has since become one of the two most reliable reconstructive tools for laryngo-pharyngectomy and/or cervical oesophagus reconstruction.²⁸

Tubed or patch fasciocutaneous flaps are the other main alternative reconstructive options for pharyngeal defects. The most common flaps used are the anterolateral thigh and radial forearm flaps.^{29–33}

Assessment and planning

As with any operative surgery, planning remains the most important step in execution of the technique. This is best considered as part of a regular multidisciplinary meeting, which includes, in addition to head and neck, oral maxillofacial and plastic surgeons, radiation oncologists, medical oncologists, pathologists, speech therapists, dieticians, physiotherapists, social workers as well as a radiologist specializing in head and neck malignancies. An estimation of the extent of the defect and the need for reconstruction is discussed at these meetings. The reconstructive team, in conjunction with the ablative team, can decide on the best reconstructive option and begin planning for surgery after assessment of all possible donor sites. Previous history of laparotomy and upper GI surgery is noted and the patient's forearms and thighs are assessed.

A decision is also made at these multidisciplinary meetings in relation to the need for peri-operative radiotherapy and preoperative dental surgery, as well as nutritional needs. Most, if not all patients requiring major reconstruction have either advanced disease or chemo-radiation failure for relatively advanced disease and suffer from malnutrition. The need for nutritional supplementation is almost universal, including during the radiation therapy period. This may require percutaneous endoscopic gastric (PEG) tube insertion, depending on the surgical unit. Nutritional status will influence wound healing and reduce fistula formation. Uncomplicated healing streamlines the programming of radiation therapy in the shortest time window possible for the best therapeutic outcome.

Reconstruction

The broad principles of pharyngeal reconstruction are:

- maintaining or recreating a patent airway and protection from aspiration;
- re-establishing the integrity of the pharyngeal pathway to allow swallowing and adequate caloric intake; and
- voice and speech rehabilitation.

All of these endeavours have in common the creation of a patent tube. Salvage of all these functions is possible to various extents. The advent of microsurgery has made possible transfer of large amounts of tissue sculpted to more or less reconstruct the created defect. In addition, introduction of tissues with a new vascular supply into settings of advanced disease requiring major resections and radiotherapy or following chemo-radiation failure, promotes better wound healing and discourages fistula formation.

The two key determinants that influence the nature of the reconstruction are: the *surgery* (i.e. the defect created as well as donor availabilities) and the *patient's general status*. Many of these patients have life long history of heavy smoking and drinking, with the attendant risk of cardiac or respiratory disease and malnourishment. These are all factors to be considered in the light of the length of surgery and the added morbidity

from choice of donor sites. The added burden of a laparotomy or a chest flap on the respiratory reserves, the speed of skin flaps versus bowel in patients with poor cardiac reserve or malnourishment and the benefits of donor sites that allow simultaneous access during the primary resection for two-team surgery are issues to ponder. Preoperative anaesthetic, respiratory and cardiac assessment is mandatory. Any reasonable optimization of the patient's status should be undertaken prior to surgery.

All patients require confirmation of continuity and integrity of the UAD tract at 7–10 days postoperatively, with a contrast study before removal of nasogastric tubes and resumption of oral intake.

Reconstructive options

Local flaps

In practical terms, these include surrounding cervical skin and soft tissue and they rarely provide a practical reconstructive option for major defects. Not only is the neck skin vasculature unreliable, especially after flap elevation and block dissection, but it is generally sacrosanct to ensure cover of vital structures such as the carotid vessels. Frequently the neck skin has been or will be irradiated. The generalized devascularization associated with the treatment modalities greatly favours the importation of new, independently vascularized tissue, either from a regional source or more commonly now as a free flap transfer. Recently, the supraclavicular flap based on the transverse cervical vessels including a deltoid skin extension has been successfully used in lower level pharyngeal reconstructions.

Regional flaps

Pectoralis major

This type V Mathes and Nahai myocutaneous flap remains one of the most reliable reconstructive options for the head and neck.⁴ With the advent of microsurgical reconstructions it has been increasingly relegated to a second choice or salvage reconstructive option. Some of the current indications for its use include:

- situations where general patient state and systemic disease dictate reduced operative time;
- in combination with another flap or alone, where an external skin defect is present or has evolved after primary surgery;
- in coverage and protection of major neck vessels;
- in closure of pharyngostome; and
- wound dehiscence.

This flap is based on the pectoral branch of the acromiothoracic axis. It is most commonly raised as a myocutaneous flap with its skin island placed medially in women to reduce breast tissue inclusion. The skin component is rolled to form a tube to reconstruct circumferential defects. In order to avoid traction on the vascular pedicle a 3 cm cuff of muscle should be retained at the clavicular origin where possible. A so-called defensive incision for accessing the muscle without violating the potential delto-pectoral

salvage flap is made and the flap is raised and, where possible, tunnelled through the neck into the defect. For partial defects the skin island can be designed as a patch and sutured into the remnant pharyngeal mucosa.

Anchoring the remnant pharynx and cervical oesophagus to the pre-vertebral fascia, interdigitating the lower flap inset with the oesophagus and de-epithelization of the leading skin edge have also been described as means of reducing stricture and fistula formation. Others have advocated removal of the skin island and using only the myofascial component of the flap for repair to reduce bulk and to discard the least vascularized tissue component.

Complications

The reduced operative time, avoidance of the complications and morbidity related to abdominal surgery as well as inherent risks with microsurgical flap procedures are often cited as clinical grounds for use of the pectoralis major flap as a primary reconstructive option. Koh *et al.* reported a comparison of the average operating times for the pectoralis major, 76 +7 min (54–105 min), and the free flap, 145 +11 min (115–205 min), components of reconstruction cases.³⁴

Complication risks for flaps in head and neck reconstruction overall include:^{35–42}

- total flap necrosis (0–2.7%)
- partial flap necrosis (4–29%)
- pharyngocutaneous fistula formation (5.5–29%)
- stricture formation (0–27.2%)
- wound dehiscence (2.8–26%)
- infection (2–24%)
- bleeding (1–7%).

Table 28.1 lists published outcome studies specific to pharyngeal reconstructions. Increased flap complication rates are reported

with hypopharyngeal reconstructions, female patients, previous radiation therapy, obesity, patients >70 years old and preoperative malnutrition (albumin <4.0 g/dL).^{37,39,43}

Flap bulk often precludes meaningful voice rehabilitation using a tracheoesophageal puncture (TEP). In addition, recurrent descent and obstruction of the stoma may require surgical revisions.

Miscellaneous

Other regional pedicled flaps described include gastric pull-up flap, the myocutaneous latissimus dorsi and trapezius flaps, as well as the traditional fasciocutaneous deltopectoral or its modification, the internal mammary artery perforator (IMAP) flap. This is essentially a paramedian vertically oriented propeller flap based on the 2nd or 2nd/3rd internal mammary perforators. It can be rotated 180° and employed for some hypopharyngeal reconstructions.

Free flaps

The most common arterial recipient vessel is the superior thyroid or facial and the most common veins are the internal and external jugular or facial veins. Free flap reconstructions are broadly divided into two groups: the *visceral free flaps*, the commonest being the jejunal transfer, and *fasciocutaneous free flaps*, from the radial forearm and anterolateral thigh.

Free jejunum

For obvious reasons, jejunum is an ideal reconstructive option for pharyngeal and upper oesophageal reconstructions. Previous laparotomy, although not absolute, is a relative contraindication to its use. Patients with minimal respiratory reserve should be considered with caution, given

Table 28.1 Pectoralis major and other myocutaneous flaps

	Defect	No. pts	Successful swallowing (%)	Stricture (%)	Fistula leak (%)	Flap failure (%)	Medical complication (%)	Immediate mortality (%)	Pts who received voice prosthesis (%)	Functional outcome (%)
Pectoralis major										
Chan <i>et al.</i> ⁴⁴	Pharynx (circumferential)	92	100	27.2	23.9	–	–	–	–	35.8
Clark <i>et al.</i> ⁴⁵	Hypopharynx	68	81	12	35	–	15	3	44	–
Chan <i>et al.</i> ⁴⁶	Hypopharynx	30	100	26.7	0	–	–	–	–	100
Espitalier <i>et al.</i> ³⁵	Hypopharynx	41	82	39	22	2.5	–	0	2	–
	Cervical Oesophagus									
Spriano <i>et al.</i> ⁴⁸	Hypopharynx	37	100	0	13	0	10	3	24	100
Saussez <i>et al.</i> ⁴⁹	Hypopharynx	12	100	17	33	0	0	0	50	83
Pectoralis major + trapezius + sternocleidomastoid										
Carlson <i>et al.</i> ⁴⁷	Cervical Oesophagus Hypopharynx	45	60	27	78	2	0	0	–	–
Range			60–100	0–39	0–78	0–2.5	0–15	0–3	24–50	35.8–100

the additional morbidity of a laparotomy. A segment of proximal jejunum up to 25 cm length can be transferred as a free flap. The luminal diameter of the jejunal flap ranges between 3 and 5 cm. Despite its advantages, this flap has several technical shortcomings:

- It is exquisitely sensitive to hypoxia, any hypoxia beyond 3 h will almost always result in flap death and subsequent potentially catastrophic fistula formations.^{50,51}
- The vein is particularly thin and will not tolerate traction.
- The pedicle remains relatively short in comparison to its fasciocutaneous counterparts; the average length is described as 4–6 cm with a maximum length of 10 cm based on superior mesenteric vascular axis.

An isolated segment of jejunum is measured under mild stretch to the length of the defect and is transferred in an isoperistaltic orientation. The loop is chosen as proximal to the duodenum as is feasible because here the mesenteric vessels are longest. The oropharyngeal inset can be technically difficult, not least due to usual size discrepancies. Either a slit in the anti-mesenteric border or closing suture of the oropharyngeal remnant may be used to readjust for size mismatches.

Island jejunal patches based on the same vascular arcade can be reoriented and inset into a split in the upper end of the tube to accommodate wide oropharyngeal discrepancies. A nasogastric tube is passed through the jejunal segment, and the tube length is adjusted and anchored at either end, such that the pedicle trails without tension into the recipient vessel site field. Definitive repair of each end awaits microvascular anastomosis so as to reduce ischaemic time. The lower inset is performed last.

All bowel anastomoses are done using interrupted sutures rather than staples or continuous sutures. Any significant redundancy in bowel length will increase the postoperative swallowing difficulties and a common error is to design the segment too long. A survey of the reported outcomes in the literature, outlined in the next section, supports its reliability and low complication rate (Figure 28.1).

Flap monitoring can be performed by visualizing the mesentery through a temporary window in the neck incision or externalization of a separate jejunal ring isolated separately on the same vascular arcade. High reconstructions may be visible through the mouth, otherwise by endoscopy.

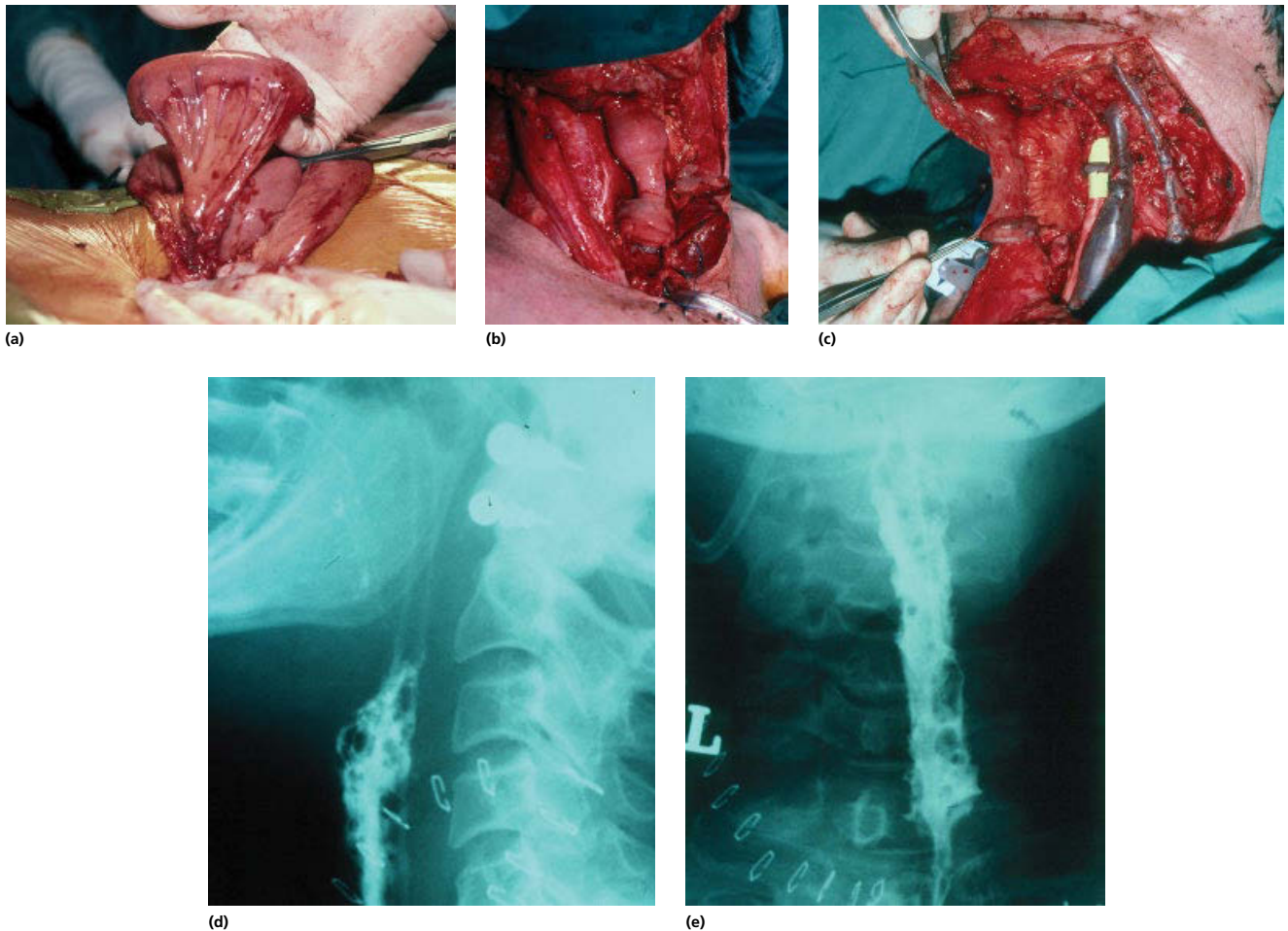


Figure 28.1 (a, b, c, d, e) Jejunum harvest and inset with follow-up contrast swallow X-rays.

Complications related to free jejunum flaps

In addition to general medical morbidity and mortality related chiefly to cardiorespiratory embarrassment, flap-related complications include:

- total flap necrosis (0–13%)
- pharyngocutaneous fistula formation (0–22%)
- stricture formation (0–16%)
- abdominal complications (0–4.35%).^{44,46,47,52–56}

In a recent series of 368 consecutive pharyngeal reconstructions the authors compared their results with a meta-analysis of published free skin flaps. They show that the jejunum was superior in all parameters (pharyngocutaneous fistula (8.2% versus 18.7%), stricture formation (10.9% versus 17.9%) and resumption of oral diet (91.6% versus 90.7%).⁵⁶

Free skin flaps

Tubed anterolateral thigh flap

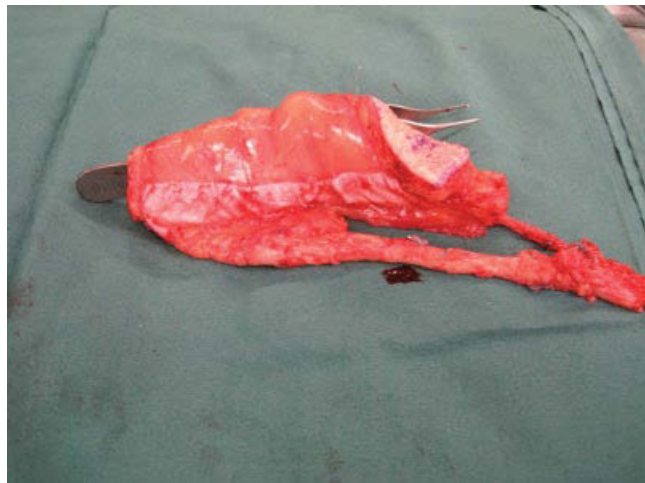
The anterolateral thigh (ALT) flap, originally described as a type B fasciocutaneous flap by Song and colleagues,⁵⁷ has been shown to be mostly a type C fasciocutaneous flap. The flap is based on the

descending branch of the lateral circumflex artery. Often multiple perforators are noted, which can be harvested after dissection through their intramuscular course.³¹ This is a robust flap with a very long pedicle. The typical head and neck cancer patient is male and has lost weight, both favourable characteristics for ALT flap usage. This makes it the preferred skin flap for pharyngeal reconstructions. Its fascial layer can be taken beyond the limits of the skin edge and permit a double layer repair without overlap of the suture lines, thereby reducing fistula and stricture formation. The long pedicle makes for easy anastomosis compared with bowel and the donor site can be closed directly. This flap has gained strong popularity in reconstruction of the hypopharynx⁵⁸ (Figure 28.2).

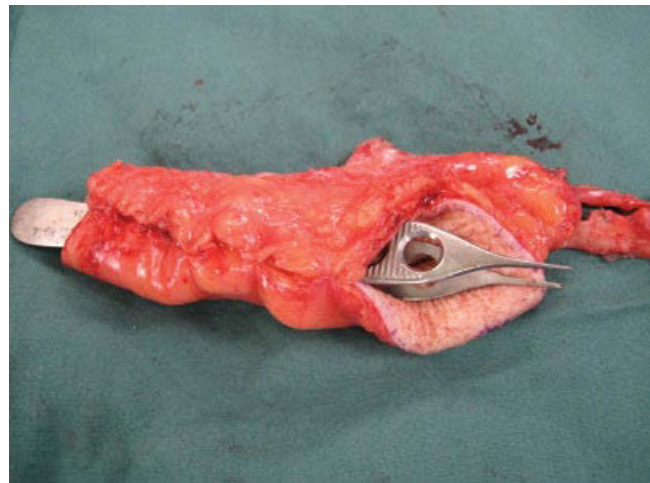
Tubed radial artery forearm flap

Assessment and operative planning for the radial artery flap starts preoperatively with confirmation of a favourable Allen's test. This confirms viable perfusion of the hand after ligation of the radial artery.

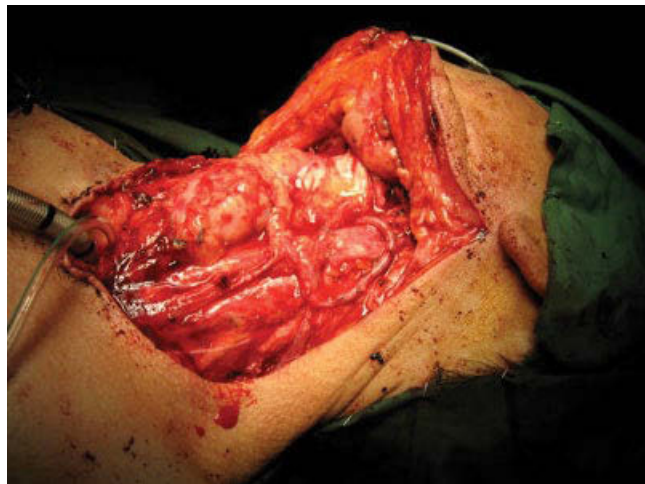
The radial forearm flap is a well-vascularized fasciocutaneous flap^{29,59–61} based on the radial artery and its venae comitan-



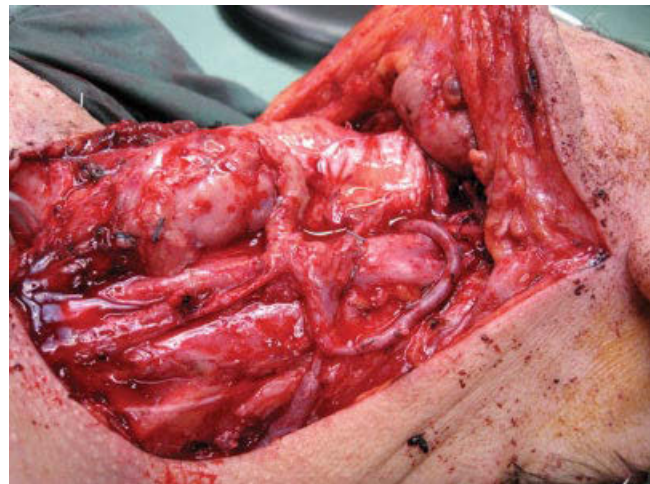
(a)



(b)



(c)



(d)

Figure 28.2 (a, b, c, d) Tubed anterolateral thigh harvest and inset courtesy of Dr Ed Ekk.

tes, a long, large-diameter pedicle that has ensured this flap's popularity. The subcutaneous venous tributaries can be taken as alternative or additional drainage. The perforating vessels penetrate the radial aspect of the thin septum separating the flexor carpi radialis (FCR) and brachioradialis tendons. The septocutaneous perforators are concentrated in the lower third of the forearm along the axis of the artery and the fascia lateral or medial to this cleft is not essential for its viability. As the flap vascularity is distal and the skin is favourably thinner distally, the flap design is correspondingly from this region. It should be noted that the forearm skin is less hairy on the ulnar side and the skin island is best harvested from this side, its vascular source being still adequate from the radial perforators carried via the fascial bridge. When the flap is designed distally it is necessary to trace the pedicle to the cubital fossa even if pedicle length is not an issue, as the venae comitantes are of very small diameter until almost the level of the cubital fossa.

Radial and ALT flap-related complications include:

- total flap failure (0–5%)
- pharyngocutaneous fistula (1–27%)
- stricture formation (2–30%)
- partial flap loss (0–5%).^{1,44,45,58,62–66}

Miscellaneous

Lateral arm, ulnar and even tensor fascia lata flaps have been described as fasciocutaneous options. Ileocolic segments using the ileocaecal valve, free colonic segments as well as gastro-omental flaps have also been described as visceral alternatives. These options represent third-line options and are beyond the scope of this text.

Voice

At its most basic, voice represents increasing complex manipulation of a fundamental tone, which finds its origin at the larynx, before passing through the chambers above and exiting as voice. Speech is conversion of this sound by the tongue, the soft and hard palates, lips, pharynx and teeth. The vocal folds open and close in rapid succession, allowing air to form periodic sound waves. The movement theory behind vocal fold function is called myoelastic aerodynamic theory.⁶⁷ In simple terms, as the pressure rises below the vocal folds, this pressure leads to release of the air and forces the vocal cords apart, thereby generating vibration. As the pressure drops momentarily the vocal folds fall back into their midline position and the cycle continues.

Voice rehabilitation devices

There are many forms of vocal rehabilitation, basically divided into *non-surgical* or *surgical*. The non-surgical means include the electrolarynx and pneumatic artificial larynx. Surgical methods of reconstruction are themselves divided into neoglottis, TEP and prosthesis, and visceral voice tube reconstruction.

Neoglottis

Using local tissues as flaps, a deliberate fistula is created between the trachea and the oesophagus. The problem with this method is that there is a high rate of regurgitation, aspiration as well as stenosis and fistula closure.

Tracheoesophageal puncture and prosthesis

In this method, a fistula is created between the posterior wall of the trachea and the oesophagus. A prosthetic device with a one-way valve is then placed across the fistula. By occluding the tracheal stoma air is forced through the fistula, vibrating sequentially up the oesophagus and being articulated into speech on leaving the mouth. The one-way valve prevents regurgitation of food from the oesophagus into the trachea. There are a variety of these prostheses on the market, including Blom-Singer, Provox and Nijdam devices.^{68–73} There is debate as to whether TEP should be performed primarily or secondarily, some arguing that the incidence of stricture formation is higher in a primary setting although other studies disagree.⁷⁴ Despite their inherent advantage in application and functional outcome there are a number of associated disadvantages, which include: dilatation of the TEP and associated aspiration and regurgitation, stenosis, uncommonly oesophageal perforation, colonization with fungus and bacteria, dislodgement of the prosthesis into the trachea leading to aspiration and obstruction from secretions and food particles. This TEP procedure can be performed between the posterior tracheal wall as well as the neo-oesophagus when constructed from pedicled or free tissue transfer.

Visceral voice tube reconstruction

Where a free jejunum or ileocolon is used, a segment of jejunum or ileum can be used as a voice chamber. These are uncommonly used. The jejunal segment or ileum is used as a conduit for air to be transferred into the neo-oesophagus and pharynx.^{16,75,76}

Complications and clinical outcomes: Study results

There are a large number of publications reporting functional outcomes, including swallowing, stricture and fistula formation, operative surgical outcomes such as flap failure, and medical complications as well as death, but there are no randomized trial studies comparing the various visceral and fasciocutaneous flap reconstructions. Many of the functional outcome measures are not consistent across the literature, making comparisons difficult. In general, it is thought the jejunal flap yields better swallowing with less chance of stricture, whereas fascio- or myocutaneous flaps, by the virtue of tubing inset and incisional closures, are more likely to stricture. The voice and thus speech quality associated with skin flaps, however, is thought to be superior to the wet quality of jejunum voice, but the evidence

is weak. One small series reports most of the speech parameters were inferior with the jejunal reconstructions.⁷⁷ In a longer term study of patients undergoing jejunal reconstructions, Sharp *et al.*⁷⁸ reported 73% of their series had no dysphonia and 89% had no or mild dysphagia.

In a large series, Deschler and colleagues reported a comparison between 919 jejunal flaps and 179 tubed forearm radial flaps. Flap losses were 7.5% versus 1.8%, whereas fistula and stenosis formation were 17% versus 23% and 9.1% versus 16%, respectively.⁷⁹ The best speech is achieved with a TEP, although tracheal, jejunal as well as ileal conduits have been reported, but remain uncommon.^{80,81} Other reports from data pooled from a large series of results suggest that the risk of stenosis or fistula formation is increased substantially with tubed fasciocutaneous radial forearm flaps when compared to jejunal reconstructions. In addition to the results discussed above, Tables 28.2 and 28.3 summarize some of the larger data series on pharyngeal reconstructions.⁸²

Management strategies for major flap complications

Pharyngocutaneous fistula

Fistulas are common complications of pharyngeal reconstructions. The literature, although varied, reflects a trend towards a higher rate in fasciocutaneous flaps, perhaps reflecting the additional seam suture line associated with tubing of the flaps, as well as, in pedicled flaps, the risk of tension on the most vital part of the flap. Various studies have sought to delineate specific predictive risk factors attributable to:

- tumour factor – extent of disease and extent of oncological resection; or
- patient factors – preoperative albumin, age, sex, weight, as well as radiation history among other parameters.

Flap type

A recent report looked at early postoperative indicators for fistula formation. Postoperative fever during the first 48 h,⁸⁷ atrial fibrillation and preoperative albumin could not be implicated. However, a persistent low postoperative albumin (as early as 3rd postoperative day) as well as elevated white cell count was shown to be an indicator of an early leak.⁸⁸

Most commonly, fistulas are managed conservatively, ideally directing drainage to the contralateral neck, away from the great vessels if a neck dissection has been performed. The great majority of small fistulas close spontaneously over the course of 1–2 weeks. Significant leaks will require judicious debridement of necrotic and infected tissues and importation of healthy vascularized tissue. Consideration of regional flaps such as pectoralis major, deltopectoral and latissimus dorsi are the likely best options.^{89–91} Clinical surveillance remains important as contrast studies may yield false-negative rates of up to 14%. Appropriate prophylactic antibiotics, antireflux medication⁹² (proton pump

inhibitors or ranitidine) and meticulous surgical technique and tension-free closure help reduce infection and fistula rates. Late fistulas during or after radiation therapy are relatively rare, but should be considered with any patient who presents with evidence of infection in the neck during the late postoperative period.

Flap failure

This is a serious setback for the patient and the surgeon. Luckily, with increased experience and better practices, failure rates continue to decline in major institutions. Monitoring is difficult but early recognition of flap compromise may enable revision of the anastomoses and salvage of the flap. This is possible with skin flaps but unlikely with bowel. Acceptance of failure and early debridement may allow immediate re-do reconstruction in the interests of obtaining healing in time for optimal scheduling of radiotherapy. But the wisdom of this secondary reconstruction is in part determined by the state of the patient as well as local cervical tissues. Where there is gross contamination and infection there is a real risk of repeat micro-surgical failure, and a regional flap reconstruction should generally be considered, after debridement, external fistula formation, salivary diversion and settling of infection. Onoda *et al.*,⁹³ however, with experience of over 600 free jejunal transfers at their unit, report use of an immediate second jejunal flap where the first flap has failed, allowing adjuvant therapy to proceed quickly. The most important clinical sign of a jejunal flap failure is per oral bleeding.⁹⁴

Stricture formation

Stricture formation, which may be related to flap type or postoperative radiation therapy, could lead to dysphagia. This is often treated with repeated endoscopic dilatations.

Future directions and tissue engineering

Current reconstructive techniques provide restoration of only the most primitive of functions. The great frontier for a reconstructive surgeon remains a nuanced reconstruction and return of function. Increasingly, tissue engineering is yielding promising results in regenerating various tissues and structural combinations. The three requirements are scaffold, cells and *in vivo* regulatory growth factors. Rudimentary tracheal reconstruction for tracheal stenosis has already been performed using a combination of Marlex and bovine collagen-based scaffold.⁹⁵

This is of course a long way away from reconstructing a functional larynx. Although still in its infancy, the future is pregnant with hope that one day *like tissue* can in fact be replaced with *like tissue*, as Sir Harold Gillies, the founder of the discipline of plastic surgery, had set in stone as the ultimate aim of restoration surgery.

Table 28.2 Microsurgical flaps

	Defect	No. pts	Successful swallowing (%)	Stricture (%)	Fistula leak (%)	Flap failure (%)	Medical/other surgical complication (%)	Mortality (%)	Pts who received voice prosthesis (%)	Functional voice prosthesis (%)
Mixed free flaps										
Clark <i>et al.</i> ⁴⁵	Hypopharynx	15 RFFF, 11 ALT, 30 jejunum	84	14	27	5	12	–	44	–
	Pharyngoesophageal	8 RFFF, 13 scapular + LD, 6 gastro-omental 3 jejunum	81	19	25	2	15	3	–	–
RFFF										
Azizzadeh <i>et al.</i> ⁶³	Pharyngoesophageal	20	85	20	20	0	–	0	20	100
Disa <i>et al.</i> ⁴	Hypopharynx	52	95	2	1	4	–	–	–	–
Murray <i>et al.</i> ^{66a}	Hypopharynx	242	66	18	14	1.5	–	1	22	87
ALT										
Murray <i>et al.</i> ^{66a}	Hypopharynx	67	98	11	16	2.3	–	1	52	88
Yu <i>et al.</i> ⁶⁵	Pharyngoesophageal	26	95	15	8	4	0	–	35	89
Chan <i>et al.</i> ⁴⁴	Pharyngeal	24	100	12.5	12.5	0	0	0	–	–
Sagar <i>et al.</i> ⁶⁴	Hypopharynx	20	65	30	25	0	10	0	100	40
Spyropoulou <i>et al.</i> ⁵⁸	Hypopharynx	55	69	5	18	2	12	0	–	–
Range			65–100	2–30	1–27	0–5	0–15	0–3	2–100	87–100

RFFF, radial forearm free flap; ALT, anterolateral thigh flap.

^aMeta-analysis pooled study results.

Table 28.3 Jejunum free flaps

Defect	No. pts	Successful swallowing (%)	Stricture (%)	Fistula leak (%)	Flap failure (%)	Medical complications (%)	Mortality (%)	Pts who received voice prosthesis (%)	Functional voice prosthesis (%)
Perez-Smith <i>et al.</i> ⁵⁶	368	91.6	10.9	8.2	2.98	3.8%	3.8	70.6	78.1
Chan <i>et al.</i> ⁴⁴	86	100	2.3	4.6	3.4	0	0	–	–
Schusterman <i>et al.</i> ⁵²	48	88	22	16	6	4	2	–	–
Oesophagus									
Carlson <i>et al.</i> ⁴⁷	26	80	16	20	4	19	0	–	–
Cervical									
Oesophagus									
Hypopharynx									
Triboulet <i>et al.</i> ⁵³	77	98.4	12	12	–	7	5	–	–
Cervical									
Oesophagus									
Hypopharynx									
Uchiyama <i>et al.</i> ⁵⁴	168	–	–	–	2	–	–	–	–
Hypopharynx									
Benazzo <i>et al.</i> ⁸³	126	–	–	–	10	15	–	68	–
Cervical									
Oesophagus									
Hypopharynx									
Disa <i>et al.</i> ⁴	29	100	10	10	3	3	0	–	100
Hypopharynx									
Okazaki <i>et al.</i> ⁸⁴	90	88	7	7	0	–	–	–	–
Pharyngoesophagus									
Pesko <i>et al.</i> ⁸⁵	20	90	0	0	0	–	–	–	–
Pharyngoesophagus									
Sarukawa <i>et al.</i> ⁵⁵	6	–	–	–	4	33	17	–	–
Pharyngoesophagus									
Yu <i>et al.</i> ⁶⁵	191	94	13	13	6	–	0	29	–
Pharyngoesophagus									
Ferahkose <i>et al.</i> ⁸⁶	31	98	7	7	7	16	0	–	22
Cervical									
Oesophagus									
Hypopharynx									
Mardini <i>et al.</i> ⁸²	12	83	0	0	0	43	7	–	–
Cervical									
Oesophagus									
Range	6–368	88–100	0–22	0–32	0–10	0–43	0–17	29–68	22–100

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CHAPTER 29

Skull base reconstruction

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Introduction

Management of skull base malignancies has over the years posed a challenge to both ablative and reconstructive surgeons. Surgical access, complete tumour extirpation, proximity and concentration of vital neurovascular structures traversing the surgical field have been tempered in equal measure by reconstructive needs for robust, watertight seal, dead space obliteration, while ideally providing adequate skeletal and soft tissue support for the defect.

Early reconstructive attempts were limited to skin grafts, which were often applied to bare dura or bone.^{1,2} Staged tubed pedicled flaps became a mainstay treatment, with temporalis muscle the only other local option for orbital reconstruction and other similar defects.^{3,4} The advent of a successful craniofacial surgical approach, development of axial flaps and free flaps, as well as increased accuracy of definition in imaging techniques have led to a steady improvement in survival and reconstructive outcomes.¹ Bakamjian, McGregor and Thompson described use of axial faciocutaneous flaps (deltopectoral and forehead flaps) for oropharyngeal and orbital reconstructions.^{5–7} The pectoralis major⁸ and latissimus dorsi⁹ myocutaneous flaps were stretched to their limits, transposed externally to the orbital level and middle cranial fossa, respectively.

The introduction of microsurgery brought about the tools and means to transfer well-vascularized tissue and bulk to large defects in all parts of the skull base. Sculpted to fit the defect, provide a watertight seal separating the cranial fossae from the external environment, free tissue transfer can also provide well-vascularized tissue to either an already irradiated field or one which may undergo postoperative radiotherapy.

Anatomy and classification

The extracranial part of the skull base includes sphenoidal sinus, nasoethmoid, nasopharynx including the pterygopalatine fossa, the infratemporal fossa as well as the orbital roof.

The floor of the anterior, middle and posterior fossae forms its intracranial surface. Multiple classifications based on reconstructive requirements as well as anatomic boundaries, pathologic process and disease spread have been devised. Jackson and Hide divided the region into anterior and posterior divisions. The posterior area being further subdivided into posterior–anterior, posterior–central and posterior–posterior based on reconstructive requirements.¹⁰

Irish and colleagues reviewed 77 skull base neoplasms and divided the region into three zones based on anatomical boundaries and tumour spread¹¹ (Figure 29.1).

- Zone I: All tumours arising from the sinuses, orbit, other anterior structures as well as those which arise from the clivus and extend posteriorly to foramen magnum are included in this zone. Resection of these tumours requires an anterior approach.
- Zone II: These tumours primarily arise in the region of the infratemporal and pterygopalatine fossae. They extend into the middle cranial fossa.
- Zone III: neoplasms from the lateral face, parotid gland, ear and the temporal bone are included in this group extending into the posterior cranial fossa.

Pathology

Pathology of skull base neoplasms is broadly divided into those infiltrating from outside the skull base inwards and those originating from internal structures. By far the most common of these infiltrating lesions are cutaneous malignancies, especially squamous cell carcinomas (SCCs), but including other pathologies, such as nasopharyngeal carcinomas, salivary glands and glomous tumours. Those originating from the inside include acoustic neuromas, schwannomas and meningiomas. Among the more unusual tumours are lymphomas and sarcomas. Tissue diagnosis is imperative as surgery may not be appropriate in all cases, such as lymphomas.

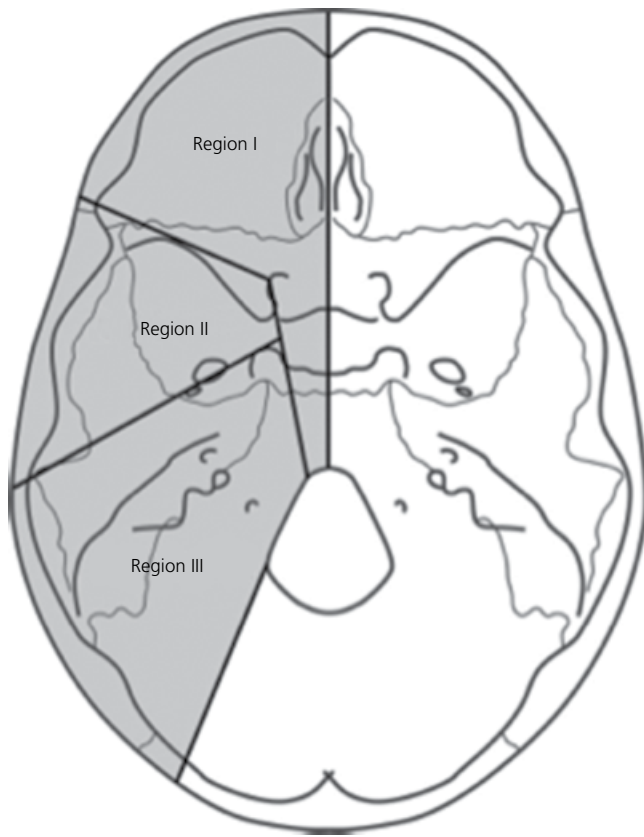


Figure 29.1 Skull base zones based on Irish and colleagues' review¹¹ of 77 patients with skull base neoplasms. From Irish *et al.* (1994).¹¹ Reproduced with permission of Lippincott Williams & Wilkins.

Principles of management

A multidisciplinary team including specialist radiologists, radiation oncologists, medical oncologists, pathologists, nutritionists, neurosurgeons, plastic as well as head and neck surgeons forms the successful approach to management of patients with base of skull neoplasms. Improvements in imaging including increased accuracy and utilization of MRIs have significantly improved preoperative resection and reconstructive plans. Precise tissue diagnosis, extent of local, regional and distant disease and the patient's general examination and medical history are considered by the team before a comprehensive oncologic and rehabilitation plan is put forward to the patient.

As with many classifications the aforementioned represent arbitrary dividing lines across disease type and anatomical terrains. The general principles of management, however, remain vital in considering individual cases. These include:

- adequate surgical access determined by site, size and type;
- oncologic clearance or palliative debulking;
- repair of dural defects where present, which may, depending on the neurosurgery approach, require a prophylactic spinal drain to reduce intraventricular pressure and reduce the risk of cerebrospinal fluid (CSF) leak;

- watertight seal of intracranial contents from the external environment of the nasopharynx to reduce the risk of ascending meningitis;
- provision of bulk and obliteration of dead space;
- introduction of newly vascularized tissue, particularly in the settings of salvage surgery post radiation therapy disease progression or need for postoperative radiotherapy considerations; and
- provision of skeletal support where possible and necessary.

Zone I: Anterior skull base

Surgical access depending on the tumour type, size and site often requires a combined craniofacial approach. A lateral rhinotomy or a maxillotomy may be combined with a bicoronal approach both for access and protection of vital structures such as the optic nerve under direct view.^{12,13} When access to the clivus is required, a combined Le Fort I osteotomy, midline mandubolotomy and palatal split is taken in order to protect all peripheral vital structures.

Primitive reconstructions using split skin grafts applied to bare dura or bone resulted in high complication rates, up to 50% CSF leak in some of Ketcham's original series.¹ Local flaps are mostly inadequate for the purposes of such reconstructions. Early options included forehead, glabellar, pericranial or combined galeal-pericranial flaps.^{7,14–18} The temporalis muscle flap is also an option. This type III Mathes and Nahai muscle is supplied via deep temporal arterial branches of the maxillary division of the external carotid artery. Medial transposition into zone I is limited in its arc of rotation, barely reaching the medial orbital wall. Its most distal and critically needed portion is vulnerable to vascular insufficiency. Furthermore, this muscle maybe devascularized in neck dissections with ligation of external carotid artery. Donor site morbidity and temporal hollowing are significant drawbacks.⁴

Although myocutaneous flaps have been superseded by free flap surgery, they still have a place in reconstructive algorithms, especially in salvage of previous failed surgery. However, all are restricted by their pedicle reach and risk of distal necrosis. Pectoralis major, latissimus dorsi as well as trapezius muscle are the chief pedicled myocutaneous flaps available.^{8,9,19–21} Free flaps not constrained by anatomical limitations of pedicle origin nor potential of distal tip vascular insufficiency offer the opportunity to sculpt tissues to the defect, provide robust vascularized tissue, encourage healing and reduce complication rates. Where the extent of the resection dictates and there is a need for replacement of skeletal support, vascularized bone is preferable and essential if the field has received radiotherapy. Options include use of the free fibular with flexor hallucis longus muscle, deep circumflex iliac crest, parascapular with bone, scapular with bone, latissimus dorsi with angle of scapula or serratus anterior with rib flaps. These flaps can provide muscle bulk for dead space obliteration as well as vascularized bone for structural support. Alternatively, orbital wall, maxillary and cranial vault defects may be reconstructed with titanium mesh, provided well-vascularized coverage can be assured.

Zone II

The middle cranial, infratemporal and pterygopalatine fossae, extending from the posterior wall of the orbit, form zone II. The vital contents traversing it include facial, vestibulocochlear, maxillary and mandibular divisions of the trigeminal nerves, along with branches of carotid artery, a veritable anatomical minefield. The most common neoplasms extending from the outside into the region are cutaneous or parotid-based tumours. Nasopharyngeal carcinomas, meningiomas as well as glomus tumours are the common tumours arising inside zone II. In their analysis of base-of-skull tumours leading to this classification system, Irish and colleagues noted poor prognosis of tumours in this zone.¹¹

The reconstructive requirements depend on the patient as well as the size of the defect. Early reconstructive attempts utilized local options such as lateral scalp rotation and temporalis flaps. Deltopectoral, often extended through a delay procedure, was the only regional option prior to the advent of myocutaneous pedicled flaps. Workhorse myocutaneous flaps include pectoralis major, latissimus dorsi and trapezius. Microsurgical options are by far the best option in this zone and allow a well-vascularized sculpted tissue bulk with reliable perfusion to be introduced into the defect. Rectus abdominis myocutaneous flap provides a large, well-vascularized flap for such reconstructive purposes, with an adequately long pedicle, but its skin component is often unfavourable and the donor site defect is potentially morbid. More recently the anterolateral thigh fasciocutaneous flap has become the favoured donor because it allows a two-team approach, simultaneous dissection, long pedicle, large skin component with often direct closure of the defect, and, where needed, a nerve component. When the two ends of the facial nerve are present, even where there is previous weakness. Iseli and colleagues reported some facial nerve recovery in 97% of patients at a median of 6.2 months post operatively.²² Another study reported some recovery in 75% after a median of 7.7 months post operatively.²³

Zone III

Zone III contents include posterior cranial fossa, internal carotid artery, internal jugular vein, glossopharyngeal, vagus, accessory and hypoglossal nerves. Although Jones *et al.* reported schwannomas and glomus tumours as the most common presenting neoplasm in zone III, our experience, like that of Irish *et al.*, was that SCCs form the majority of these malignancies.^{11,24} Where a neck dissection is undertaken there is a likely devascularization of local flaps, including the temporalis muscle. Depending on size and site, regional options such as latissimus dorsi or trapezius muscles maybe used, or microsurgical options including faciocutaneous or myocutaneous flaps. Considerations should be given to patient position for flap donor site to allow simultaneous surgery by the resecting and reconstructive teams.

Technical consideration

Flap inset and dural seal are particularly important parts of the reconstructive consideration, complicated by gravity pulling the flap away from the base of skull and the dural repair site. Fibrin glue at the dural-free flap interface strengthens the repair. Thought must be given to providing enough soft tissue support to completely obliterate the dead space, abutting the flap as closely as possible against the repair site; suspension of the flap against the skull base provides additional support. In zone I the facial and superficial temporal vessels are the best recipients; care must be taken with the superficial temporal vein, which is thin walled. Where these are not available, a longer pedicle is needed and upper neck vessels may be used. For zones II and III, neck vessels are the most common recipient. Given their anatomical site, these flaps are commonly buried, especially in zone I. Implantable dopplers may be used, or a monitoring paddle can be exteriorized. Monitoring the pedicle distal to the microanastomosis site with a hand-held doppler is reassuring but does not detect venous obstruction.

Increasingly, the anterolateral thigh flap is used for reconstruction. In addition to allowing a two-team approach, having a low donor site complication rate, providing a long vascular pedicle, this flap permits harvesting of muscle for tissue bulk where needed as well as lateral cutaneous nerve of thigh as a graft source where primary facial nerve reconstruction is undertaken.

Complications

In addition to the standard medical complications, early surgical complications are most often related to communication of the dural space with the aerodigestive tract. Dural exposure, CSF leakage (up to 7%), ascending meningitis and local infections are the main risks.^{23,25} Free flap losses have steadily decreased; large series have showed a decline from 4% to 1.9% as a result of technical refinements.^{23,26,27} The overall complication rates remain significant, ranging between 29% and 60%.^{27–29} A more recent series reports overall complication rates of 30%.²³ Increasingly, free flaps are utilized to treat these defects. Neligan and colleagues reported higher complication rates where pedicled flaps were utilized.²⁸

Long-term limitations of the reconstruction include facial asymmetry, deformity, diplopia, trismus and nasal obstruction. These are inherent to lack of bony support, flap tissue bulk as well as a sequelae of radiation fibrosis.²⁵ This is hardly surprising considering the gross imbalance in sophistication between the tissues resected and tissues, which are chosen to replace them. Head and neck reconstruction remains the one great frontier for imaginative advances to be made in reconstructive surgery.

Superior imaging, refined surgical techniques and better preoperative planning have made reconstruction of the skull base malignancies, once considered inoperable and dangerous, routine at major centres. Appropriate consideration has to be

given to access, oncologic resection, vital structures traversing the region and technical considerations necessary for an optimal reconstruction.

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CHAPTER 30

Cheek reconstruction

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Introduction

Defects of the cheek pose a significant challenge for the reconstructive surgeon. It is a highly visible area that may easily be compared to the opposite side, highlighting any asymmetry. This is perhaps one area in plastic surgery where reconstructive principles and aesthetic ideals are intertwined inextricably. An advanced understanding of facial anatomy and relevant reconstructive techniques is essential in tailoring the best solutions to the defect.

Anatomy

The cheek is a region on the face bordered by the eye, nose, lips, chin, mandible and ear (Figure 30.1). Functionally, it is important for mastication, swallowing and speech.¹ Aesthetically, it was classically described by Menick as 'a relatively flat, expansive surface, except for the soft roundness of the nasolabial folds and the cheek prominences'.² In greater detail, it is bordered by infraorbital rim and zygomatic arch superiorly, the lower lid–cheek junction, nasofacial junction, nasolabial fold, marionette line and labio-mandibular crease medially, the mandible inferiorly, and the preauricular contours of the tragus and helix of the ear laterally.^{2–4}

Skeletal

The facial skeleton underlying the cheek includes the mandible, zygoma, and maxilla.

Layers

In general, the layers of soft tissue in the cheek correspond to the basic five layers found elsewhere in the body (Table 30.1). From superficial to deep, the layers are skin, or dermis, followed by a layer of dense subcutaneous fat which is separated into lobules enclosed by fibrous septa. The third layer, the superficial

musculoaponeurotic system, or SMAS, was first described in 1976 by Mitz and Peyronie.⁵ It is continuous with the frontalis muscle superiorly, and the platysma inferiorly. There is another, looser layer of fat, between this and the fifth layer, that of the periosteum or deep fascia, which is often absent in the face where the tissues do not overlie bone or skeletal muscle.⁶ It may be helpful to remember them by the widely taught 'SCALP' mnemonic, corresponding to skin, connective tissue, aponeurotic layer, loose connective tissue and periosteum. In areas of the face not overlying bone, different layers are encountered. For example, in the parotid region the parotid fascia corresponds to the deep fascia, which takes the place of the periosteum; and in the buccal subunit of the cheek, the innermost layer of the cheek is the buccal mucosa. The superficial layers are otherwise the same.

Retaining ligaments

Fascial structures provide an anchor between bone and skin and provide soft tissue contours throughout the body.⁸ The retaining ligaments of the face have been studied in some detail as they are of particular relevance for aesthetic plastic surgical procedures such as rhytidectomy. They are of less importance in cheek reconstruction, as they will often have been disrupted by the trauma or oncologic resection which results in the defect. Nevertheless, a working knowledge of the retaining ligaments is helpful for the reconstructive surgeon. These are referred to elsewhere in this book. Briefly, the retaining ligaments of the cheek were first examined by Furnas in 1989.⁹ He described groups of ligaments that remain essential to our understanding of the soft tissue structure of the cheek:^{10–14}

- tear trough ligament
- orbicularis retaining ligament
- zygomatic cutaneous ligaments (McGregor's patch)
- masseteric cutaneous ligaments
- mandibular cutaneous ligament
- platysma auricular ligament.

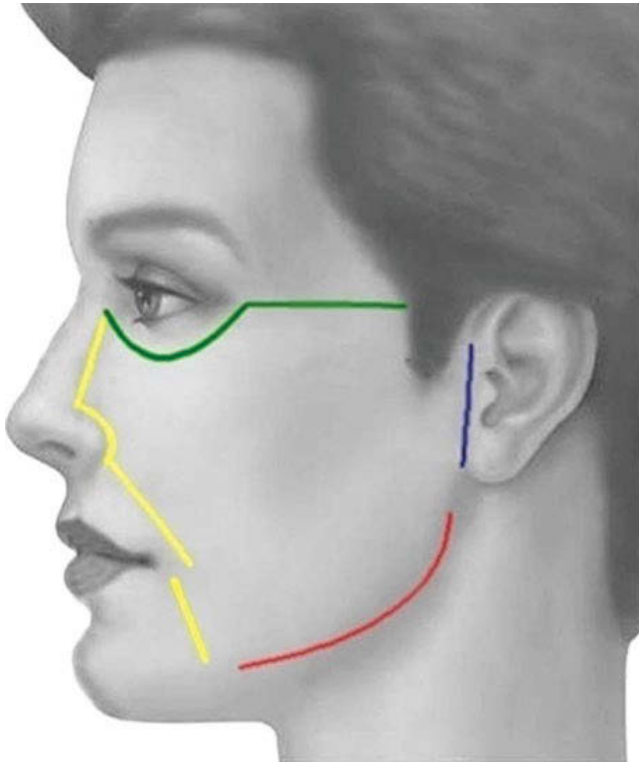


Figure 30.1 Borders of the cheek. It is outlined by the infraorbital rim and zygomatic arch superiorly (green line), the lower lid-cheek junction, nasofacial junction, nasolabial fold, and labiomandibular crease medially (yellow lines), the mandible inferiorly (red line), and the preauricular crease laterally (blue line). Source: Dobratz and Hilger, 2009.⁴ Reproduced with permission of Elsevier.

Table 30.1 Tissue layers of the face

1	Dermis
2	Subcutaneous layer
3	Superficial musculoaponeurotic system (SMAS) layer
4	Loose areolar tissue
5	Periosteum and deep fascia, or buccal mucosa

Source: O'Brien *et al.*, 2013.⁷ Adapted with permission of Lippincott Williams & Wilkins

These ligaments provide dense connections between either bone or fascia and the overlying skin and help to create many of the creases of the face. In reconstruction, it may be necessary to disrupt intact retaining ligaments in order to gain sufficient mobilization of flaps, or to recreate the effect of the retaining ligaments using deep sutures to underlying structures.

Muscles

The majority of the muscles of the cheek are muscles of facial expression. They are embryologically derived from the second branchial arch and are supplied by the nerve of that arch: the

facial. The exception is masseter. As a muscle of mastication it is derived from the first branchial arch, and is supplied by the mandibular division of the trigeminal nerve.¹⁵ The muscles of the cheek are:

- orbicularis oculi
- levator labii superioris
- levator labii superioris alaeque nasi
- depressor anguli oris
- risorius
- zygomaticus major
- zygomaticus minor
- platysma
- levator anguli oris
- buccinator
- masseter.

Blood supply

Branches of the external carotid artery supply all the tissues of the cheek. The facial artery (previously the external maxillary) arises from the external carotid artery in the region of the carotid triangle, runs across the inferior margin of the mandible anterior to the insertion of masseter, where the pulse may be palpated, runs a course that is convex anteriorly across the cheek deep to the zygomaticus muscles and becomes the angular artery at the medial canthus. Its branches are the ascending palatine, tonsillar, submental, glandular, inferior labial, superior labial and lateral nasal. It is accompanied by the facial vein, which runs lateral to the artery. The angiosome of the facial artery includes the medial cheek.¹⁶ The infraorbital artery is a branch of the maxillary artery (itself a terminal branch of the external carotid). It branches from the first part of the maxillary artery in the infratemporal fossa, then runs through the infraorbital canal to emerge on the face from the infraorbital foramen, where it anastomoses with the facial artery. The infraorbital vein accompanies the artery in its course and is a tributary of the facial vein. The superficial temporal artery runs upward within the substance of the parotid gland between the TMJ and the external acoustic meatus over the posterior zygomatic arch. It gives off two branches in the cheek: the transverse facial artery and the zygomatico-orbital artery.

The parotid duct and the buccal branches of the facial nerve accompany the transverse facial artery, the angiosome of which comprises the parotid gland and lateral cheek.¹⁶ The zygomatico-orbital artery runs superior to the zygomatic arch in parallel with the transverse facial artery as far as the orbicularis oculi muscle. It is accompanied by the retromandibular vein, which is formed by the union of the superficial temporal and maxillary veins from the pterygoid plexus. It divides into anterior and posterior branches, the former joining the facial vein before emptying into the internal jugular vein, the latter forming the external jugular vein with the posterior auricular vein.^{15,17}

Lymphatics

The pattern of lymphatic drainage of the cheek roughly mirrors the venous system, and is chiefly to the submental, submandibular, and periauricular nodes.¹⁸

Nerves

When operating on the cheek, the anatomic structure of chief importance is the facial nerve. As the sole motor supply to the muscles of facial expression, any facial nerve injury can have significant and lasting effects on patients quality of life. After emerging from the stylomastoid foramen the facial nerve enters the parotid gland. From this point on the ramification pattern of the facial nerve is virtually infinitely variable. In general, upon emerging from the parotid it has frontal, temporal, buccal, mandibular and cervical branches, and various landmarks have been described for locating individual branches.^{19,20} Depth of facial nerve branches also varies according to the area of the cheek being examined and the presence of tissue spaces, though in general they are deep to the SMAS laterally before becoming more superficial to supply muscles of facial expression medially.

The skin of the cheek, and of the face in general, is supplied almost exclusively by branches of the fifth cranial nerve: the trigeminal. The sole exception is an area of skin overlying the parotid gland and the angle of the mandible, which is supplied by the great auricular nerve, a branch of the cervical plexus. The remainder of the cheek is supplied by branches of the lower two divisions of the trigeminal nerve. The maxillary division provides two branches to the skin of the cheek. The infraorbital nerve supplies the cheek immediately beneath the eyelid, while the zygomaticofacial nerve supplies an area over the zygoma just lateral to the eye. The infraorbital nerve may be blocked by infiltration of local anaesthetic around where it exits the infraorbital foramen, which is located below the medial third of the infraorbital margin. The mandibular division also provides two branches. The buccal nerve supplies an area beneath the zygoma, between the distribution of the infraorbital and great auricular nerves. The mental nerve emerges from the mental foramen in line with the second premolar tooth and supplies the lower lip and surrounding skin.¹⁵

Aesthetic subunits

The cheek may be divided into four subunits: medial, lateral, buccal and zygomatic (Figure 30.2).^{1,21} In some texts, infraorbital and parotidomasseteric subunits are described, which are roughly analogous to the medial and lateral subunits, respectively.²² The medial subunit, otherwise known as the midcheek, may be further divided into prezygomatic and infrazygomatic areas.²³

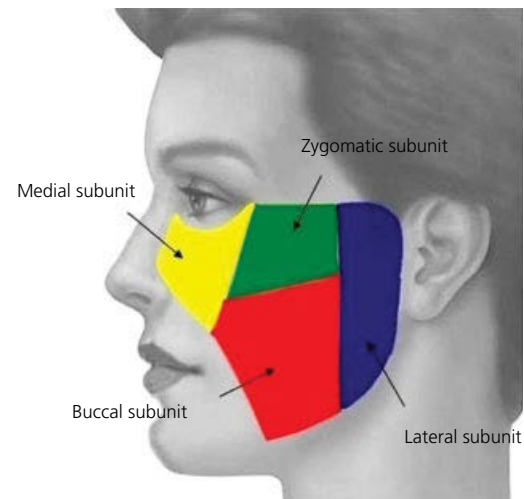


Figure 30.2 Aesthetic subunits of the cheek. Source: Dobratz and Hilger, 2009.⁴ Reproduced with permission of Elsevier.

Management

As is the case throughout reconstructive plastic surgery, the approach to reconstructing a cheek defect should be individualized for the patient, considering their overall health, prognosis, and functional status. The defect should be examined and characterized, noting such features as defect site, size, shape and depth, in addition to wound condition and aetiology, and any associated injury, where relevant.² Clinical photography may be helpful as a record. General principles are helpful to bear in mind while planning and executing the reconstruction, and Menick²⁴ has provided the following useful guidelines for reconstruction of facial units, which may be applied to cheek reconstruction:

- 1 Rebuild or resurface entire units, do not fill defects.
- 2 Alter the wound in site, size, and outline to assist reconstruction.
- 3 Consider separate flaps for individual aesthetic units when reconstructing composite defects.
- 4 Replace like with like – select the best match for skin colour, texture, hair quality, and thickness.
- 5 Free flaps may be used to provide bulk, but local facial skin is preferred for aesthetic covering.
- 6 In composite defects including the nose, the lip or cheek should be repaired initially due to the possibility of distortion during healing.
- 7 Individualize the reconstruction to the defect, allow time for the defect to mature and to plan the repair.
- 8 During revisional procedures, use the contralateral normal as the template.
- 9 Consider use of cartilage and bone grafts where appropriate to recreate the underlying architecture.

It should be borne in mind that in the cheek the crucial element for a good aesthetic result is uniformity of skin colour and texture, rather than contour or outline.²⁴ For this reason, local and

regional flaps from adjacent units are generally preferred over grafting. Exceptions to this rule are in cases requiring total facial resurfacing, in which case a complete skin graft or complete flap are preferred to a combination of other techniques. Distant skin is a poor match for facial skin, but for some wounds (large, contaminated, irradiated) transfer of distant tissue using microvascular techniques is the only solution. In these situations a free flap may be used to provide mucosal lining and tissue build, with local and regional flaps mobilized secondarily to provide permanent external skin. For smaller defects that may be amenable to direct closure, scars should be placed either in the borders of aesthetic units or in parallel with relaxed skin tension lines (RSTLs), which are perpendicular to lines of maximum skin extensibility (Figure 30.3).²⁵

Techniques for reconstruction of cheek defects are as follows:

- secondary intention
- direct closure
- tissue expansion
- local flaps
- cervicofacial flaps
- regional flaps
- skin grafting
- free flaps
- prefabrication.

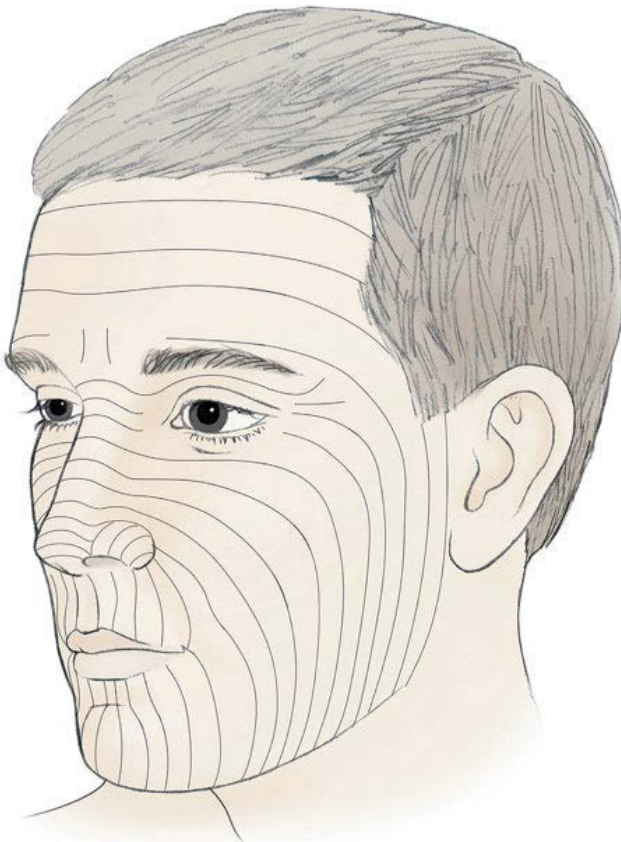


Figure 30.3 Relaxed skin tension lines (RSTLs) of the face. Source: Baker, 2007.²¹ Reproduced with permission of Elsevier.

All of these are useful in the management of defects of the cheek. However, as discussed above, nearby tissues provide the best colour and texture match for cheek skin, and as such local and cervicofacial flaps are generally preferred where the lesion is not amenable to direct closure. Use of each of these techniques in reconstruction of the cheek is discussed below.

Secondary intention

Wounds able to form granulation tissue may be allowed to heal by secondary intention following resection of tumour, or debridement of devitalized tissue following trauma. Defects less than 1 cm may produce an acceptable result when left to heal by secondary intention, however the aesthetic appearance is generally inferior to other approaches due to wound contraction.¹⁸ As such it is generally not appropriate for defects close to the eyelid, nose and lip due to the potential for distortion.⁴

Direct closure

The simplest means of reconstructing a cheek defect is direct closure. It has been suggested that defects of up to 4 cm in size are amenable to direct closure, and that this should be the technique of choice where scars may be concealed in borders of aesthetic units or in parallel with RSTLs.^{18,26} Prior to attempting direct closure, it is important to check that there is sufficient skin laxity to allow tension-free closure. Preoperative examination and intraoperative tailor-tacking may be of assistance in assessing this.³ Undermining of wound edges by up to 4 cm may allow further mobilization of wound edges to directly close defects which would otherwise be under excessive tension.¹⁸ Dog-ears should be excised at the time of initial closure, as it is widely perceived that the cheek is an area in which they are unlikely to settle with time.^{3,27} Areas where direct closure may be a particularly viable option include the perialar and preauricular areas. In both these areas, abundance of skin laxity and proximity to the border of the cheek may allow larger defects to be closed with a well-concealed scar, particularly with the aid of undermining.³

Tissue expansion

Tissue expansion is a useful adjunct for other means of managing a defect in the cheek. Internal or external tissue expanders may be used to increase available skin to enable a repair which would otherwise result in excessive wound tension.^{28,29} However, caution should be exercised to avoid injury or compression of branches of the facial or trigeminal nerves.²⁸ Furthermore, since weeks are required for useful tissue expansion, circumstances such as oncologic urgency may preclude its use in some cases.¹

Local flaps

The most robust and aesthetic means of reconstructing cheek defects not amenable to direct closure are local flaps such as advancement, rhomboid and bilobed flaps. The use of skin

from the same or adjacent aesthetic unit as the defect provides a good match for skin colour and texture, and wound contraction is not as significant an issue as with other methods of closure. Local flaps are of greatest usefulness in defects of the medial cheek, which has greater skin laxity than elsewhere in the face.²⁵ These have been described in detail earlier in this book.

Advancement

A simple advancement flap may be used to close wounds that are too large or where direct closure would result in excessive wound tension. The term is generally used to describe flaps that have a linear configuration, in which the flap is slid into the defect.³⁰ Excision of equalizing or Burow's triangles to lengthen the wound edge may prevent bunching up of tissues and the formation of dog-ears. Laterally based unipedicle and V-Y advancement flaps are particularly useful in the medial cheek, due to the relative mobility and elasticity of tissues.³⁰ Advancement is less useful in the lateral cheek due to the lesser amount of subcutaneous fat and denser connections to the underlying fascia via retaining ligaments.

Rhomboid

The rhomboid flap was developed by Limberg in Russia during the early twentieth century and first described in English in 1946.³¹ It is essentially a variety of random pattern transposition flap utilizing a flap shaped as an equilateral parallelogram (a rhombus).^{32–35} Advantages of the rhomboid flap include flexibility of arrangement, the surgeon being allowed to choose how to place the secondary defect to minimize wound tension and disguise the scar along RSTLs. The technique requires the excision of a rhombus of tissue with internal angles of 60 and 120° around the defect (Figure 30.4a). The flap is then created by making an incision approximately the length of the diameter of the defect outward from its midpoint, followed by a second incision of similar length at a 60° angle to the first.³⁴ The flap is then transposed into the defect. It should be noted that in general, the greatest degree of tension resulting from this method of closure is at the donor site, and that this method requires the excision of additional normal tissue to create the rhombus-shaped defect.³³ To address these problems, modifications of Limberg's original method have been described.

The Dufourmentel flap is one such modification (Figure 30.4b). It is more complex, but allows the surgeon to create a rhombus-shaped defect without being constricted by the original technique's requirement for 60 and 120° internal angles. As for the Limberg flap, the defect is excised in an equilateral parallelogram of tissue. However, the angles may be customized to minimize the amount of normal tissue excised. Lines are then drawn extending from the short diagonal and the midpoint of the defect (see Figure 30.4b). The incision is then made midway between these two lines for a length approximating the length of the sides of the defect. The second inci-

sion is then made for a similar length in the direction of the long axis of the defect. The resulting flap is transposed into the defect.^{32–34}

The Webster flap is a further modification of the Limberg flap (Figure 30.4c). Its design is similar to that of the Dufourmentel flap, but it utilizes an acutely angled W-plasty at the base of the flap to limit the length of the excision and preserve skin.³² Disadvantages of the Webster modification are the increased possibility of contracture, and the additional limb of resulting scar. Other variations on the rhomboid flap include the use of a circular incision and a curved flap³⁶ and the use of bilateral flaps to reconstruct larger defects.³⁷

Bilobed

The bilobed flap was developed by Esser in 1918 and described in English by Zimany in 1953.³⁸ Like the rhombic flap, it is a random pattern transposition flap. It consists of two lobes based on a single pedicle that allows the surgeon to utilize nearby tissue that is not immediately adjacent to the defect.^{34,39} It is most useful for reconstructing defects of the lower one third of the nose, but may be helpful for medium-sized cheek defects. It is also a useful alternative to a larger cervicofacial flap for large cheek defects.³⁹ In Essen's original design, each lobe was the same size as the defect and was rotated 90°. This tended to result in the formation of dog-ears at wound edges and the risk of pincushioning (also known as trapdoor deformity) of the lobes of the flap. To prevent these undesirable aesthetic outcomes, Zitelli described the modified bilobed flap in 1989.³⁸

The first lobe of the modified bilobed flap is placed immediately adjacent but at an angle to the defect, and the second next to the first at a similar angle. The first lobe should be designed slightly smaller than the defect, and the second lobe smaller again. Prior to making the incision it is important to ensure that the donor site for the second lobe can be closed primarily – a pinch test may be helpful in confirming this.³⁹ Once this is done, the first lobe is transposed into the defect, and the second lobe into the secondary defect left by the first lobe. Any resulting dog-ears should be dealt with at the time of the initial operation by excision of an equalizing triangle.

Cervicofacial flaps

The cervicofacial flap, developed from the cheek advancement flaps of Mustardé,⁴⁰ have become the method of choice to reconstruct facial defects that are too large for a local flap. They are the preferred method for even medium-sized defects (larger than 2 cm) for some authors, who find that local flaps result in an aesthetically displeasing scar.³ They are a combination rotation/advancement flap that make use of cervical skin, which provides a good match for facial skin colour, texture and thickness.³ They are useful for large defects of both the lateral cheek, where there is relatively less skin laxity,²⁵ and the medial cheek, where the scar may be con-

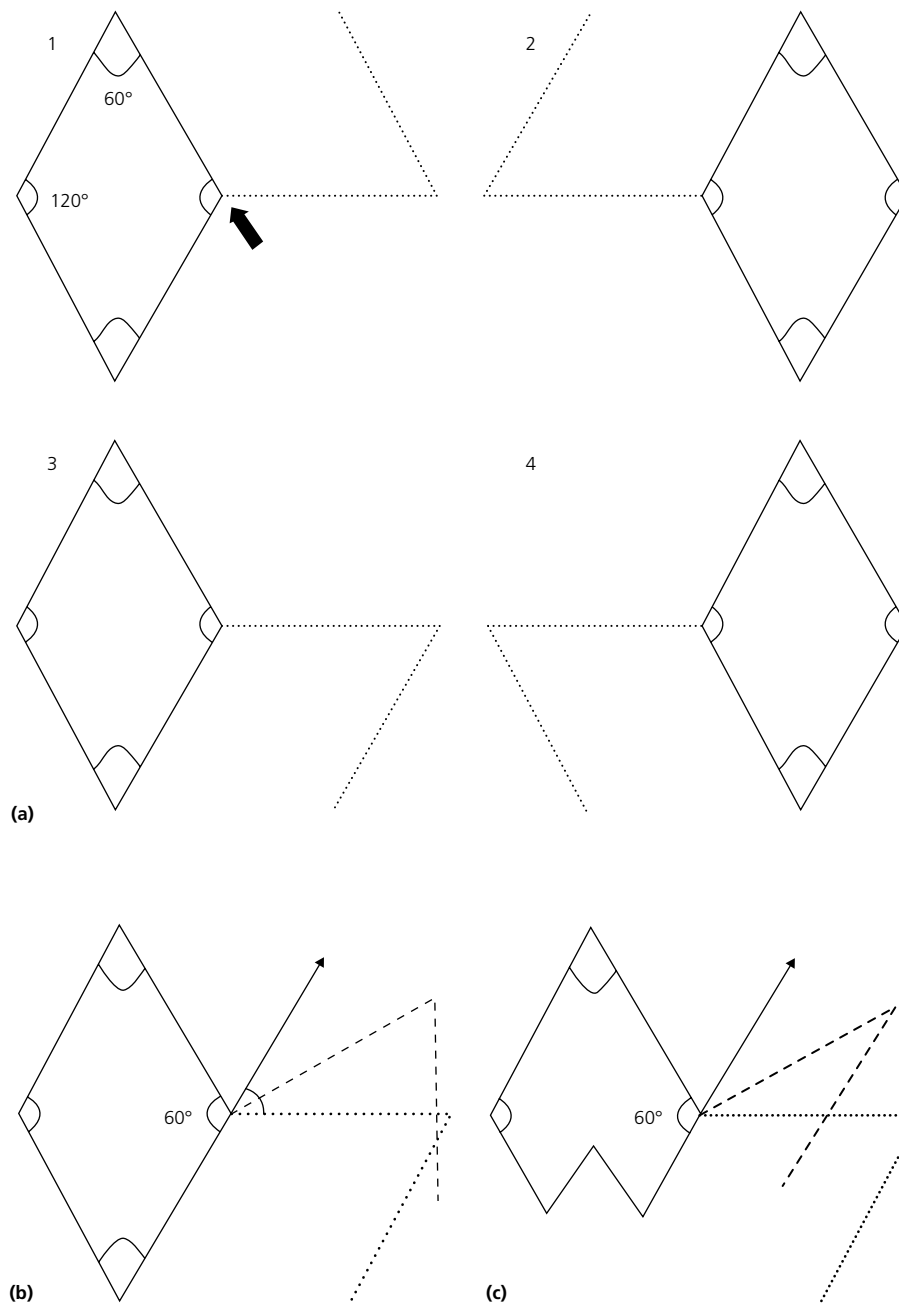


Figure 30.4 Main subtypes of rhomboid pattern flap. (a) Rhomboid flap, (b) Dufourmentel flap, (c) Webster flap. Source: Chu and Byrne, 2009.³⁴ Reproduced with permission of Elsevier.

cealed along the borders of the cheek. Cervicofacial flaps may be anteriorly or posteriorly based depending on the position of the defect and the direction of advancement of the flap. Posteriorly based cervicofacial flaps have a more robust blood supply than anteriorly based flaps due to preservation of the transverse branches of the facial and superficial temporal arteries.²⁷

The incision should follow the borders of the cheek where practical, and extend down onto the neck either anteriorly or

posteriorly depending on the decision of where to base the flap (Figures 30.5 and 30.6). In anteriorly based flaps, the incision follows the superior border of the cheek, the preauricular crease, around the ear toward the occipital hairline, before being extended inferiorly for as far as is necessary to mobilize sufficient tissue prior to back-cutting.¹ For larger defects or those extending into the neck, the incision may be extended inferiorly, creating a cervicothoracic flap.³⁴ For a posteriorly based flap, the incision follows the nasolabial crease and extends onto the

upper neck.⁴¹ Flaps extending to the posterior neck should be kept posterior to the anterior border of trapezius to avoid webbing of the scar.²⁷

Following the incision, the flap must be dissected in order to mobilize it into position. The dissection may be either subcutaneous or in a deeper plane. The deep plane cervicofacial flap (DPCFF) is based on the technique of the sub-SMAS facelift technique,⁴¹ designed to include cutaneous perforators of the facial artery in the case of anteriorly based flaps, or of the superficial temporal artery in posteriorly based flaps. Advocates of the deep plane approach praise its more robust blood supply, allowing more mobility of the flap, and the greater thickness of the flap providing a more aesthetically pleasing contour.^{41,42} Opponents of this approach suggest deep plane dissections are more difficult, with a greater risk of facial nerve injury and a greater risk of ectropion due to the increased weight of the flap.^{3,43} Published rates of flap necrosis are similar for subcutaneous and deep plane approaches.^{41,43} However, there is widespread agreement that the deep plane approach is preferred in patients in whom blood supply to the flap is a significant concern, such as patients with diabetes, smokers or those who have undergone radiotherapy to the area. The senior author's preference is to dissect only in the subcutaneous layer above the zygomatic cheek and overlying the parotid, and in the sub-SMAS plane below.

Regardless of the plane is chosen, the dissection should be extended into the temporal region to create sufficient laxity to prevent tension from the weight of the flap, which may result in ectropion.³ Periosteal suspension sutures may also be used to anchor the leading edge of the flap at the cheek/eyelid junction.^{1,44} Care should be taken during dissection to avoid injury to the branches of the facial nerve, particularly immediately anterior to the parotid where the branches are no longer protected by intervening parotid tissue.⁴² The cutaneous branches of the cervical plexus and mandibular

division of the trigeminal nerve should also be protected. Following dissection, the flap is advanced into the defect and sutured into place.

Regional flaps

For patients with large defects of the cheek in whom cervicofacial flaps are not viable, regional flaps are a robust option. Flaps to consider include the supraclavicular and deltopectoral flaps. The supraclavicular flap is an axial pattern flap based on the supraclavicular artery, which originates from the transverse cervical. After raising the flap, it may be tunnelled subcutaneously to reach the cheek, though care should be taken to avoid pressure on the pedicle. The deltopectoral is supplied by perforators from the internal mammary artery, though the distal part is supplied by branches of the thoracoacromial trunk which are cut during raising of the flap. For this reason a delay prior to transfer of the flap is required to ensure adequate vascularization of the distal part of the flap.⁴⁵

Skin grafting

Use of skin grafts to close a cheek defect is not preferred, as they generally provide a poor match for colour and skin texture of facial skin. Furthermore, secondary contracture may result in distortion which may not be desired, especially in proximity to eyelid, nose and lip.¹ Nevertheless, there is a place for skin grafting for patients with large defects not amenable to direct closure or a local flap, and in whom a more complex reconstruction may not be practical, such as the elderly or those with significant comorbidities. Full-thickness skin grafts are generally preferred as they are less likely to contract and have a better match for colour, texture and thickness. Common donor sites for facial defects are the preauricular and supraclavicular areas, and the nasolabial fold.⁴⁶ Due to their disadvantages, split-thickness skin grafts are generally only

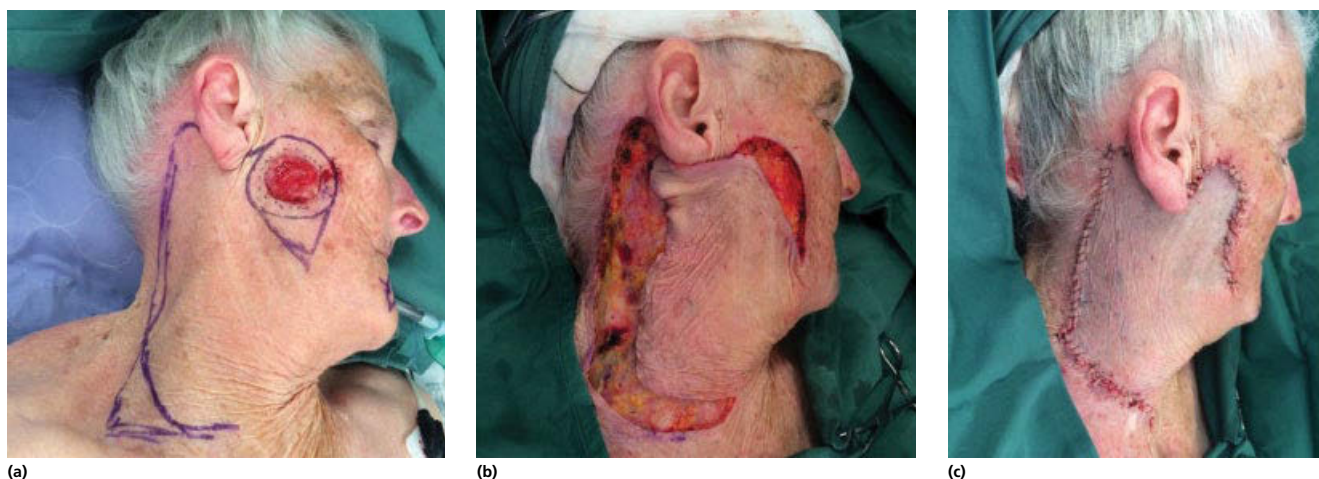


Figure 30.5 (a) Cervicofacial flap, preoperative marking. (b) Intraoperative appearance following raising of flap. (c) Postoperative appearance.

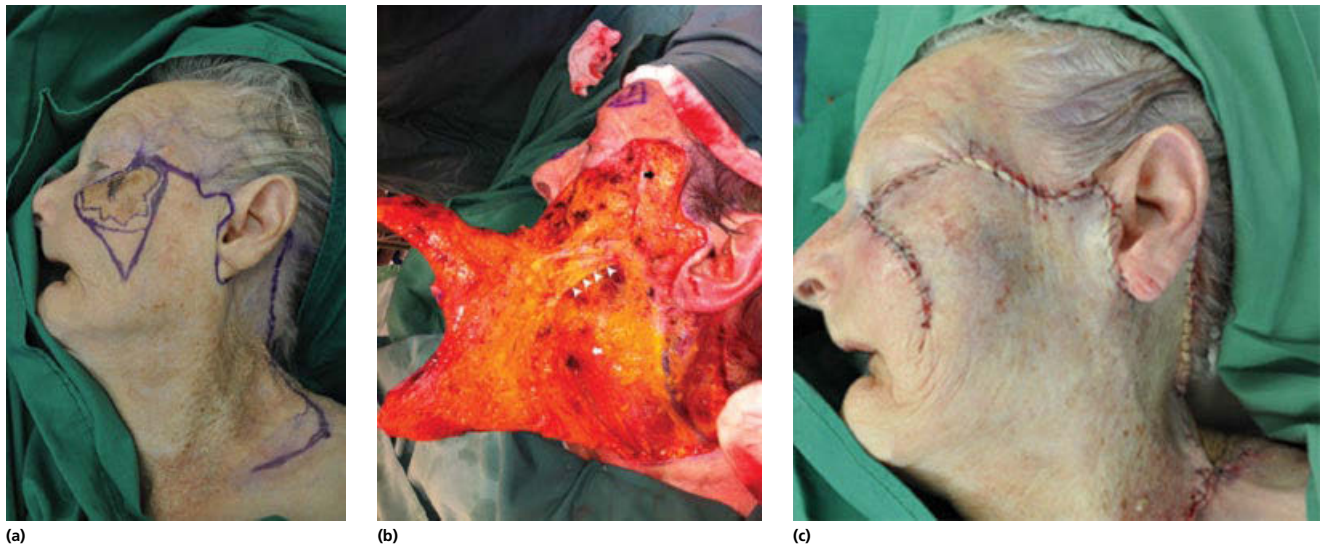


Figure 30.6 (a) Cervicofacial flap, preoperative marking. (b) Flap raised, demonstrating subplatysmal plane in neck, facial nerve branches emerging from parotid and subcutaneous dissection into temple (note that this image is taken from a different procedure to the other images in this series). Black arrow: subcutaneous dissection into temple. White arrow: subplatysmal dissection into neck. White arrowheads: facial nerve branches emerging from parotid. (c) Postoperative appearance.

used for temporary coverage while a definitive reconstruction is being planned, or in situations where oncologic recurrence is a possibility.

Free flaps

The most advanced option for reconstruction of very large defects, or in patients in whom other reconstructive options are not viable, is free tissue transfer. Suitable flaps include the radial forearm flap⁴⁷ and anterolateral thigh flap;⁴⁸ for patients requiring a bony element to their reconstruction, the osteocutaneous free fibular flap is particularly useful for reconstructing mandible.⁴⁹ Myocutaneous flaps based on rectus abdominis or latissimus dorsi may also be useful, though their use may result in a bulky reconstruction requiring significant revision.

Prefabrication

The complexity of soft tissue defects of the face has resulted in some novel techniques being developed. Two such techniques are prefabrication and prelamination. Pribaz has written extensively about the use of prefabricated and prelaminated flaps in head and neck reconstruction.^{50–52} He has defined prefabrication as the ‘pre-transfer implantation of a non-native vascular pedicle into tissue desired for reconstruction’, while prelamination describes the ‘pre-transfer implantation of anything else’.⁵² Thus prefabrication is a technique allowing the transfer of otherwise non-transferable tissue, and prelamination is the assembly of different types of tissue to reconstruct a composite defect prior to transfer. The technique of prefabrication has been named the ‘crane principle’, as the implanted pedicle acts as a ‘vascular crane’, a contrivance for lifting tissues from one

Table 30.2 Complications of cheek reconstruction

Surgical	Wound complications
	Flap necrosis
Functional	Oral incompetence
	Eyelid ectropion
Aesthetic	Loss of symmetry
	Loss of skin quality
	Abnormal hair distribution

location to another.⁵¹ One disadvantage of these techniques is the prolonged period required for the reconstruction: by their nature they require at least two and perhaps several staged procedures. However, it is a technique that may be helpful where simpler alternatives are not viable, as in extensive burns or trauma.⁴⁵

Complications

Complications of cheek reconstruction may be divided into surgical, functional, and aesthetic complications (Table 30.2). Surgical complications include wound complications such as breakdown and infection and flap necrosis, which may be subdivided into superficial, partial and total necrosis according to the area of tissue involved. It is generally acknowledged that the rate of surgical complications is vastly increased in smokers or those with vascular disease.^{3,53} Functional complications, such as oral incompetence and lower eyelid ectropion, result from displacement of the angle of the mouth or the lateral canthus due to excessive flap tension, swelling or weight. These complications

may be avoided by careful flap design and the use of strategically placed deep sutures to fix the flap to underlying structures.² Finally, aesthetic complications such as loss of symmetry, loss of skin quality and abnormal hair distribution generally result from inappropriate selection of tissue used for the reconstruction. Revisional procedures may be required to improve the final result.

Contour defects

Some patients may present with a complex deformity resulting from a condition causing widespread facial atrophy. Since the predominant contour of the cheek is that of a smooth convex surface, this has a significant effect on facial appearance, producing obvious asymmetry when unilateral, or a 'starved' appearance when bilateral.^{54–56} The eponymous names for the unilateral and bilateral syndromes are Parry–Romberg^{57,58} and Barraquer–Simons,^{55,59} respectively. These have been described previously in this compendium. A number of techniques may be used in the treatment of widespread facial atrophy. These include the use of autologous tissue, as in fat grafting⁶⁰ or free flap surgery,^{54,55} or synthetic material such as filler and bone graft substitute.^{56,61} Synthetic fillers that have been used for hemifacial atrophy include biodegradable materials such as hyaluronic acid gel, and non-biodegradable materials such as silicone.^{62,63} However, due to concerns regarding long-term complications, use of silicone for treatment of facial atrophy has fallen out of favour.^{57,64}

It is essential to recognize the existence of two distinct problems underlying the deformity in facial atrophy: a paucity of soft tissue volume and an abnormal bony contour. Frequently, reconstruction of the bony contour by will be required in addition to restoration of soft tissue volume.⁶⁵ A variety of free flaps have been used for soft tissue restoration in facial atrophy, including omentum,⁶⁶ transverse rectus abdominis myocutaneous (TRAM),⁶⁷ scapular and parascapular⁶⁸ and groin flaps,⁶⁹ to name a few. Ultimately, the choice of technique will be determined by the degree of atrophy and the volume of tissue required, and risk of morbidity associated with the chosen reconstruction compared with the effect of the deformity on the patient's quality of life.

Conclusion

The approach to reconstructing a cheek defect should be tailored to the individual patient. Assessment of the patient's overall health, in addition to the characteristics of the defect, is essential in order to select an appropriate reconstructive technique. Detailed understanding of the facial anatomy and aesthetics are vital to the creation and execution of a sound plan. Taking these basic principles into consideration allows the surgeon to achieve harmonious aesthetic and functional results in reconstructing the full range of cheek defects.

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CHAPTER 31

Lip reconstruction

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The lips constitute the main feature of the lower third of the face, and have been a focus of interest by reconstructive, cleft and aesthetic surgeons. Functionally, the lips play many important roles – articulation and enunciation in speech, maintenance of oral competence during eating, drinking and forceful blowing, and expression of emotion and kissing. These roles are possible because, unlike the other elements of the face, the lips are dynamic and mobile. Aesthetically, the vermillion is the hallmark. Reconstruction of the lips, therefore, aims to maintain these characteristics both for function and appearance. This chapter reviews the anatomy of the lips and presents an algorithm for reconstruction.

Anatomy

Embryology

The upper lip is derived from two distinct structures, namely the paired maxillary and nasomedial processes. Fusion of these processes gives rise to the characteristic features of the upper lip, such as the philtral columns, philtral dimple, Cupid's bow and white roll. The lower lip is formed from fusion of the paired mandibular processes, which result in a less defined lower lip. The topographical landmarks of the lip are illustrated in Figure 31.1.

Musculature

The muscles that animate the lips are illustrated in Figure 31.2. The principal muscle is the orbicularis oris, which forms a complex sphincter muscle. This muscle arises both from intrinsic fibres of the lip and from other muscles that insert into the lip. The deep and oblique fibres of orbicularis oris approximate the lips to the alveolar arch, while the superficial fibres purse and protrude the lips. The orbicularis oris can be dissected from its supportive muscles while preserving its viability, sensation and motor function, an important consideration in the choice of reconstruction.¹

Levator labii superioris elevate the upper lip, levator anguli oris and zygomaticus major draw the lateral upper lip up and back, while buccinator and risorius clear the gingival sulcus. The lower lip is depressed and retracted by the platysma, depressor anguli oris and depressor labii inferioris. The mentalis muscles cause elevation and protrusion of the central aspect of the lower lip. Loss of these muscles leads to lip incompetence and incisor show.

Vasculature

The blood supply to the upper and lower lips is the superior and inferior labial arteries respectively. These originate from the facial artery from either side of the face. The average distance of the superior labial artery from the oral commissure (as it branches off the facial artery) is 12 mm, and can be found along its course within 10 mm from the inferior border of the lips. However, the anatomy of the labial arteries can be variable. Magden *et al.* reported that 29% of superior labial arteries have a unilateral origin.³ This finding has important implications when raising pedicled flaps such as the Abbe or Estlander flaps. The superior labial artery has a mean diameter of between 1 and 2 mm at its origin, and is therefore large enough for microsurgical anastomosis.^{4,5} There are numerous reports and case series of lip replantation in the literature, and problems attributed to replantation are mainly related to the venous drainage of the replanted tissue.

The origin of the inferior labial artery varies between the lower margin of the mandible and the oral commissure.

Lymphatic drainage

The lymphatic drainage of the lips is ipsilaterally into the submental, submandibular and parotid nodes. Tissues in the midline drain bilaterally. Submental lymph nodes then drain into ipsilateral submandibular nodes. Submandibular and parotid nodes secondarily drain into the ipsilateral jugulodigastric lymph nodes.

Innervation

The facial nerve (VIIth cranial nerve) provides the motor innervation to the perioral musculature via its buccal and marginal mandibular branches. There are interconnections between both branches of the facial nerve distal to the parotid gland. The motor nerves are located on the undersurface of all the perioral musculature except for buccinator, levator anguli oris and mentalis, where the nerve lies on the superficial surface of these muscles.

The maxillary and mandibular branches of the trigeminal nerve (Vth cranial nerve) provide sensory innervation to the perioral region. Specifically, the infraorbital nerve, which is a terminal branch of the maxillary nerve, innervates the upper lip. The infraorbital foramen faces downward and medial and lies 4–7 mm below the infraorbital rim, on a vertical line that descends from the medial limbus of the iris. The nerve runs beneath the levator labii superioris and superficial to the levator anguli oris to supply the lateral nasal sidewall, ala, columella, medial cheek and upper lip. The lower lip, down to the labio-mental fold, is innervated by the mental nerve, which originates from the inferior alveolar nerve, a branch of the mandibular nerve. The mental foramen is located below the apex of the second mandibular bicuspid with 6–10 mm of lateral variability.

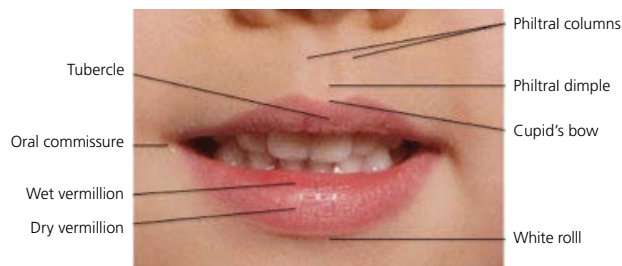


Figure 31.1 Topographical anatomy of the lips.

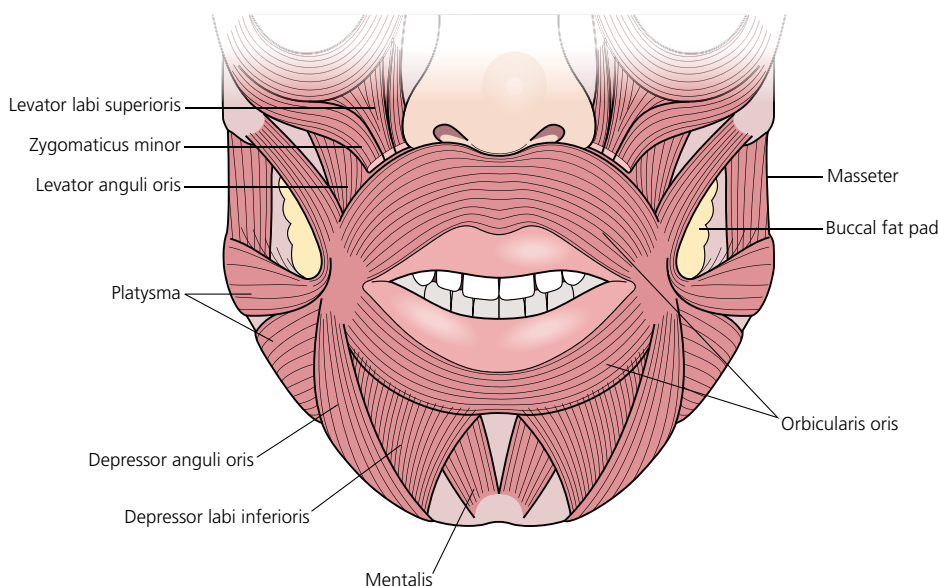


Figure 31.2 Lip musculature.

Anaesthesia

Surgical procedures to the perioral region should be preceded by a regional block, followed by local infiltration to the affected area for the adrenaline-facilitated vasoconstriction. Prior to local infiltration of the lip tissue, it is important to mark or tattoo the white roll of the lip either side of the planned resection in order to match this up afterwards. Any local infiltration, particularly with adrenaline, will obliterate the white roll. A 1 mm discrepancy between the vermillion and skin is noticeable at conversational distance.

The entire upper lip can be anaesthetized by bilateral infraorbital nerve block. This can be carried out either percutaneously or transorally.⁶ A 25- to 27-gauge needle is used and does not need to pass into the foramen to desensitize the lip. For the percutaneous route, the needle is placed medial to the nasolabial fold but lateral to the alar base. For the transoral route, the needle is passed transmucosally above the canine eminence.

The lower lip is anaesthetized by bilateral mental nerve blockade. This is performed intraorally by rolling the lower lip outward and stretching the mucosa to visualize the mental nerve just below the mucosa at the canine root. If the nerve is not readily visible, the surgeon should infiltrate local anaesthetic into the submucosa at the apices of the canines into the lower lateral buccal sulcus.

Goals of reconstruction

The three main goals of lip reconstruction are:

- preservation of oral continence – this requires a muscular integrity, normal sensation and an adequate oral aperture;
- preservation of function for speech and ingestion of food: and
- cosmesis.

Aetiology of lip defects

The commonest indication for lip reconstruction is following tumour extirpation. The lower lip is much more frequently affected by neoplasia compared to the upper lip, presumably due to greater actinic exposure. The vast majority of skin cancers are squamous cell carcinoma in origin and are more frequently encountered in males (2.4 times) as a result of greater sun exposure, alcohol and tobacco.⁷ Fortunately, these lesions often involve the area between the midline and the oral commissure, avoiding the need for commissural reconstruction. Other causes of lip defects include trauma, infection (e.g. noma), vasculitis and congenital causes such as vascular malformation or clefts.

Analysis of the defect

Before embarking on lip reconstruction, the site, size, depth and involvement of commissure all need to be contemplated.

Site

The upper lip has more topographical landmarks than the lower lip. The lower lip, lacking in features, is theoretically more expendable as donor tissue for reconstruction. However, functionally and socially, the lower lip deserves more consideration. The lower lip serves to maintain oral competence by containing solids and liquids within the oral cavity. Therefore, any reduction in sulcus depth, compounded by loss of sensation and poor muscle function will result in liquids leaking out of the mouth.

Hence, the surgeon needs to deliberate carefully in the following situations: when using lower lip tissue to reconstruct the upper lip, denervating the lower lip during the raising of local flaps, or the use of insensate and non-functional tissue such as the nasolabial or cheek tissue to reconstruct the lower lip.

Size

Although the absolute size of the tumour and excision margins is important, the percentage of the lip that has been ablated is crucial in deciding the reconstructive option. As previously mentioned, the lower lip is more expendable, especially in elderly patients with more laxity in the tissues, thereby permitting transposition, advancement or rotation flaps more readily. However, it is vital not to leave the patient with a microstomia. Although amenable to stretching, microstomia does result in imminent problems with dental care, insertion and removal of dentures.

Depth

The four layers of the lip from the oral cavity outwards are mucosa, muscle, vermillion and skin. With regard to the depth of the defect, the surgeon should 'replace like with like' and consider the reconstructive options accordingly.

Involvement of commissure

If the lip defect involves the oral commissure, then effort must be made to reconstruct the commissure as this is an important aesthetic landmark of the lips and face.

Algorithm for reconstruction

Vermillion defects

The vermillion is a unique feature of the lip. The vermillion is the transition zone between the stratified keratinized squamous epithelium of the skin and the non-keratinized squamous epithelium of the oral mucosa. This exists only in humans, and is the partially keratinized layer, which is red due to the reflection of the underlying blood vessels. The wet and dry vermillions are distinct; the dry vermillion tends to take on lipstick more readily.

For small lesions confined within the vermillion, the lesion should be excised as a vertical ellipse and sutured directly, avoiding extension of the incision onto the skin. This technique can also be employed for notch or whistle deformities of the lip, with the addition of a Z-plasty to avoid recurrence.

Large superficial defects confined to the vermillion can be left to heal by secondary intention, and tend to heal within 3–4 weeks with a good cosmetic result.

For full-thickness defects resulting in a deficiency of less than 50% of the total vermillion, the options include the axial musculo-vermillion advancement flap described by Goldstein⁸ or musculo-mucosal V to Y advancement flaps.⁹ For the axial musculo-vermillion advancement flap, the position of the labial artery is identified from the resection of the lesion and the flap is elevated deep to the labial artery, and then advanced into the defect (Figure 31.3). Although cross-lip flaps for vermillion reconstruction have been widely reported in the literature,^{10–12} it is our opinion that they should be avoided. Cross-lip flaps should not be undertaken lightly as they not only compromise the other intact lip, but also are difficult to tolerate as they are executed as a staged procedure with an intervening period of lip adhesion (i.e. the pedicle of the flap attached).

The facial artery myomucosal flap (FAMM) described by Pribaz *et al.* in 1992¹³ is a good option for vermillion defects of more than 50%. This flap can also be used for total vermillion reconstruction by raising bilateral flaps. The FAMM flap is a composite flap, which is raised from the lateral cheek, based on the facial artery as it courses though the cheek lateral to the buccinator muscle. It consists of mucosa, submucosa, a small amount of buccinator, a segment of the facial artery and venous plexus. It may be used as a superiorly based flap, which has retrograde perfusion via the angular artery, for the upper lip. Alternatively, the inferiorly based flap based on the facial artery is used for the lower lip. Wide flaps, up to the entire width of the cheek, and up to 7 or 8 cm in length, can be raised safely but the authors advocate thin flaps to reduce donor site morbidity (Figure 31.4).

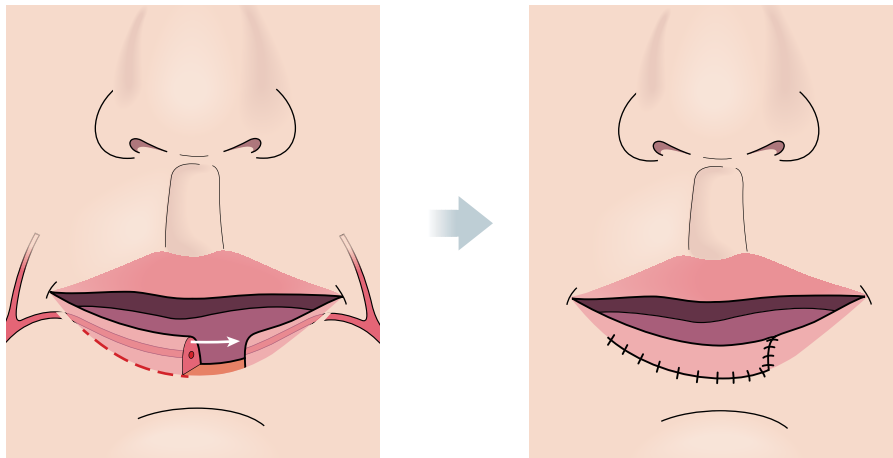


Figure 31.3 The axial musculo-vermillion flap for full-thickness vermillion defects less than 50% of the total vermillion. The flap is elevated deep to the labial artery.

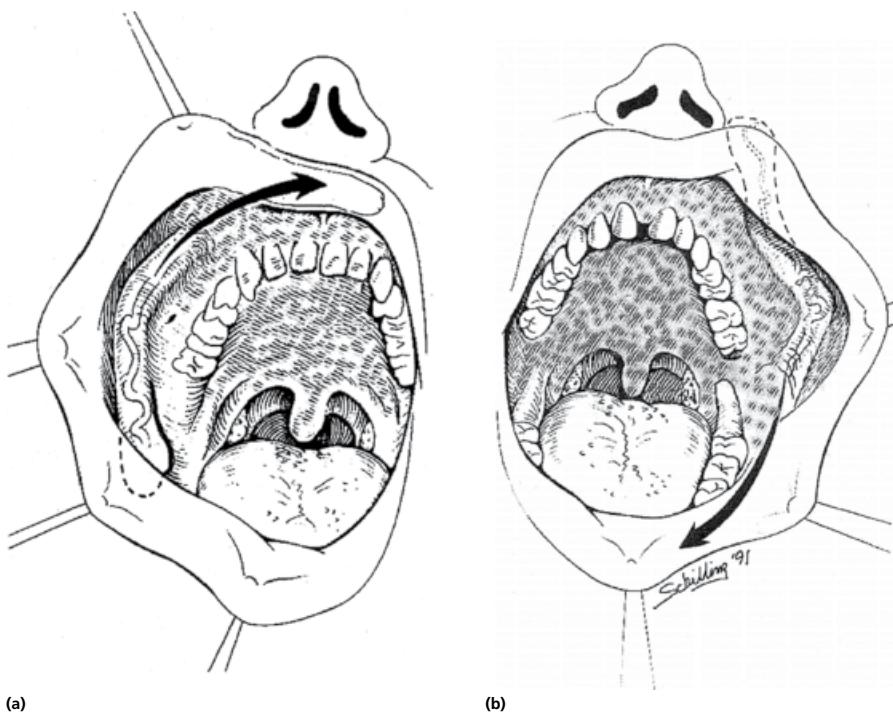


Figure 31.4 Illustrations of the superiorly based (left) and inferiorly based (right) facial artery myomucosal flaps (FAMM flaps) described by Pribaz. Source: Pribaz *et al.*, 1992.¹³ Reproduced with permission of Lippincott Williams & Wilkins.

Some patients undergo total vermillionectomy for severe actinic damage or unstable residual epithelium adjacent to a malignant lesion. In these cases of total vermillion deficiency, buccal mucosal advancement as a bipedicle flap described by Lustig *et al.*¹⁴ can be raised to resurface the defect. An incision is made in the buccal sulcus, and the bipedicle flap is advanced as a myomucosal flap including the labial artery. The donor site is left to heal by secondary intention (Figure 31.5).

Full-thickness lower lip defect

In addressing full-thickness defects of the lower lip, the rule of thirds provides an effective algorithm for reconstruction (Figure 31.6).

Defects less than 1/3 width

For defects up to a third of the total width of the lower lip, the edges of the defect can be approximated directly. It is important to carry out primary closure according to the layers; that is, the lining (buccal mucosa), the muscle and the cover (vermillion and skin). The buccal mucosa is sutured using an absorbable suture (e.g. polyglactin/Vicryl® or Vicryl Rapide®). The orbicularis oris muscle is also approximated with an absorbable suture (e.g. Vicryl, Monocryl® or PDS®/polydioxanone). The vermillion is sutured using an absorbable suture such as Vicryl Rapide.

As previously mentioned, prior to infiltration of any local anaesthetic agent, the vermillion junction and/or the white roll must be carefully marked to facilitate accurate approximation of the edges. Techniques for marking the vermillion junction or the white roll include tattooing or placing a single suture at the

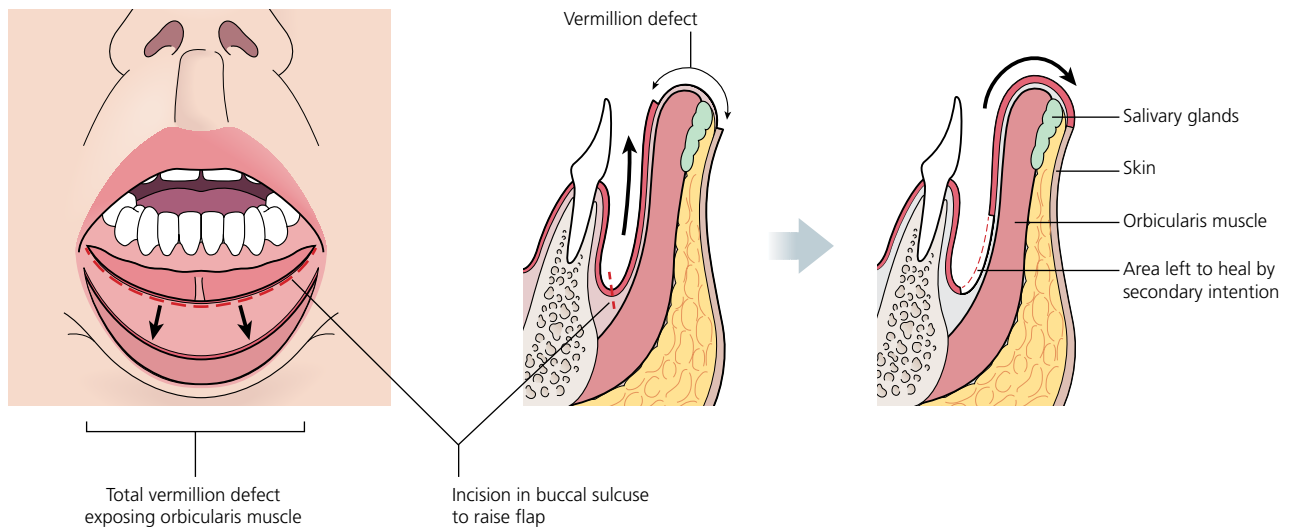


Figure 31.5 A schematic diagram of the bipedicle myomucosal advancement flaps described by Converse for total vermilion reconstruction. The donor defect is left to heal by secondary intention. Source: Pribaz et al., 1992.¹³ Reproduced with permission of Lippincott Williams & Wilkins.

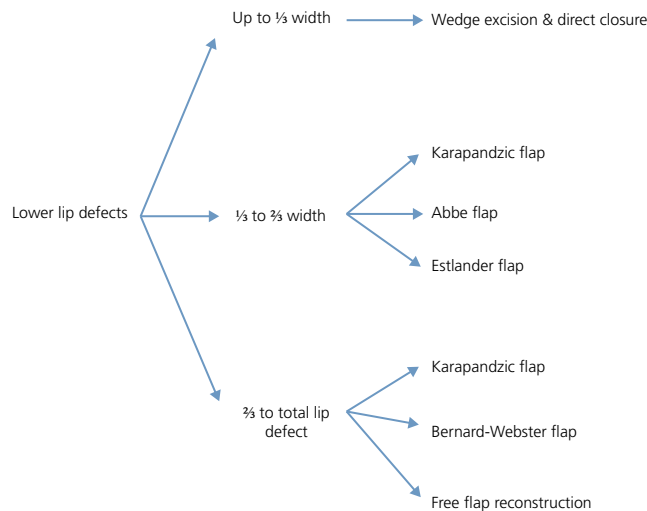


Figure 31.6 The rule of thirds as an algorithm for reconstruction of full-thickness lower lip defects.

corresponding points either side of the defect. In some elderly patients, defects between a third to half of the width of the lower lip can be approximated, primarily due to increased laxity and redundancy of the tissues with age (Figure 31.7), however, for the majority of patients, defects greater than a third in width require flap reconstruction to avoid microstomia.

Defects between 1/3 and 2/3 widths

Defects between a third and two thirds of the width of the lower lip will require reconstruction with a flap. For these defects, local flaps offer the best option. The three main choices are the Karapandzic flap, Abbe flap and Estlander flap.

The *Karapandzic flap*, first described in 1974,¹⁵ is a rotation-advancement flap, which re-establishes the circumoral sphincter. The Karapandzic flap contains skin, innervated orbicularis oris

and mucosa, and can be used to reconstruct both upper and lower lip defects (Figure 31.8). The key element in raising this flap is to translate the vertical height of the lip defect to the width of the flap. Curvilinear incisions are made through skin and muscle, releasing the muscle fibres and suspensory ligaments but preserving the superior and inferior labial arteries and buccal branches of the facial nerve. The mucosal incisions are more limited, placed in the buccal sulcus long enough simply to enable advancement of the musculocutaneous component of the flaps. In the superiorly based Karapandzic flaps for lower lip defects, the mucosal incisions should only extend up to the mental nerve at the canine root in order to preserve sensation to any residual lower lip tissue. Limited mucosal incisions also prevent effacement of the buccal sulcus, which can lead to salivary incompetence.

The Karapandzic flap should be the first choice of reconstruction for moderate-sized defects as it is innervated, dynamic and therefore functional. The main disadvantage of the Karapandzic flap is the potential risk of microstomia, and the need for surgical revision to deepen the buccal sulcus if it is effaced.

The *Abbe flap*, described in 1898,¹⁶ is a staged lip-switch flap which provides effective reconstruction of defects of up to two thirds of the width of the lip not involving the oral commissure. The Abbe flap is raised from the junction of the middle and lateral thirds of the opposite lip, with a flap of approximately half the width of the defect but equal in height. Prior to raising the flap, the vermilion border on either side of the incisions (i.e. four points) are carefully marked to enable accurate closure of the donor site and inset of the flap. The flap is pedicled on the corresponding labial artery; its location can be identified on the free edge of the flap as the artery can be palpated. The pedicle is divided between 14 and 21 days later (Figure 31.9).

The main advantage of the Abbe flap is the ability to replace the vertical height of the defect and import vermilion

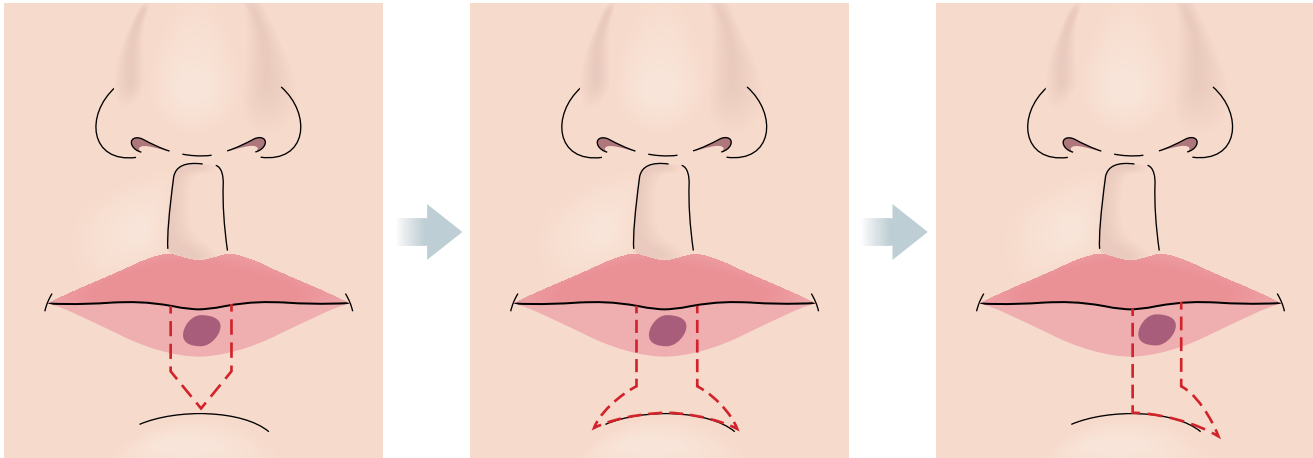


Figure 31.7 Variation in wedge excision and primary closure of lower lip defects.

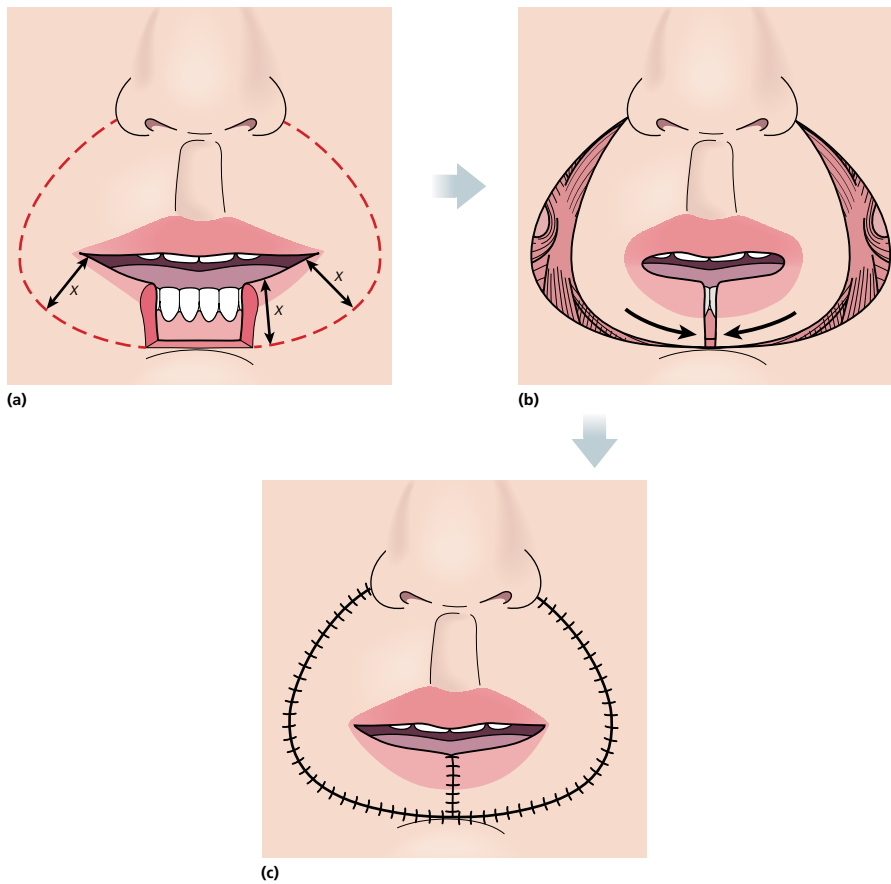


Figure 31.8 Schematic diagram of the Karapandzic flap. (a) The defect following tumour extirpation, note the height of the defect, x , is equivalent to the width of each flap. The mucosal incisions are more limited. (b) The flap raised, preserving the neurovascular bundles (*) to each flap. (c) The final appearance of the lips, with a smaller oral aperture.

tissue into the defect. However, the disadvantages include a period of lip adhesion, imbalance of the white roll and vermillion and the insensate segment of imported tissue. Furthermore, as previously mentioned, around a third of the population have a unilateral origin of the superior labial artery, which has implications on the pivot point of the flap (i.e. the pivot point should be more proximal where the calibre of the artery is larger).

The *Estlander flap*, described in 1872,¹⁷ is a single stage lip-switch flap for defects involving the oral commissure (Figure 31.10). The flap is based medially, and is rotated into the defect to reconstruct the commissure and lip. Similar to the Abbe flap, the flap is designed to be equal in height but half the width of the defect. The reconstructed oral commissure usually has an obtuse or blunted angle, and frequently requires revision by means of a commissuroplasty. This is described further below.



Figure 31.9 (a) Schematic diagram of the Abbe flap. The flap is raised at the junction of the middle and lateral thirds of the contralateral lip. The four points denoting the vermilion border either side of the incisions for the flap are marked. The width of the flap (x) is half that of the defect ($2x$), but the height of the flap (h) is equivalent to that of the defect. The flap pedicle containing the labial artery is left attached for 2–3 weeks before division. (b) Design of upper lip Abbe flap for recurrent squamous cell carcinoma following previous wedge resection. (c) Raised flap and nasolabial excisions at donor site. (d) Inset of the first stage Abbe flap. (e, f) Division and inset during second stage at 21 days. (g, h) 3 months post-second stage division and a month after ending postoperative radiation therapy cycle. Source: Photographs courtesy of Mr Ross Farhadieh. Reproduced with permission.

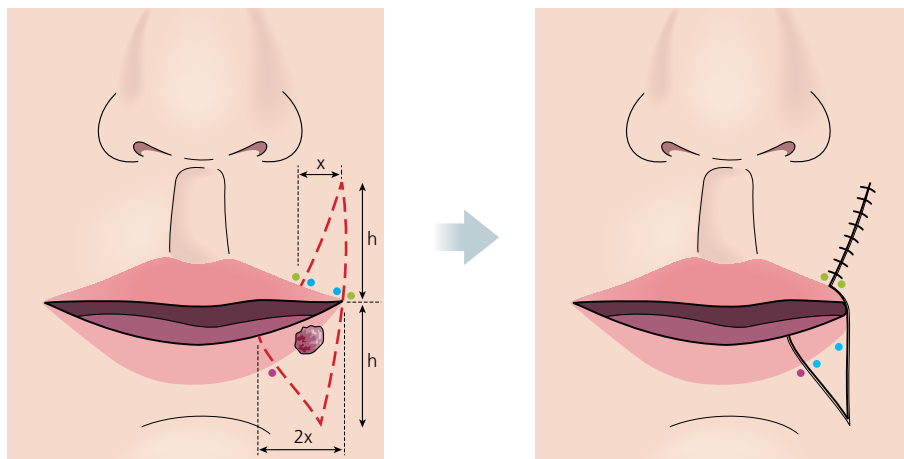


Figure 31.10 Schematic diagram illustrating the Estlander flap to reconstruct defects involving the oral commissure. The four points denoting the vermilion border either side of the incisions for the flap are marked. The width of the flap (x) is half that of the defect ($2x$), but the height of the flap (h) is equivalent to that of the defect. Note the blunted neo-commissure, which is common sequelae of this flap. This may require a commissuroplasty at a later stage.

Defects more than 2/3 width or total lip defect

Lower lip defects greater than two thirds of the total width can be addressed with local flaps in selected cases. The Karapandzic flap previously described can be used for defects up to 80% of the total width. The main advantage of this flap is that it is innervated and therefore dynamic. However, the disadvantage of the Karapandzic flaps is the resulting microstomia. Other local flaps, such as the Gillies fan, McGregor and Nakajima flaps, have been described for large defects of the lower lip but these flaps are largely obsolete in our practice. The Gillies fan flap distorts the oral commissure, the McGregor and Nakajima flaps reconstruct the lip but the muscle fibre direction is changed and a new vermilion is required.

The *Bernard flap*, described in 1853,¹⁸ with the Webster modification provides the best reconstructive local option for total lip defects. The original Bernard operation involves a triangular wedge excision of the whole lower lip. The incision is extended laterally from both oral commissures with full-thickness triangular excision at the nasolabial area to enable direct closure of the lower lip wedge (Figure 31.11). The Webster modification, described in 1960,¹⁹ employs the principles of:

- quadrilateral or rectangular resection;
- medial advancement of one or two cheek-lip flaps;
- excision of skin and subcutaneous tissue only for removal of tissue excess;
- minimal incisions through the muscle laterally;
- buccal mucosa flaps for vermilion replacement; and
- Z-plasties on all vertical suture lines.

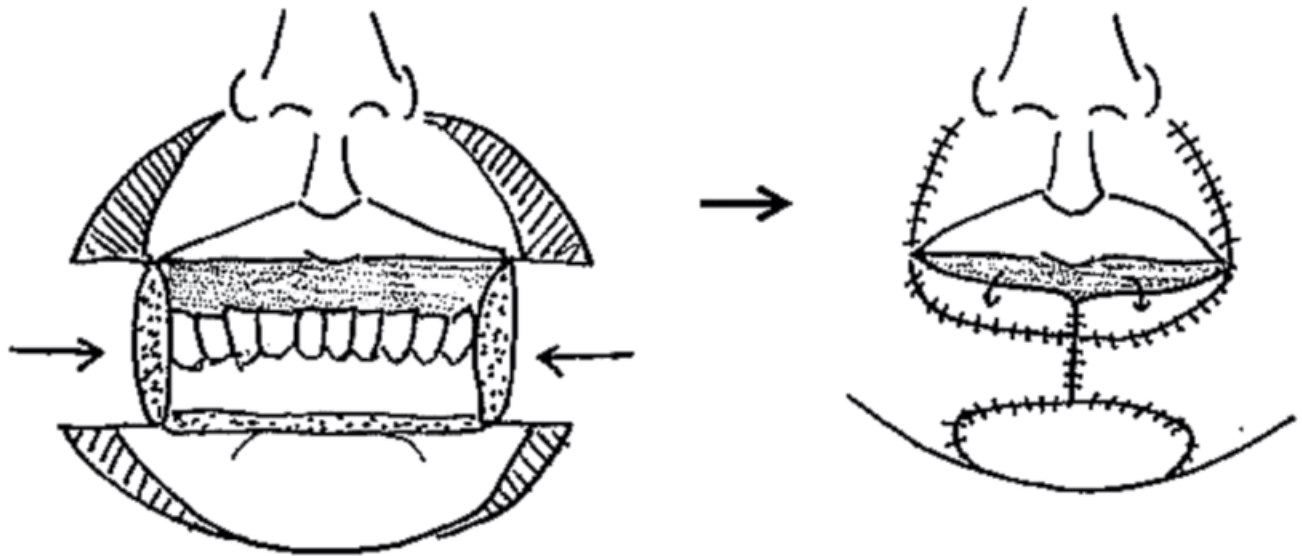
Hence, the planning of the Bernard–Webster flap begins with the cheek advancement flaps. The length of each advancement flap is equivalent to half the length of the horizontal lip defect. Then, superior curvilinear incisions are made along the nasolabial fold, integrated into Burrow's triangles, to enable advancement of the horizontal cheek advancement flaps. Inferiorly, incisions are made along the labiomental fold, also incorporated into Burrow's triangles. These incisions are deepened through skin, subcutaneous tissue and muscle to the level of the mucosa. The mucosa is only incised medially to allow advancement of

the cheek tissue, preserving as much of the intraoral lining as possible. These medial incisions are also placed superior to the skin flap design to allow mucosa to be delivered anteriorly and everted to create the vermilion of the reconstructed lip. Finally, the medial edges of each flap are inset with a three-layer closure. There is a relative contraindication for this flap in patients who have had previous facial surgery, or neck dissections resulting in ligation of the facial artery, thus compromising the cutaneous circulation. The other disadvantages of this flap are the potential for microstomia, notching of the central lip and effacement of the buccal sulcus, leading to oral incompetence.

The other less favoured option for total lip reconstruction is free tissue transfer. Whenever possible, local tissue must be used to reconstruct the lips. However, this may not be possible due to the need to reconstruct composite defects involving the whole lip and adjacent regions such as the chin and cheeks, the lack of suitable local tissue as the result of previous surgery or radiotherapy, and finally the increasing proficiency of plastic surgeons in performing microsurgical reconstruction. Care and attention should be paid when deciding on the choice of free flap reconstruction. Factors to take into consideration include skin colour match, the volume required to fill the defect, the ability to contour the flap to fit the defect, and the feasibility of suspending the flap to prevent ptosis.

Surgeons have proposed a multitude of flaps for lower lip reconstruction. The list includes radial forearm, anterolateral thigh, parascapular, rectus abdominis²⁰ and lateral arm flaps. These flaps can either be used in isolation, in combination with another flap either locally based or free tissue transfer, or pre-lamination of a free flap. The radial forearm free flap is the most popular choice because it provides a good colour match, is thin and therefore can be easily folded, has a long pedicle, can be harvested simultaneously during the oncological resection of the lower lip or preparation of neck recipient vessels, and can be harvested with the palmaris longus tendon as a static sling to suspend the lower lip reconstruction (Figure 31.12).

When undertaking free flap reconstruction, the first step involves obtaining a template of the defect. The surgeon needs to



(a)



(b)



(c)



(d)

Figure 31.11 (a) The Bernard-Webster flap demonstrating the quadrilateral or rectangular defect following tumour extirpation, medial advancement of the two cheek-lip flaps, excision of skin and subcutaneous tissue only for removal of tissue excess (hashed area), minimal incisions through the muscle laterally and buccal mucosa flaps for vermilion replacement. (b, c, d) Photos of a modified Webster-Bernard flap for a total lower lip reconstruction following excision of recurrent squamous cell carcinoma. Source: Photographs courtesy of Mr Ross Farhadieh. Reproduced with permission.



Figure 31.12 Total lower lip reconstruction with radial forearm free flap. The flap has been suspended over a palmaris longus graft which was secured to the zygomatic arches.

be aware that the defect usually appears larger than the actual size of the tissue lost. This is due to the pull of the perioral and facial mimetic muscles, which retract the edge of the defect laterally. Thus, it is important to place tension on the edges of the defect medially before obtaining the template. Once the free flap has been raised, inseting the flap is another crucial process. The tension of the flap needs to be taut enough to prevent ptosis leading to salivary incompetence, but not too tight resulting in compromise to flap perfusion. Attention must also be directed towards the depth of the gingivobuccal sulcus, ensuring adequate depth to prevent drooling. If a static sling is placed simultaneously to suspend the flap, the sling is attached to a relatively stable structure, and the senior author prefers the periosteum of the zygomatic arch. Some authors advocate attaching the sling to remnants of zygomaticus major muscle to obtain a measure of dynamic movement.

The disadvantage of free flap reconstruction is that both the tissue and sling can relax with time, leading to ptosis, and oral incompetence. Furthermore, the results are aesthetically inferior to local flaps.

Full-thickness upper lip defect

We previously alluded to the topographical difference between the upper and lower lips, and due consideration has to be given to the philtral columns and Cupid's bow when planning the reconstruction. In men, the hair-bearing area of the upper lip is a further confounding factor in choice of reconstruction.

Similar to the lower lip, the rule of thirds is also used for the upper lip. Figure 31.13 illustrates a useful algorithm for upper lip defects.

Defects less than 1/3 width

Upper lip defects that do not involve the vermillion can be reconstructed with a full-thickness skin graft or local flaps such as the nasolabial flap. In men, hair-bearing tissue from the temporal scalp^{21–23} provides a reconstructive option which restores the moustache.

Lateral defects located between the philtral columns and the oral commissures are closed directly as for the lower lip,

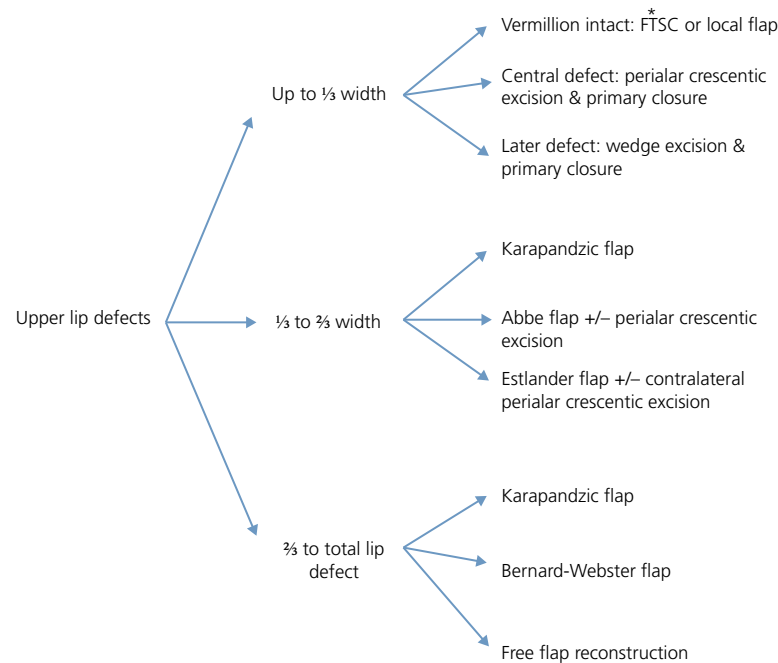


Figure 31.13 The rule of thirds is a useful algorithm for reconstruction of full-thickness defects of the upper lip. *FTSG = full-thickness skin graft

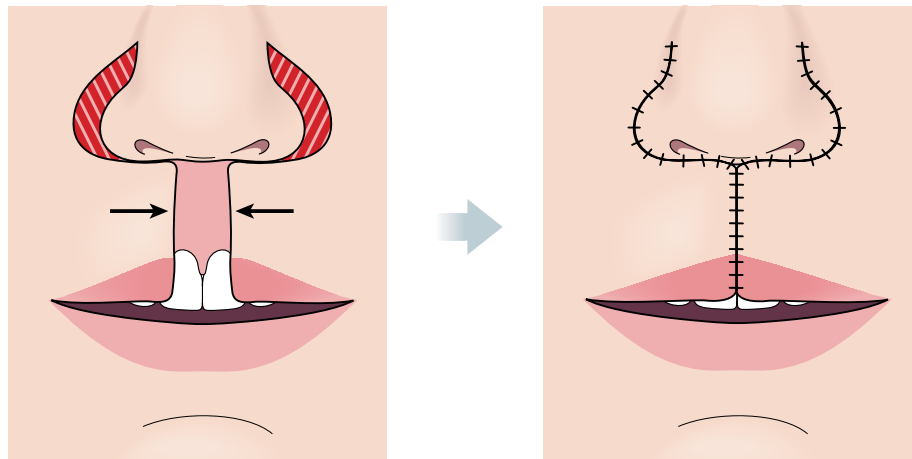


Figure 31.14 Schematic diagram illustrating the Webster's perialar crescentic advancement flaps. The hashed area denotes skin excision only.

ensuring accurate approximation of the white roll and vermilion border. Central defects involving the philtral columns and Cupid's bow are also closed directly, but a useful adjunct is Webster's perialar crescentic excision and bilateral cheek advancement flaps (Figure 31.14).

Defects between 1/3 to 2/3 widths

As for the lower lip, the Karapandzic flap remains the first choice for local flap reconstruction of the upper lip, as it is dynamic and innervated, thereby providing a functional reconstruction. The alternatives are the Abbe and Estlander flaps. The former is employed for central defects, and the latter for defects involving the oral commissure. The perialar crescentic excision is a useful adjunct for the Abbe flap in reconstructing central

defects, and can also aid in closure of lateral defects together with the Estlander flap (by means of a contralateral perialar crescentic excision).

Defects more than 2/3 width or total lip defect

The same algorithm as for the lower lip applies for the upper lip (i.e. the Karapandzic flap, Bernard-Webster flap or free tissue transfer).

Commissuroplasty

Commissuroplasty is indicated in patients with microstomia, or a blunted oral commissure following lip reconstruction with local flaps such as the Estlander or Karapandzic flaps.



Figure 31.15 Left commissuroplasty following an acid burn. The right had been previously released.

In these patients, wherever possible, the vermillion is utilized either as advancement or transposition flaps. If the vermillion is too small or inadequate, then the buccal mucosa is used instead or in combination. Several methods have been modified from Dieffenbach's approach in 1831,²⁴ whereby a triangular wedge of scar tissue is excised and superior, lateral and inferior mucosal flaps are advanced to resurface the corner of the mouth.

A popular contemporary method is *Converse's commissuroplasty*, described in 1959 (Figure 31.15).²⁵ A triangular area of skin is excised to accommodate the elongated commissure. The buccal mucosal flaps are designed as shown in Figure 31.14, incised and advanced to form the neo-vermillion.

Conclusion

Lip defects can be approached with a stepwise algorithm ranging from primary closure to free tissue transfer. This chapter has by no means provided a comprehensive catalogue of the reconstructive

options available. We have not explored the surgical techniques for congenital lip defects, such as cleft lips, craniofacial clefts, macrostomia or microstomia; nor have we elaborated on the indications and techniques of face transplantation. However, as surgeons, it is imperative we adhere to the basic principles of replacing like with like, and focus on both function and form for lip reconstruction.

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CHAPTER 32

Nasal reconstruction

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Introduction

The social stigma of nasal amputation for the patient represents a huge challenge for the reconstructive surgeon to achieve the good results that are possible today. Totally different from modern technical exploits such as face transplants or supermicrosurgery, the majority of cases can be performed by general plastic surgeons without the need for specialist equipment and training. However, the key to success is preoperative examination and planning.

Through the ages, nasal reconstruction has followed a slow but regular improvement due to advances in technique (the first forehead flap was recorded in the *Sushruta Samitha* (500 BC)¹), and closer attention to detail. Thus, results have become progressively more and more acceptable so that now in good hands excellent aesthetics may be expected.

Of all the surgeons who actively participated in the advancements in nasal reconstruction, we particularly thank Drs John Marquis Converse, David Ralph Millard Jr, Gary C. Burget and Frederick J. Menick.

This is a broad topic. For the interested reader, we recommend the book by Burget and Menick² and the more recent publication by Menick.³

Surgical anatomy of the nose

The nose is a composite tissue structure composed of three layers: the nasal skeleton, an internal lining of mucosa and an external cover.

Nasal skeleton

The nasal skeleton is the supporting structure of the nose. It consists of the nasal bones and ascending processes of the maxilla in the upper one third, the paired upper lateral cartilages in the middle third, and the lower lateral cartilages (or alar) in the lower one third.

The upper lateral cartilages are attached tightly to the caudal edge of the nasal bones and the dorsal border of nasal septum,

the 'keystone area', allowing them to remain suspended above the nasal cavity. At the junction of septum and upper lateral cartilages, the dorsal border of the septum widens to form a 'T'. This broadening is crucial since it maintains the aperture of the internal valve during nasal inspiration.

The paired alar cartilages support the lower third of the nose by uniting to form a tripod configuration as first described by Anderson.⁴ The paired medial crura form the central leg of the tripod and are attached to the anterior nasal spine and septum in the midline. The lateral crura compose the two lateral legs of this tripod, and they are attached to the pyriform aperture. Between lateral and medial cruras, the dome defines the apex of the alar cartilage. It supports the nasal tip and is responsible for the light reflex of the tip. The region of fibrous connection of alar cartilages with upper lateral cartilages is known as the 'scroll' area. The normal ala does not contain cartilage but a compact stiff fibrofatty tissue. Its reconstruction has to include stiff tissues, however, to prevent collapse and retraction.

Nasal lining

The nasal lining consists of a thin layer of vascular mucosa. It tightly adheres to the deep surface of the nasal skeleton. Mobility of the mucosa is limited by this dense adherence. Consequently only the smallest mucosal defects (<5 mm) can be closed primarily.

The anterior septal branch of superior labial artery, medial internal nasal branch of anterior ethmoid artery, septal branch of posterior ethmoid artery, posterior septal branch of sphenopalatine artery and greater palatine artery supply nasal mucosa (Figure 32.1). Anterior septal branch of labial artery and medial internal nasal branch of anterior ethmoid artery may be used for lining flaps as described below.

Nasal cover

From deep to superficial, nasal skeleton is covered by perichondrium/periosteum, areolar tissue plane, musculoaponeurotic plane, subcutaneous layer (a few millimetres of fat), dermis and epidermis.

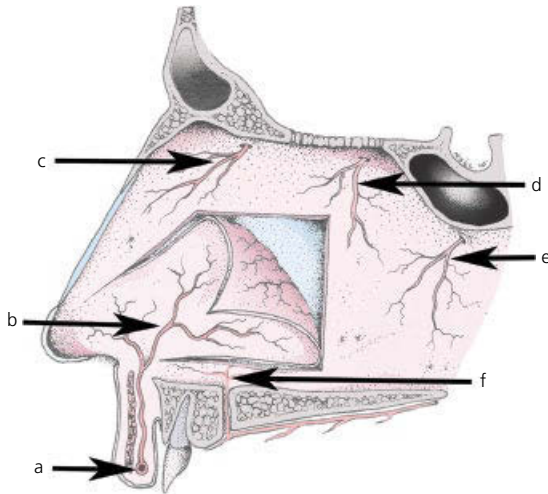


Figure 32.1 Vascularization of septal mucosa (+ elevation of an ipsilateral septal flap). (a) Superior labial artery; (b) anterior septal branch of superior labial artery; (c) medial internal nasal branch of anterior ethmoid artery; (d) septal branch of posterior ethmoid artery; (e) posterior septal branch of sphenopalatine artery; (f) branch of greater palatine artery.

Nasal vasculature runs in the subcutaneous layer. For each side of the nose, it derives from dorsal nasal artery (a branch of ophthalmic artery that courses over the dorsum toward the tip), lateral nasal artery (that branches off the facial artery or angular artery, and passes medially along the cephalic margin of the lateral crura, just above the alar groove) and columellar artery (a branch of superior labial artery). Lateral nasal artery and columellar artery generally meet over the domal region to form an alar arcade that runs along the cephalic margin of the lateral crura.

In the upper two thirds of the nose, the skin is somewhat mobile, with a slight excess. Proceeding inferiorly from the glabella, its thickness decreases over the cartilaginous dorsum and upper lateral cartilages. Conversely, as it reaches supratip, tip and ala, the skin becomes thick, sebaceous and tightly adheres to the alar cartilages, limiting its mobility. Thus, whatever the design, local flaps will generally mobilize the two upper thirds of skin excess to resurface inferior defects.

Principles of reconstruction

Defect analysis

Except for microvascular transfer, most nasal reconstruction procedures are technically simple. A good analysis of the defect and design are vital for good aesthetics and functional outcomes.

Defect analysis will determine the size, location and thickness of the defect, quality and laxity of the remnant skin and lining, skeleton loss, local and regional scar or graft. Good-quality photographs are recommended.

Wound retraction, where present, gives the illusion of a smaller defect. Mental recreation of the original defect is essential in the reconstructive design. A functionally patent

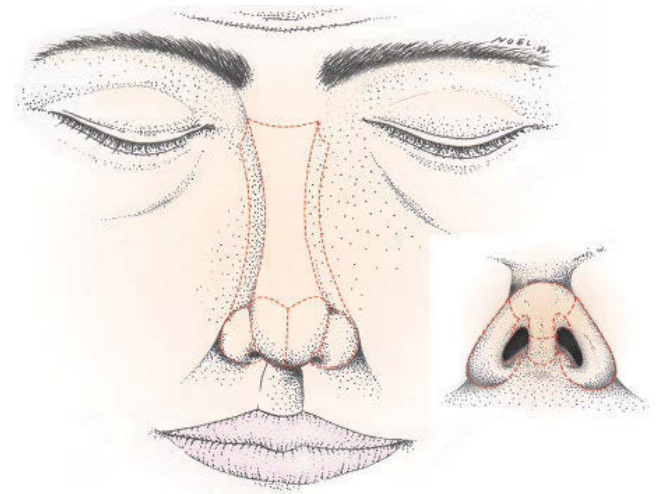


Figure 32.2 Aesthetic subunits of the nose: 1 dorsum, 2 sidewalls, 1 tip (can be divided in two hemi-tips), 2 alar lobules, 2 soft triangles, 1 columella.

nasal airway as well as a good aesthetic outcome are basic reconstructive aims.

Three-layered reconstruction

Overlooking a lining or cartilage loss because it is hidden by skin covering will lead to retraction, collapse and landmark distortion. A three-layered reconstruction is essential to any success.

Subunit principles

As described by Millard⁵ and modified by Burget and Menick,^{6,7} the naturally lit ridges and shadowed valleys of the nose divide its surface into several more or less concave surfaces called aesthetic subunits (dorsum, sidewalls, alar lobules, soft triangles, columella and tip that can be divided into two hemi-tips) (Figure 32.2).

When a skin defect greater than 50% of a convex subunit area is repaired with a transposition flap, it is often better to discard the remnant skin of this subunit and resurface it entirely to avoid a patchwork appearance. This will then place the scars in natural ridges and valleys at the border of the cosmetic units rather than break off the continuous surface of the subunit. Furthermore, when trapdoor contraction occurs, the entire reconstructed subunit bulges, which leads to further aesthetic distortions. For Menick,³ this rule does not apply to the repair of dorsal or sidewall defects, which have indistinct border outlines and relatively flat surfaces, where skin grafts do not undergo trapdoor contraction.

Small and superficial defects

Nasal defects can be classified in two different groups: small superficial defects and large and/or full-thickness defects. The first group will need a skin replacement whereas the second will

necessitate a three-layered reconstruction consisting of inner lining, cartilage and skin. Obviously, defect analysis and reconstruction technique will be completely different for each case.

Small and superficial defects entail the loss of skin and underlying subcutaneous fat or muscle that can be left to heal by secondary intention or repaired by direct closure, full-thickness graft and local skin flaps. Such defects can generally be repaired with a one-stage procedure.

Secondary intention healing

Provided that the wound bed is well vascularized, almost every superficial defect of the nose can heal spontaneously. For example, after rhinophyma excision, it is possible, and often a good choice, to let the entire nose surface heal by secondary intention.

Granulation tissue, epithelialization and wound contracture will lead to a flat, thin, shiny and atrophic scar, at least at the beginning. Thus, the best results will be obtained in thin skin areas and flat or concave surfaces (two upper thirds of the nose). Conversely, use of this method for defects encompassing adjacent landmarks or the alar rim would result in distortion of the shape and normal appearance of the nose.

Finally, in our opinion, secondary intention healing finds its best indications on sidewalls (especially near the internal canthus) and dorsum defects. It can also provide good results on the tip and in alar regions when the defect is small (less than 5 mm) and more than 8–10 mm away from the alar rim. The postoperative care is easy. The patient just needs to wash the wound every day and cover it with a moist dressing. In our experience, no antibiotic or local antiseptic is necessary.

Direct closure

When certain that it will not distort the nasal landmarks or result in nostril margin retraction, direct closure is always the best technique. It will generally leave a good-quality scar, especially when its axis is longitudinal and in the midline. Undermining is often necessary in order to decrease tension.

It is generally possible to directly close wounds up to 1 cm in the two upper thirds of the nose where there is a slight excess of skin. Conversely, we do not recommend it in alar, tip or columella, which are areas of poor skin laxity, as it would distort landmarks and retract the nostril.

Full-thickness graft

Despite their unpredictable final outcome, full-thickness skin grafts can be a good method in many cases, provided that the recipient bed is well vascularized. Like every skin graft, immobility and direct contact with the recipient bed is vital. With this end in view, we employ quilting sutures and tie-over dressing, using a petroleum gauze. Generally, it is removed within 4–7 days, and earlier if any signs of infection are noted.

Preauricular, retroauricular, supraclavicular and forehead skin (this last one is thicker and stiffer) are the best donor sites, with good colour and texture match. Depending on the wound's depth, it can be employed as a full-thickness skin graft or as

a composite graft if underlying soft tissue is carried with the skin. In general the greater the soft tissues included, the better the outcome and the higher the risk of graft failure.

In general, skin grafts can provide good results in thin skin and flat areas such as dorsum, sidewall and columella, whereas it is a poor match in thick and sebaceous skin zones such as ala, tip and supratip (forehead skin is probably the best donor site in these locations³).

Local skin flaps

Local skin flaps mobilize the slight excess of skin within the upper two thirds of the nose and glabellar zone. Unlike skin grafts, the colour and texture of such flaps are predictable since the intrinsic blood supply of the raised skin is preserved. As they are also thicker than grafts, they can better supply missing subcutaneous tissue, but should not be an option when a significant cartilage support is missing. In such cases, the reconstructed cartilage framework would most likely not resist the skin tension of a local flap and would risk losing its resistance and shape, leading to collapse and landmark distortion.

Many local flaps have been described and we present some of the most frequently used below.

Rieger's flap

Described by Rieger in 1967,⁸ the dorsal nasal flap is vascularized by vessels of sidewall and medial canthus. Skin, subcutaneous tissue and muscles of the two upper nasal thirds and glabellar area are elevated just above perichondrium and periosteum and mobilized toward the lower third of the nose (tip and supratip). A dog-ear is excised inferiorly and glabellar donor site is then closed with a V-Y closure (Figure 32.3). Mobilization of this flap can be emphasized by Marchac's modification⁹ that narrows the pedicle around the only medial canthus zone with a counter-incision at the junction between cheek and sidewall.

Despite this, even for defects smaller than 1.5–2 cm tip and supratip defects, we do not use this flap frequently. This is because of differences in skin thickness along the wound margin and because its inferior border often leaves a visible and depressed scar crossing the round tip subunit. Additionally, it tends to retract alar rim, approximates eyebrows and creates a radix fullness that often necessitates a revision.

Bilobed flap

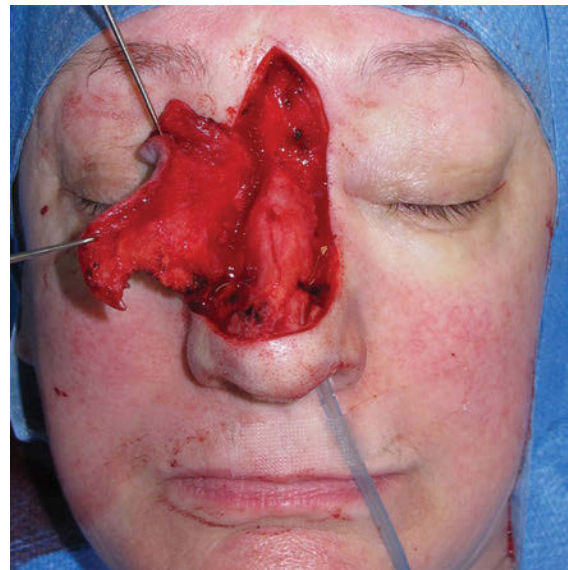
Zitelli's bilobed flap¹⁰ is a double transposition flap widely used for defects up to 1.5 cm in diameter located in the lower third of the nose (Figure 32.4).

The first lobe is designed adjacent to the defect with the same diameter. The second one is adjacent to the first one, slightly smaller and has to lie in an area of laxity to allow its direct closure (two upper thirds). The angle of transposition is approximately 45° for each lobe. In general, the flap is based laterally for tip and supratip defects and medially for alar defects.

After resection of the inferior dog-ear between pivot point and defect, the flap is incised and elevated. We recommend wide



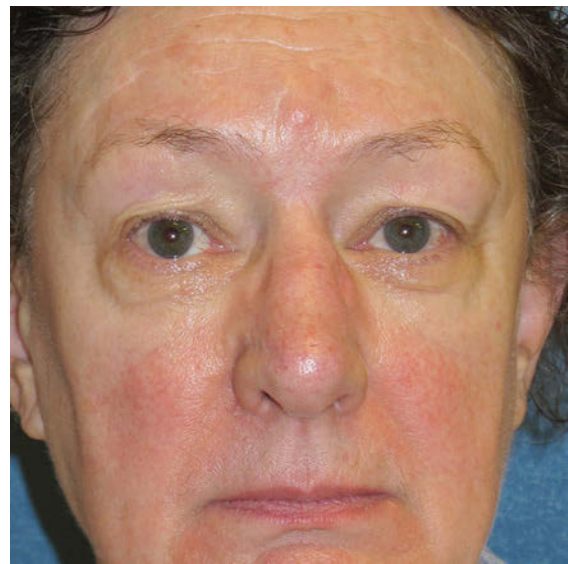
(a)



(b)



(c)



(d)

Figure 32.3 Rieger's flap. Source: Dr B. Sarfati. Reproduced with permission.

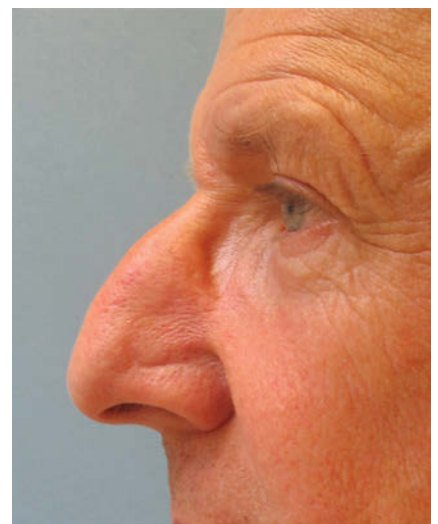


Figure 32.4 Bilobed flap (note the alar crease fullness). Source: Dr B. Sarfati. Reproduced with permission.

undermining of the defect borders, flap base and donor site just above the perichondrium and periosteum in order to facilitate transposition and minimize distortion. The first lobe is then mobilized to cover the defect and the second one shifts to repair the second defect created by the first lobe. This final defect is closed with direct suture.

Even if lower skin of the nose is resurfaced with a well-matched nearby skin, the surgeon should be cognizant that swelling may be significant; persistent erythematous scar and nostril retraction when it lies less than 1–1.5 cm away from the margin, often obliterating the alar crease, are major considerations. The patient should be advised that revision is often necessary.

Single-stage regional skin flaps

Regional skin flaps mobilize skin of nearby units of the face from the glabellar and nasolabial area.

Glabellar flap

Like the Rieger's flap, laxity of the glabellar area is used to cover defects of dorsum, medial canthus or lateral wall. Based on the medial canthus, it can be mobilized using advancement, rotation or transposition.

As the donor site is closed with a V-Y closure, it leads to eyebrow approximation. It also often creates differences in skin thickness along the wound margin. For these reasons we generally keep this procedure for frail elderly patients who need a simple and one-stage reconstruction.

Single-stage nasolabial flap

Directly adjacent to the nasolabial fold, excess skin of the medial cheek can provide very good-quality skin for lateral defects of the nose. The colour and texture are very similar to those of alar and sidewall.

Single-stage nasolabial flap is generally indicated for defects of alar and sidewall up to 2 cm in width (Figure 32.5). This size corresponds to the maximum width of skin that can be harvested in the medial cheek without leading to unacceptable distortions of the nasolabial fold or asymmetry of the oral commissure due to excessive tension during donor site closure. (If more than 2 cm in width is needed for a total alar subunit, for example, the pattern will have to lay parallel to the nasolabial fold, therefore a two-stage flap will be necessary.)

In contrast to the two-stage flap, the single-stage nasolabial flap mobilizes the excess skin of the medial cheek as a random

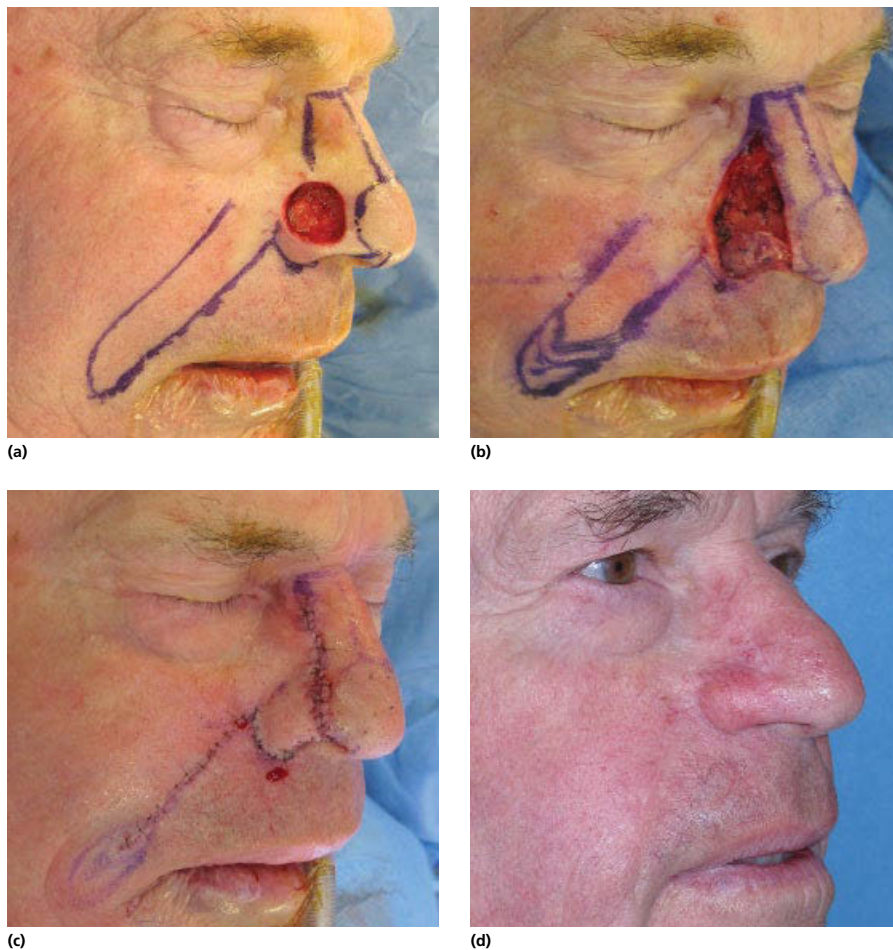


Figure 32.5 One-stage nasolabial flap. Source: Dr B. Sarfati. Reproduced with permission.

pattern extension of a cheek advancement. It means that the blood supply is not by arterial perforators of facial artery but from subcutaneous and dermal vessels of the cheek. The nasolabial fold has to be outlined on an awake patient, especially in young people, as the crease can be indistinct and only a wide smile can precisely locate it.

Prior to skin replacement, an extra-anatomical cartilage graft should brace the lining to prevent collapse and nostril margin retraction. This graft should be quilted to lining and be of good shape and size. In this location, we use conchal cartilage because of its natural round shape. The flap is designed immediately adjacent to the crease. Considering the position of the pivot point and angle of flap transposition, it can be difficult to determine exactly at which height the pattern of the defect should be positioned to inset without tension. The distal margin of the flap lies in the dog-ear and may be trimmed at closure. Thus only the maximum width of the defect needs to be measured at the nasolabial fold.

The nasolabial flap and its distal dog-ear are incised and raised in continuity with 2 or 3 mm of subcutaneous fat. The lateral incision should not extend higher than the top of the nasolabial fold. This may compromise the blood supply. The cheek is then elevated just above the superficial musculoaponeurotic system (SMAS). The wider the nasolabial flap, the more extensive the cheek undermining required in order to decrease tension at closure.

The cheek flap is advanced and attached to deep tissues with solid underlying long resorption sutures along the groove between nose and cheek. This fixation is of vital importance as it will recreate this groove (otherwise a fullness appears), close the nasolabial incision and eliminate tension on the nasolabial extension flap. A dog-ear appears on the sidewall, just above the defect, and should be trimmed.

Ideally, the alar crease may be recreated with quilting sutures between the flap and lining, at the superior border of the cartilage graft. Great attention is given to blood supply and, in case of doubt, quilting sutures should be removed. Finally the flap is thinned again, if necessary, precisely trimmed on its distal margin to fit perfectly, and all incisions are closed.

We often use the single-stage nasolabial flap for elderly or fragile patients. In such cases, this technique is efficient and generally provides acceptable aesthetic results. Nevertheless, its maximum width of 2 cm (often less on young patients) often precludes reconstruction of a total ala following the subunit principles. For this reason, each time a patient demands the best procedure in terms of results without consideration for number of stages, we prefer the two-stage nasolabial flap or forehead flap.

Large and/or deep defects

Rather than purely relying on measurements, we prefer to consider defects that we know will necessitate at least a two-stage procedure. In many cases, it is possible to close a defect (even a

full-thickness one) with a one-stage technique, but cosmetic and functional outcome would be greatly improved with a multistage approach. In such cases, the different possibilities have to be discussed with the patient in terms of result, total time and number of operations. In our experience, this is only a concern for elderly or very fragile patients.

Even in larger defects greater than than 1.5–2 cm defect remnant skin of the nose may be sufficient to close the defect but insufficient to resurface nasal skin without any distortion of landmarks and rim or columellar retraction. A large skin graft is always an option, but colour, thickness and texture are unpredictable and may well not be satisfactory, especially in the lower part of the nose. In such cases, additional skin mobilized from the cheek or forehead should be used, which necessitates at least two stages.

Concerning the depth of the nasal defect, as soon as it involves cartilage, the reconstruction needs cartilage replacement. If support is not restored, it will lead to cosmetic failure (landmark deformities, nostril and columella retractions) as well as functional problems (nasal airway collapse). A cartilage graft precludes use of a skin graft. The columella, infratip lobule and defects very close to the nostril rim will generally need additional skin in order to avoid distortion in these delicate areas.

Skin replacement

Large and/or deep defects need additional skin and it is sometimes necessary to harvest this from a distant location (Washio flap or distal free flap). The best skin match is the skin closest to the defect and therefore the best donor sites are medial cheek and forehead.

Two-stage nasolabial flap

First stage

This flap will transfer the excess skin of the medial cheek to the lateral part of the lower nose, directly adjacent to the nasolabial fold. In our clinic, we only use it for reconstruction of the entire alar subunit. When harvested in two stages, nasolabial flap is vascularized by perforators vessels coming from facial artery and crossing the muscular plane below, through and above levator labii muscle. Therefore, this flap is a transposition island flap, with the pivot point located at the junction between cheek, alar groove and lip. The nasolabial fold has to be outlined on an awake patient.

A very precise template of the contralateral alar subunit is then transposed adjacent to the nasolabial fold (Figure 32.6). As the flap will be transposed with an angle of about 110°, one should place the medial part of the template distally in the flap. Positioning of the template has to be calculated to reach the ala without any tension. It is usually positioned just above the oral commissure.

Before harvesting the flap, it is strongly recommended to brace the lining (reconstructed or not) with a cartilage graft to avoid collapse and alar rim retraction. Despite lack of cartilage

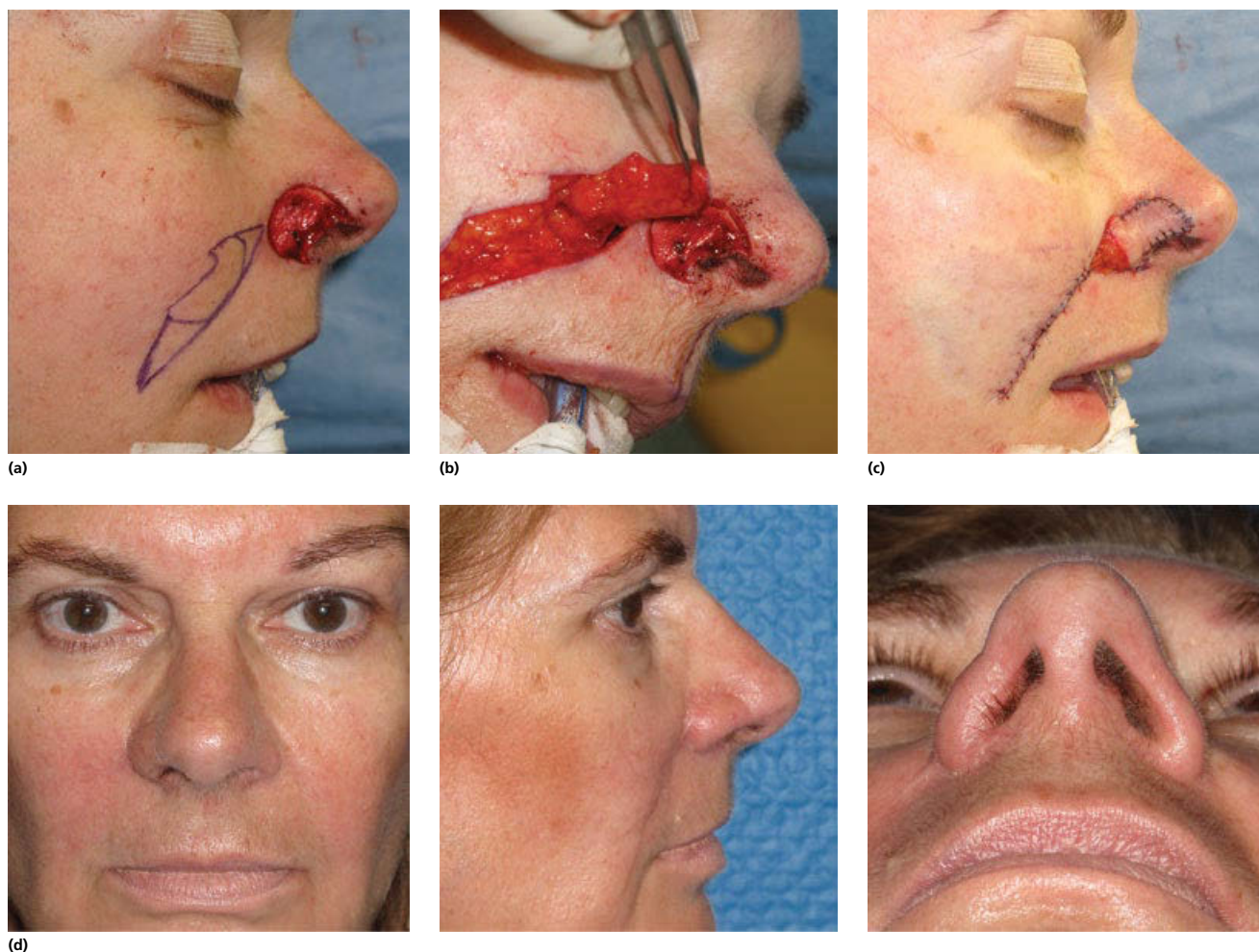


Figure 32.6 Two-stage nasolabial flap. (a, b, c) After alar subunit excision, a cartilage graft is positioned. The flap is then designed directly adjacent to the nasolabial fold (probably too high in this case), elevated in the subcutaneous plane and sutured. (d) Result six months after the pedicle division.

support in the normal ala, the fibrofatty middle layer is normally able to act against collapse during nasal inspiration. However, with a significant amount of alar skin loss, this ability decreases and collapse is more likely to occur. Furthermore, this cartilage graft will help to recreate the round shape of ala and its upper and external border will design the alar groove. In this location, we use concha cartilage because of its natural round shape. This graft will be designed a few millimetres smaller than the skin template.

The flap is elevated where possible. We prefer general anaesthetic since infiltration would distort the landmarks of the nasal subunits and nasolabial fold, and adrenaline will make blood supply difficult to evaluate during the procedure. Dissection is performed from distal to proximal. The distal part of the flap is elevated with 2 or 3 mm of fat to avoid secondary thinning of this region, but dissection becomes deeper as it reaches the perforators area. In this medial part, the subcutaneous base is protected and dissection has to be gentle, with small scissors in order to keep the perforators. There is no

reason to cut muscles. Once the flap reaches the defect without any tension, undermining is stopped and the medial part of the flap is precisely sutured to the defect. The surgeon should be aware that, although reliable, blood supply is more fragile than that in the forehead flap, and distal necrosis may occur where there is tension (even slight) or extensive dissection of the pedicle. For the same reason, we do not use the nasolabial flap as a real propeller flap. We consider this a risky proposition without improving the final result.

Before donor site closure, depending on the flap's width and cheek's laxity, the cheek is more or less undermined to decrease tension and oral distortion. Concerning the inferior dog-ear, we like to harvest it with the flap and trim it just before suturing the flap. It may be used to handle the flap without injuring its distal blood supply.

Second stage

Although we prefer to wait four weeks, many authors perform pedicle division after three weeks.

As it is already thinned during elevation, the medial part of the flap does not need additional work. Conversely, as dissection was deep in this area during the first stage, the lateral part is thick and bulky. Therefore, after division, the external skin flap is re-elevated and thinned, keeping just 2–3 mm of underlying fat. The tail is then sutured and the superior dog-ear (the flap's pivot point) is excised.

In general, the skin colour matches well with the remnant nasal skin and the trapdoor effect will contract the flap into a sphere shape, which will often improve the result with time. A good design and location of the donor site makes the final scar hardly visible. Nevertheless, in some cases the cheek may be flattened and necessitate a contralateral excision of nasolabial fold to re-establish symmetry.

This technique usually provides good cosmetic results and for reconstruction of the entire alar subunit we feel this is its only indication. As soon as a defect involves part of the tip or sidewall, we prefer to use a forehead flap, which is also an excellent option for alar reconstruction.

The two-stage paramedian forehead flap

The forehead skin¹ is by far and away the best for nasal reconstruction. Its colour, texture, thickness and amount of available tissue make it our first choice for large and/or deep defects.

Forehead flaps have been described and used in numerous fashions and designs, depending on its blood supply (one or two supratrochlear arteries, supraorbital artery, superficial temporal artery), its localization on the forehead (median, paramedian, lateral) and its axis (vertical or oblique).

For us and for most authors, it is now clear that the best option is the paramedian forehead flap vascularized by just one supratrochlear artery. In our opinion, the converse scalping flap¹¹ does not increase the amount of forehead skin transfer and is a high-morbidity procedure for the patient, will temporarily blind one of the patient's eyes with a large, heavy, hairy and uncomfortable pedicle, and necessitates the use of an evident and unsightly skin graft to resurface the lower forehead, just above the eyebrow. With regard to the oblique forehead flap that is still widely used when the forehead is supposed to be too short, we think that it leads to a much more visible scar and is a more risky proposition, unless the frontalis muscle is harvested along the entire pedicle length. In addition, this leads to unattractive paralysis of the hemi-forehead.

Conversely, the paramedian forehead flap is very reliable and can provide enough skin to resurface the entire nose. The lower part of the scar is generally almost invisible and, if designed narrow enough, the eyebrow approximation is very acceptable. Concerning the upper part of the donor site that is often too wide to allow direct closure, we advise against skin graft that could be a temptation to close the defect. In our experience, skin graft matches the forehead remnant skin very poorly. It produces a strong 'patchwork' effect due to its thickness, difference in colour and texture. Furthermore, it will stop the contraction

of the wound that would decrease the size of the final scar. The authors consider that, even for a very wide flap, secondary intention healing provides the best result. It will heal in a few weeks with a daily wash and dressing, leaving a significantly smaller scar because of contraction. This scar is red at the beginning but it will become paler with time and may be hidden by hair as it lies in the upper part of the forehead. After several months, in some cases, a new laxity of forehead can allow a scar revision to make it more narrow.

Vascularization

It is now well known that only one supratrochlear pedicle is able to supply a forehead flap resurfacing the entire nose. This artery is a branch of the ophthalmic artery, which originates from the internal carotid artery. It leaves the orbit at its medial angle, crosses the orbital rim under the lion's wrinkle, between corrugator and frontalis muscles, before coursing vertically upwards.

It is at first subperiosteal, but as it passes over the brow it lies above the periosteum, protected by the corrugator first and then by the frontalis. This means that above the orbital rim the flap can be safely elevated over the periosteum. Under the orbital rim, dissection has to be under the periosteum. At about the mid-forehead level, the artery will course through the frontalis muscle to lie in the subcutaneous plane, increasingly becoming superficial as it reaches the hairline.

First stage

When the nasal defect is lateral, the flap is harvested on the same side. When it is median, it can be elevated on either sides. (hairstyle is considered when choosing the side in order to hide the upper part of the scar).

The lion's wrinkle has to be outlined on an awake patient, especially for young people. This is sometimes invisible and only a wide grimace will allow the surgeon to precisely locate it. It is recommended for safety to verify the location of the supratrochlear artery preoperatively with Doppler.

Where possible, we prefer to perform this surgery under general anaesthesia because infiltration would distort the landmarks of the nasal subunits and adrenaline makes the blood supply difficult to evaluate during the procedure. If a significant amount of cartilage is missing, the lining has to be braced with cartilage grafts before harvesting the flap. This will avoid collapse, retraction and landmark distortions.

A very precise template of the defect (respecting the subunit principles and, when intact, derived from the contralateral side) is designed and transposed to the paramedian forehead, just off the midline. This is positioned as high as possible, but below the hairline if possible to avoid hair transfer to the nose.

The pedicle of the flap is then marked out, narrowed from the pattern to the lion's wrinkle. The base pedicle width can be 1.2–1.5 cm in width. The blood supply is thus safe and a narrow pedicle will augment the arc of rotation, decrease closure tension and limit approximation of the brow.

The flap is elevated vertically downwards. As a two-stage flap, the distal part of the flap is thinned directly at this first stage. This means that it is elevated with a few millimetres of fat (trying to obtain the same thickness as the remnant skin of the nose), but respecting the frontalis muscle. This decreases blood supply and may lead to local necrosis in smokers or in very large flaps. (In such cases, it may be safer to elevate the flap with muscle and plan an intermediate stage for thinning.)

About 1 cm above the mid-forehead level, dissection goes under the frontalis muscle (but over the periosteum) to protect the artery. It can stay in this plane until the brow, after which dissection should continue under the periosteum. The flap is then rotated medially and when the paddle reaches the defect it can be closed precisely without tension in a single layer.

We like to use thin quilting sutures for 2–3 days in order to prevent haematoma and to help to recreate natural creases of the nose, such as the alar crease. Even gently tied, they can jeopardize the distal blood supply. For this reason, the flap has to be checked 1–2 h after surgery and if there is any doubt they are removed.

The forehead is then undermined on both sides in the subgaleal plane to decrease tension and precisely sutured in layers. Special care is given to maintaining eyebrow level during closure (one should not elevate the ipsilateral one). If the upper part is too wide to be closed, it will heal by secondary intention. It does not distort the brow and the final result is generally better than a skin graft.

Finally, for the comfort of both patients and nurses, we always skin graft the pedicle. This significantly decreases bleeding, which generally completely stops after 2 days. Obviously, this graft should not compress the artery.

Second stage

Some authors perform the second stage three weeks later. For greater safety, we prefer to wait four weeks. Before division, it is recommended to check the viability with a pedicle compression. If flap perfusion is maintained the pedicle can be divided.

After transection, the superior part of the flap is re-elevated. Excess subcutaneous tissue is excised in order to recreate natural convexities and creases of the nose. Further than 1.5 cm of distal flap margin elevation risks devascularization of the flap.

When the desired shape and thickness is obtained, the superior flap margin is trimmed to fit the defect precisely and sutured. Quilting sutures are recommended for at least 2–3 days to prevent haematoma and help recreate the natural creases of the nose. Flap perfusion has to be checked 1–2 h after surgery and there is any doubt they are removed.

Finally, the inferior forehead scar is reopened and the proximal pedicle is returned to the donor site and sutured in a small inverted 'V' with quilting sutures to avoid haematoma. It can also be completely excised, leaving a barely visible scar on the eyelid, but this will bring the eyebrows closer. We advise against a large inverted 'V' high on the forehead as they often become bulky and develop a retracted scar.

The three-stage paramedian forehead flap

Thinning the flap at the first stage could compromise blood supply in smokers or when a large flap is needed. In such cases, we advise three stages for greater safety and improved results.

Millard¹² first introduced an intermediate stage which was later modified by Burget. During the second stage, the pedicle was left intact and the distal inset was left attached up to 1.5 cm from its distal margin. This is effectively a bipedicle flap, allowing the middle part of the flap to be thinned safely; however, there are difficulties due to poor visibility, and the distal part of the flap could not be addressed at that stage.

In our practice, we prefer to use the full-thickness three-stage forehead flap described by Menick.³ During the first stage, except if a skin graft or a folded forehead flap is used for lining replacement, primary cartilage grafts are placed. Then, a full-thickness forehead flap (skin, subcutaneous fat and frontalis muscle) is elevated and sutured to the recipient bed.

In the second stage four (or three) weeks later, the flap can be completely re-elevated safely in the subcutaneous plane, with a thickness of 2 mm, due to the delay phenomenon. The most proximal part of the flap can be thinned during the final stage. The underlying recipient bed, comprising fat, frontalis muscle, scar and cartilage, is then sculpted precisely to recreate landmarks and creases. If needed, additional grafts can be used. The thinned flap is then repositioned.

Finally, division is performed four (or three) weeks later.

For us, this technique is by far and away the best with regard to both reliability and results.

Lining restoration

Restoring the lining is, of course, of primary importance. Even if hidden by skin resurfacing, leaving a lining defect precludes the use of cartilage grafts and will always lead to poor results. The raw undersurface of the flap will contract during secondary healing and create distortions of the shape and obstruct the airway.

Full-thickness skin graft

A full-thickness skin graft can be used in different ways for nasal lining. It will provide a thin reconstruction but will tend to retract and will need to be strongly braced by cartilage grafts.

Prefabricated forehead flap

The prelaminate forehead flap was first described by Konig. Later, it was popularized by Gillies¹³ and then modified by Converse.¹⁴ In this technique, the flap lining and cartilage framework is first built on the forehead and then transferred onto the nose.

The first stage consists of designing a precise template of the skin defect and another one for the lining defect. The template of missing skin is transferred to the forehead, exactly like a traditional forehead flap. Only the distal part of the flap is incised and undermined between frontalis and periosteum.

A full-thickness skin graft is then harvested with the template of the missing lining. This skin graft is sutured with fine absorbable sutures to the underside of the frontalis muscle.

Finally, cartilage grafts are inserted in a tunnel between skin and frontalis muscle. In order to maintain maximum connection between the skin and muscle, when possible, it is better to introduce the cartilage grafts through an incision on the lateral border of the flap. Four weeks later the flap is completely elevated, positioned and sutured into the defect. Before division, an intermediate stage can be added.

We do not feel that this technique has great advantages for the patient. The number of stages is not reduced and it is difficult to obtain optimal results because precise positioning of the cartilage framework is difficult to achieve given that the 3D shape is difficult to predict when the flap is still flat on the forehead.

'Classical' skin graft

Although it has been used in many ways for a long time, the classical skin graft has given poor results for the restoration of nasal lining. Menick recently modified the technique.³ A full-thickness skin graft can be sutured to the borders of the lining defect. Then a full-thickness forehead flap is elevated and positioned on the nose. Quilted absorbable sutures fix the graft to the flap. A skin graft naturally precludes the use of a cartilage graft.

Four weeks later, during an intermediate stage, the flap is re-elevated with 2–3 mm of subcutaneous fat. Excess scar, subcutaneous tissue and frontalis muscle is excised from the recipient bed. The graft is normally well vascularized and the cartilage framework can be placed without compromising the graft vascularization. Finally, the flap is replaced and sutured to the nose. Four weeks later, the pedicle is divided.

In our experience skin graft take is often unpredictable and for this reason the patient is checked regularly. If the skin graft fails, it can be replaced with another, delaying the intermediate stage.

Hingeover flaps

After a full-thickness amputation, sutured together or not, remnant skin and lining will join by scar. After several weeks (6–8 depending on authors) neighbouring skin vascularized across the scar can be elevated and hinged over to replace lining. These flaps are often thick and not easy to shape with a cartilage graft as they contain scar tissue. Nevertheless, they can be very useful for small defects or in association with intranasal lining flaps when a few millimetres of lining are required.

Intranasal lining flaps

All of these lining flaps can be used alone or in an association. Although well vascularized, we advise against their use in smokers. They are generally used with primary cartilage grafts and should be used if necrosis of the lining leads to cartilage graft exposure, resulting in probable infection and graft resorption.

They can be technically difficult to perform, especially when the full-thickness defect is small due to poor visibility. Even when strongly fixed with cartilage graft, such flaps will often tend to retract toward their pivot point, which means inside the nose. For this reason, stenosis regularly occurs and can necessitate a revision. Additionally, lining flaps will often lead to bleeding and crusting that can obstruct airway and compromise patient comfort. Nevertheless, they are often very useful for lining restoration.

Residual nasal lining advancement

When the lining defect is less than 4 mm along the nostril rim, the remnant vestibular lining can be separated from alar (and upper lateral cartilage if needed) to be pulled downward. To avoid secondary retraction, this lining advancement has to be braced in position with a primary cartilage graft.

Bipedicle flap

In the same area, if the lining defect is bigger, intact vestibular skin can be freed from cartilage and incised superiorly. Thus, a bipedicle flap can be moved caudally. A new superior lining loss is created and has to be restored by another flap, generally an ipsilateral septal flap.

Ipsilateral septal flap

Described by Burget and Menick,^{2,16} this flap is vascularized by the septal branch of the superior labial artery (Figures 32.1, 32.7 and 32.8). This artery comes from the facial artery and passes medially through the orbicularis oris muscle, at the junction between skin and vermillion. Just lateral to the philtrum, it gives rise to the septal branch that goes vertically upward from lip to septum, passing laterally to the nasal spine.

This branch is theoretically able to vascularize the entire ipsilateral septal lining. Thus, an ipsilateral septal mucoperichondrial flap can be elevated from posterior to anterior and can restore lining losses of the lower third of the nose. When the defect is small this flap can be challenging to harvest because of poor visibility.

Contralateral septal flap

This flap is vascularized by the medial branch of anterior ethmoidal artery that runs parallel to the dorsum, less than 1 cm from it (Figure 32.9). After elevation of the ipsilateral mucosa (used as a flap too or replaced after contralateral flap elevation, depending on lining loss), the septal cartilage is removed, preserving a septal 'L' strut for nasal support. Then a dorsally based flap of contralateral septal mucosa is incised, maintaining at least 1 cm of its dorsal blood supply. This flap is then swung laterally in order to line the mid-vault. Finally, if the ipsilateral mucosa is not used to line the lower part, it is replaced in order to repair the fistula. (In its first description, De Quervain¹⁵ used this flap as a chondromucosal flap, keeping the cartilage attached to mucosa, but it seems easier to us to remove it before cutting the flap.)

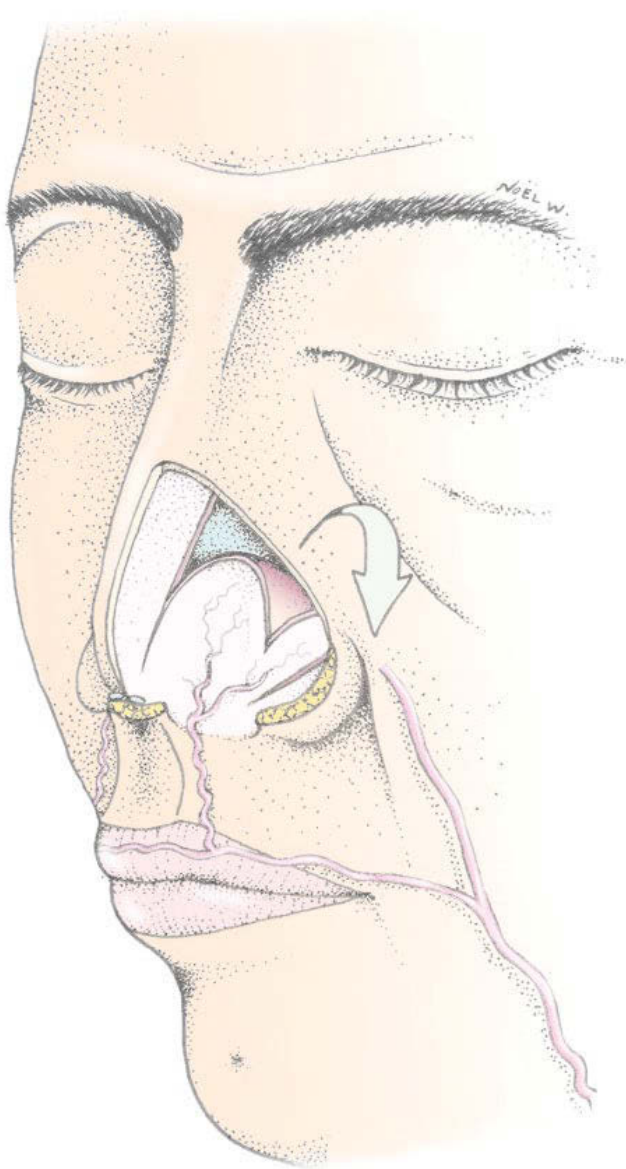


Figure 32.7 Elevation of an ipsilateral mucosal flap based on the anterior branch of the superior labial artery. Source: Dr Warren Noel. Reproduced with permission.

Ipsilateral and contralateral flaps are frequently used for large defects of the hemi-nose (Figures 32.10, 32.11 and 32.12). This will result in a fistula which is unlikely to whistle due to its width.

Septal pivot composite flap

This flap, described by Burget and Menick,² is useful for central full-thickness defects of the lower half of the nose (Figure 32.13). It will provide the dorsal support and restore lining of the mid-vault, but it is not able to restore lining of the entire lower third of the nose.

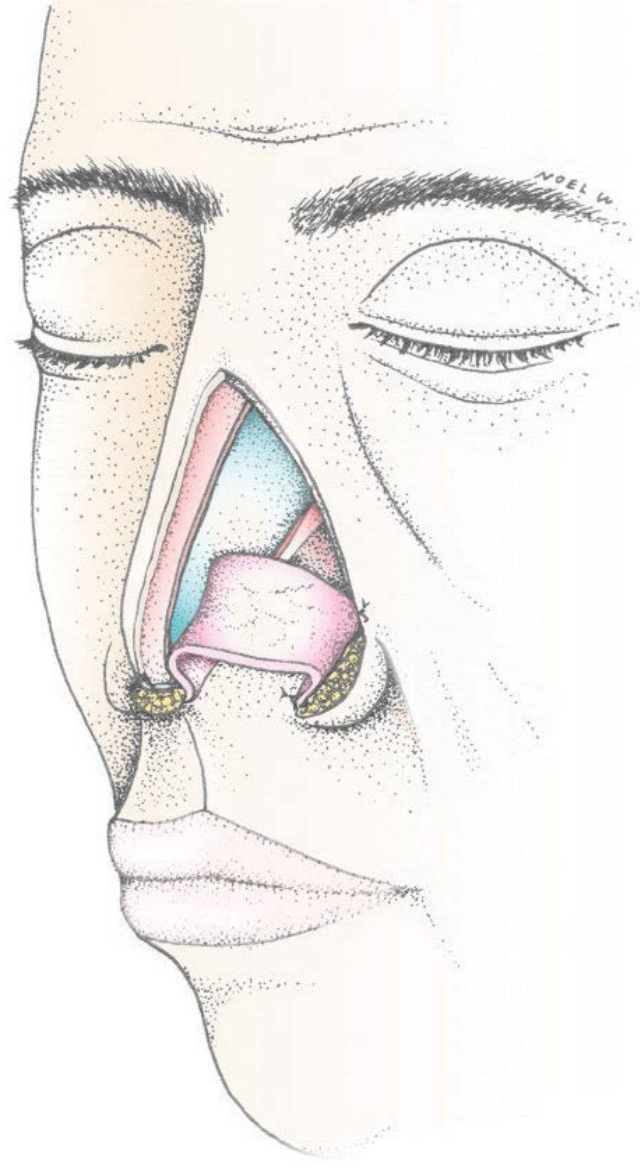


Figure 32.8 Twist of an ipsilateral mucoasal flap sutured to the ala. Source: Dr Warren Noel. Reproduced with permission.

It can be used when amputation leaves the posterior part of the septum intact, which is normally the case. The minimum required is that the remaining septum is flush with the face. If the resection involves more of the septum, the septal branches of the superior labial arteries are probably cut and flap survival is poor.

The full-thickness septum is cut superiorly, posteriorly and inferiorly until 1.2 cm from the nasal spine. In the region of the nasal spine, the mucosa is preserved as it contains the two septal branches. The flap is now only retained by attachments between cartilage and nasal spine. Therefore mucoperichondrium has to be very cautiously separated from each side in this region in order to cut the septal cartilage attachments without compromising vascularization. The composite flap is then rotated forward, out of

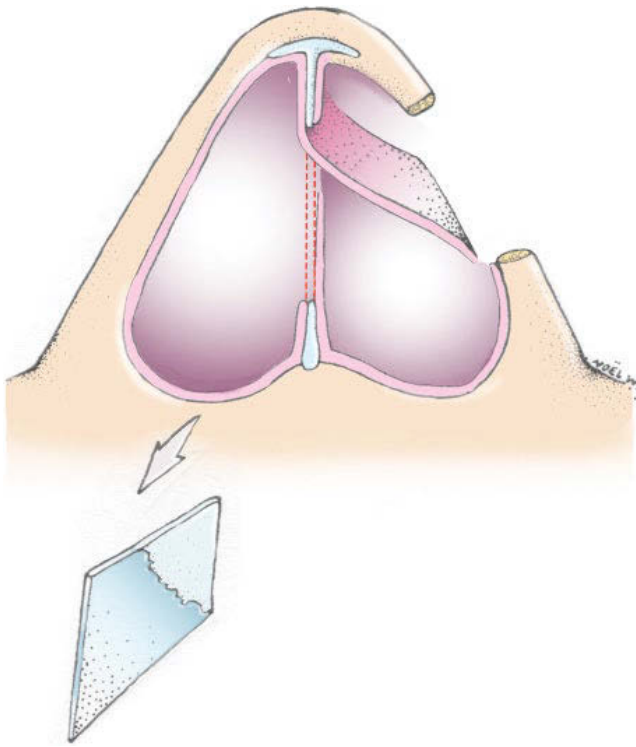


Figure 32.9 Contralateral mucosal flap (septum cartilage partially removed and ipsilateral mucosa repositioned). Source: Dr Warren Noel. Reproduced with permission.

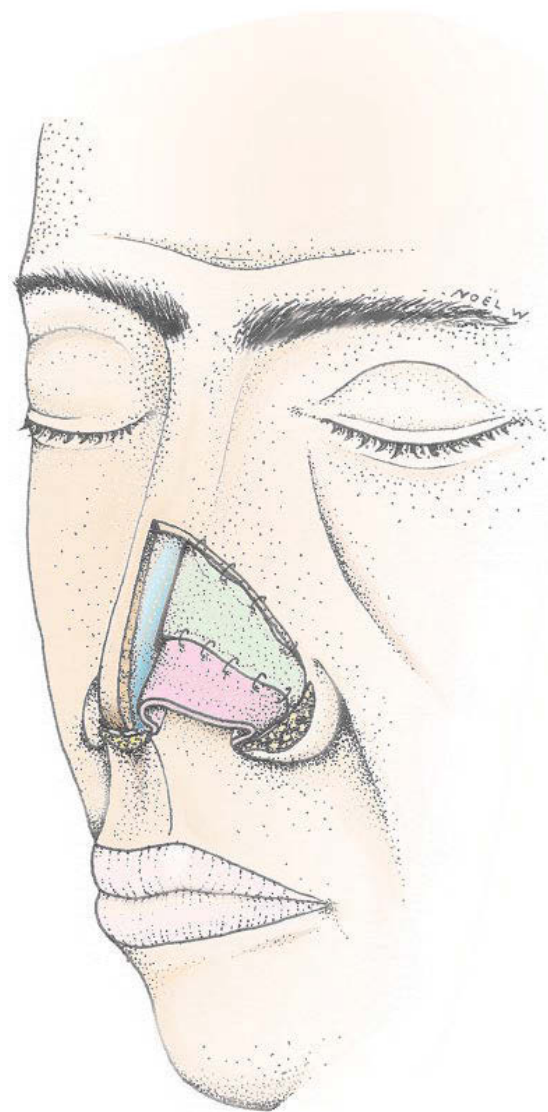


Figure 32.11 Association of ipsilateral and contralateral mucosal flaps, lateral view. Source: Dr Warren Noel. Reproduced with permission.

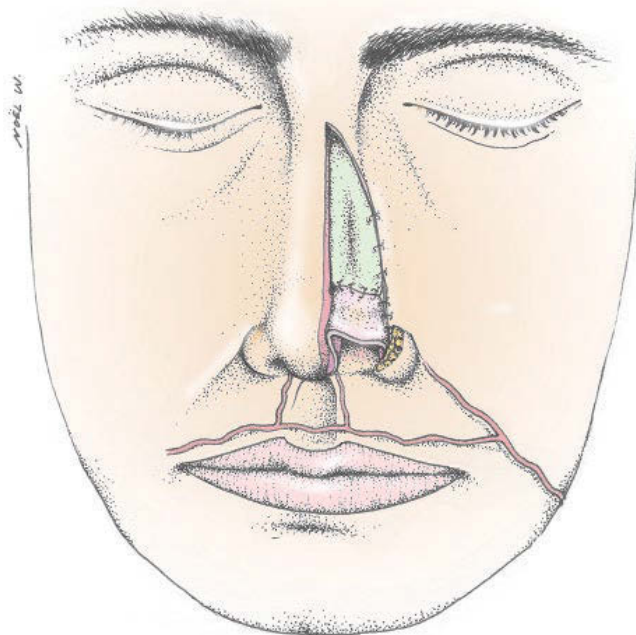


Figure 32.10 Association of ipsilateral and contralateral mucosal flaps, frontal view. Source: Dr Warren Noel. Reproduced with permission.

the nose, and fixed superiorly to nasal bones and/or upper lateral cartilages. This will recreate the midline support of the nose.

Regional flaps

The use of a nasolabial flap for lining was popularized by Millard.¹² Vascularized by perforators from the facial artery, it can restore the lining of alar bases and vestibule. Nevertheless, because of the risk of necrosis with proximal dissection and of not being able to thin the flap adequately we prefer to find another solution, otherwise there is a risk of airway obstruction and lateral displacement of the alar base.

A second paramedian forehead flap can be used to resurface a hemi-nose lining defect. However, if the cause of amputation was a skin malignancy, one should always consider the risk of recurrence and that the patient may require another forehead flap.

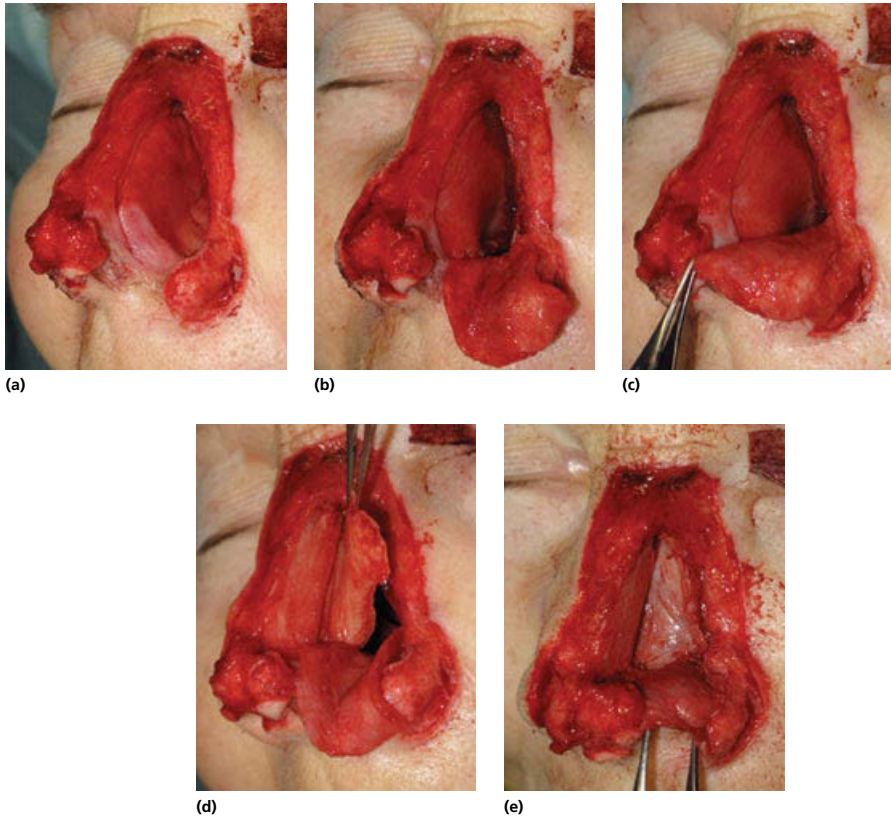


Figure 32.12 Association of an ipsilateral and a contralateral mucosal flap in a hemi-nose defect. (a, b, c) Incision, elevation and twist of the ipsilateral flap. (d) Contralateral flap incised and swung laterally after septal cartilage removal (preserving a septal L for support). (e) Both flaps sutured together and to defect margin, ensuring hermetism for cartilage grafts.

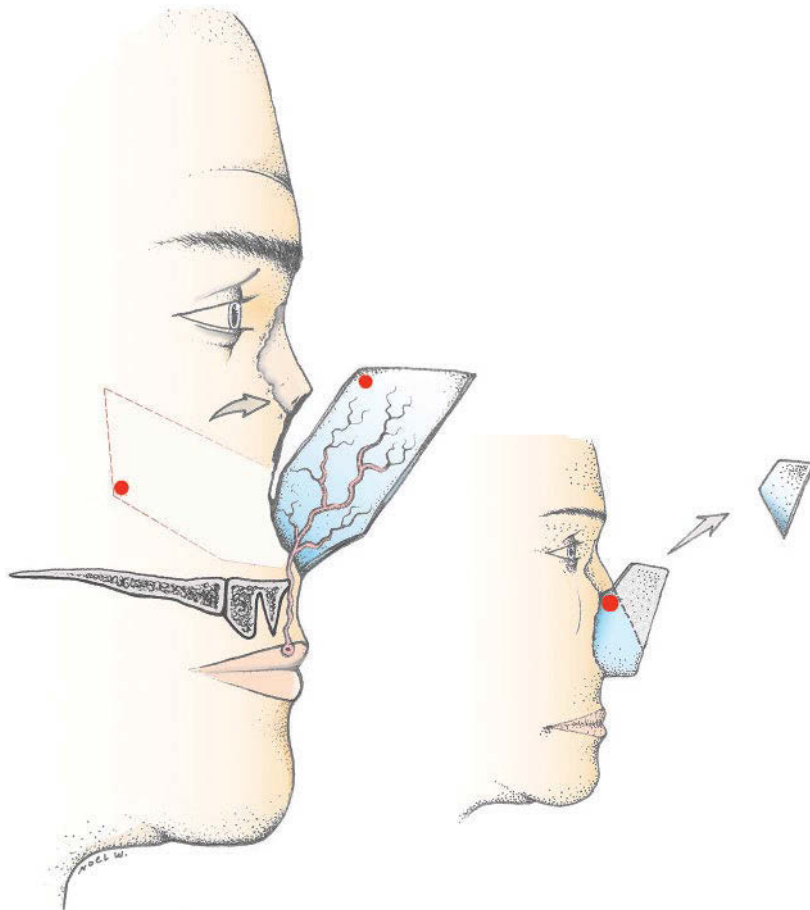


Figure 32.13 Septal pivot composite flap.
Source: Dr Warren Noel. Reproduced with permission.

The superiorly based facial artery musculomucosal flap described by Pribaz *et al.*¹⁷ can also be used to reconstruct the mid-vault.

Three-stage folded forehead flap

Until recently, folding the end of a forehead flap for lining reconstruction was a traditional two-stage method. Adding an intermediate stage, Menick¹⁸ modified this technique and it has become our preferred technique for the reconstruction of unilateral and bilateral defects of up to 3.5 cm in the lining. Preserving septal lining minimizes bleeding, permanent crusting and intranasal retraction of lining flaps.

During the first stage, a precise template of the missing lining is transferred to the forehead as an extension of the forehead flap, distally to the missing skin pattern. If required, this pattern can lie in the hair line as it will replace lining. A few millimetres of extra skin are added between the two patterns in order to fold the flap more easily during its inset. After elevation of the full-thickness forehead flap, the distal lining extension is folded inward and sutured to the residual mucosal lining. No cartilage graft is added at this stage.

A marginal incision is performed 3–4 weeks later and the proximal part of the flap is completely re-elevated, leaving the reconstructed lining on the nose. Each side of the flap is defatted and sculpted before the cartilage framework is precisely reconstructed. Before closing (or during marginal incision) the surgeon should note that the extra skin area has to be precisely removed. Finally, the flap is sutured back onto the nose. The pedicle is divided 3–4 weeks later. This is one of our most favoured techniques (Figure 32.14).

Distant free flaps

When the lining loss is too great, microvascular distant tissue transfer must be considered. The radial forearm free flap is the most commonly used for inner lining reconstruction due to its thinness and its reliability. This is harvested suprafascially and is anastomosed to the facial or neck vessels.

When a free flap is needed we advise coverage with a temporary skin graft. Two months later, the skin graft is excised to allow thinning, the addition of cartilage grafts and resurfacing with a two- or three-stage forehead flap.^{19,20}

Structural support

After nasal amputation, the bone and cartilage framework has to be replaced to recreate both strong support and the normal landmarks of the nose. If not precisely replaced, whatever skin and lining reconstruction is used, the result will be poor in terms of aesthetic and functional outcome.

With unilateral reconstruction the cartilage framework aims to recreate the contralateral hills and valleys as precisely as possible. When the defect crosses the midline the surgeon must determine the dimensions of the nasal reconstruction.

The ideal time for the reconstruction of the structural support is during the primary, intermediate or final stages rather than secondarily. It is possible to perform small augmentations and refinements with grafts but the skin is more difficult to mould and a complete framework will be difficult to recreate at this time.

Septal cartilage is very efficient for columellar strut, only dorsal graft, tip grafts and sidewall grafts. A dorsal and caudal L has to be preserved for midline support and tip projection.

Conchal cartilage is very useful for alar reconstruction because of its natural contour. Sutured back to back, two pieces of ear cartilage can also create a straight columellar strut or dorsal only graft. It can be harvested through an anterior or posterior incision. As long as 5 mm of posterior wall and the root of the helix are preserved, almost the entire concha can be safely harvested.

For larger defects and strong midline support, rib cartilage is the best material. Our preference is the floating ninth and tenth ribs because they curve less than the sixth. This can be used for columellar strut, sidewall and dorsal graft. They are easily harvested from medial to lateral through a 2–5 cm incision with care to avoid a pneumothorax.

Bone is not generally used due to the risk of resorption and graft exposure.

Grafts must be precisely designed and curved to obtain the optimum shape. Horizontal mattress sutures can increase or decrease convexity and the grafts must be sutured firmly to residual skeleton, other grafts and to lining using PDS or permanent sutures.

Midline support

L-Strut technique

The L strut consists of a longitudinal piece of cartilage that is seated on the nasal radix and extended along the dorsum to the tip. The caudal part of the L is strongly sutured to the anterior nasal spine with X sutures.

Hinged septal flap

Described by Millard,²¹ this L-shaped flap is carved from the remaining septum and hinged on the caudal end of the septal bones. As it pivots upward, it augments the nasal angle and recreates the dorsal support. The caudal part of the L is strongly sutured to the anterior nasal spine.

Septal pivot flap

As described above, this can provide dorsal support as well as restoring lining of the mid-vault.

Cantilever rib graft

A longitudinal graft extending from the dorsum down to the tip, which does not require fixation support of the distal tip. Proximally it can be embedded in the frontal bone, screwed to the nasal bone, or both. In order to keep the same dorsal



(a)



(b)

Figure 32.14 Three-stage folded forehead flap. (a) Full-thickness amputation of part of the tip and part of the ala for a basal cell carcinoma. (b) Result one year after a three-stage folded forehead flap.

projection, it is often necessary to lower the nasal bone before fixing the cantilever graft.

The best material for cantilever graft is 9th and 10th ribs. Designed at the osteocartilaginous junction, the bony portion of the graft is in contact with the nasal bones while the distal cartilaginous portion keeps the nasal tip pliable and resists resorption.

Lateral support

After the restoration of the midline support, upper and lower lateral cartilages have to be reconstructed. Further extra-anatomical cartilage grafts are often required for the soft triangle, alar rim and ala. If these areas, which do not normally contain cartilage, are not braced, contraction during healing will tend to collapse and retract. Isolated alar defects are best treated with

grafts that are positioned and buried in adjacent subcutaneous pockets and secured with temporary percutaneous sutures.

Composite and extended defects

Frequently the defect may encompass several adjacent facial subunits, including the lip and the cheek. Each cosmetic area should be treated individually as each facial subunit has different reconstructive requirements that could not be recreated with a single flap. Several flaps with multiple stages will be required to recreate the features of the normal face.

The ideal nose has its foundations on a solid and symmetrical platform which should be reconstructed first and for this reason it is advised to start with maxillary, cheek and lip reconstruction followed by nasal reconstruction (Figure 32.15).

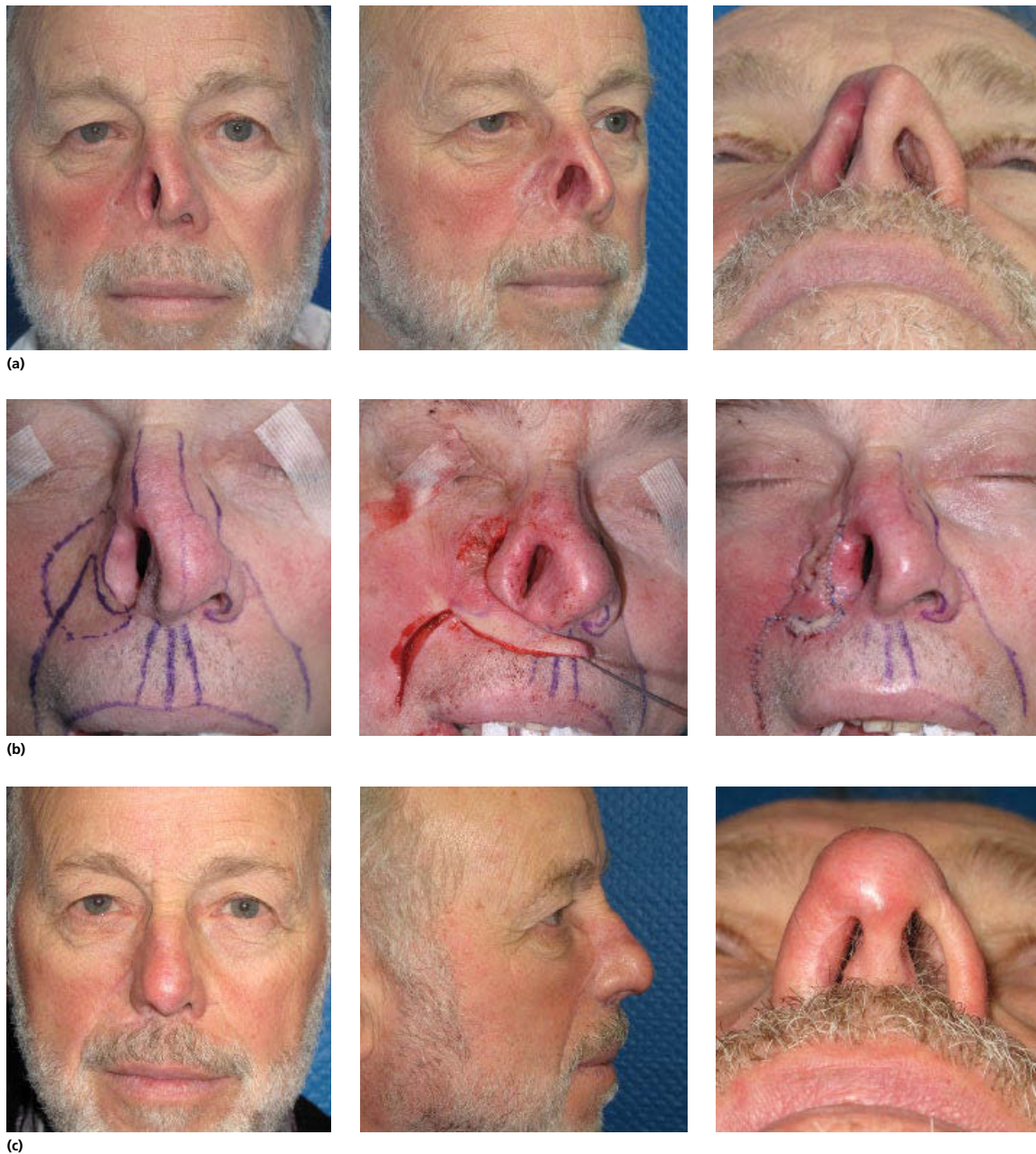


Figure 32.15 Reconstruction of composite defect. (a) After angiosarcoma resection, the defect encompasses the nose, the cheek and the upper lip (skin graft on the cheek and the upper lip). (b) Preliminary surgery consisted in replacing the wide skin graft of the cheek and lip with good-quality skin. Subunits of the lip were marked and skin graft outlined. Nasolabial flap and cheek advancement totally replaced the graft. Just six weeks later the nasal reconstruction was started. (c) Result one year after three-stage forehead flap.

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CHAPTER 33

Eyelid reconstruction

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Anatomy

A detailed knowledge of the anatomy of the periocular region is key to carrying out safe and effective reconstructive surgery.

The marginal eyelids are composed of two structurally and functionally important lamellae. The anterior lamella consists of skin and pretarsal orbicularis oculi muscle, and the posterior lamella consists of tarsus and conjunctiva. The tarsus measures approximately 8–12 mm in height in the upper eyelid and 3.5–4 mm in height in the lower eyelid. Beyond the tarsus the eyelids can be considered to have three lamellae: the *anterior lamella* consists of skin and preseptal orbicularis oculi muscle, the *middle lamella* consists of orbital septum and eyelid retractors and the *posterior lamella* consists of conjunctiva. Fat pads lying within the middle lamella serve as useful landmarks during surgery. The lamellar components of a defect should all be considered to create functional eyelids (Figure 33.1).

The eyelids receive a rich blood supply from branches of both the internal and external carotid arteries. Knowledge of the major blood vessels within the eyelids is important to ensure adequate haemostasis during surgery. The upper and lower eyelids both receive contributions from medial and lateral. Medially, the ophthalmic artery branches to form the superior and inferior medial palpebral arteries supplying the upper and lower eyelids respectively. These arteries form the marginal arteries present in both the upper and lower eyelids. The marginal arteries run on the anterior surface of the tarsus, 4 mm from the eyelid margin in the upper eyelid and 2 mm from the margin in the lower eyelid. In the upper eyelid an additional peripheral arterial arcade runs superior to the tarsus on the anterior surface of Müller's muscle. The marginal vessels and the peripheral arcade described anastomose with lateral contributions from the superior and inferior lateral palpebral arteries, which are branches of the lacrimal artery (Figure 33.2).

Function

Eyelids serve to protect the eyes. The ability to blink is important in maintaining a healthy ocular surface and being able to close the lids forcibly to protect against trauma. In addition to these physiological functions it is important not to underestimate the role of the appearance of the eyelids in human interaction. The eyelids are an important aesthetic subunit, derangement of which can result in significant psychological morbidity.

Reconstructive options

There are many reconstructive options that should be at the disposal of the oculoplastic surgeon, ranging from 'laissez faire' to the use of local, regional and distal flaps. The options should be considered in the context of the patient not just the defect. A technique that may be ideal for a certain defect may not suit all patients. For example, the tarsoconjunctival 'Hughes' flap for a lower eyelid defect may be unsuitable if it involves the only 'seeing' side of a one-eyed patient.

Aesthetic units

The face is split into a number of aesthetic units. These units should be respected to create the most naturally appearing outcome. The whole periocular region is considered to be one unit, and is further subdivided into four subunits:

- the medial canthus above the midline;
- the lateral canthus (crow's feet area);
- the lower eyelid; and
- the upper eyelid.

In addition to these areas we would recommend considering the upper eyelid in two parts: above and below the skin crease.

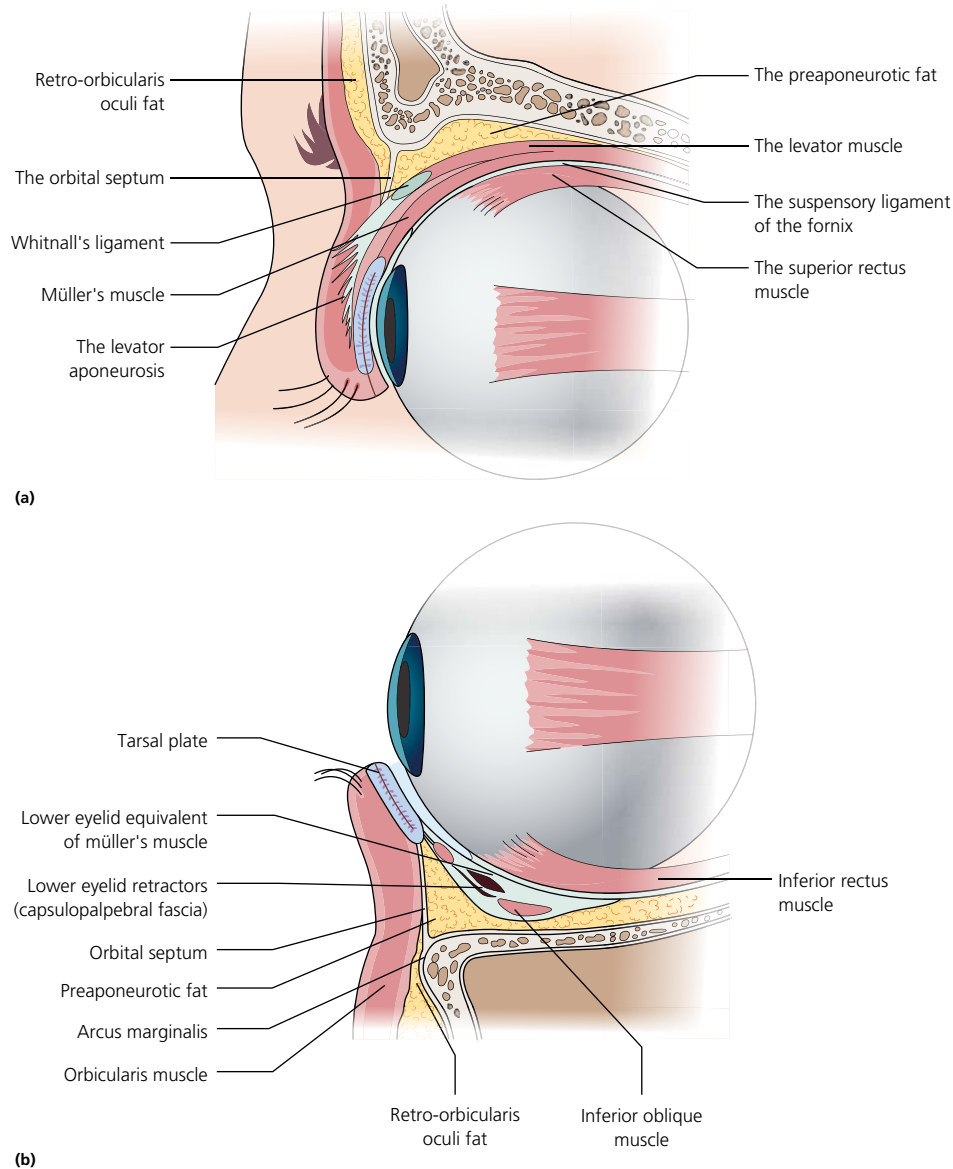


Figure 33.1 Cross-sectional anatomy of the upper (a) and lower (b) eyelids illustrating the lamellar structure. Source: *Oculoplastic Surgery*, 2nd edition, Brian Leatherbarrow. Reproduced with permission of Taylor & Francis.

Skin tension lines

A number of different skin tension lines of the face have been described, varying from Langer's lines to relaxed skin tension lines. The aim of these has been to aid the surgeon in choosing the direction of their incision wisely to minimize scarring. The variety of different theories published can be confusing and contradictory. In the periocular region wound healing is generally good, with relatively little scarring and the priority in incision site/direction is maintaining eyelid function. Thus despite relaxed skin tension lines and Langer's lines running parallel with the eyelid margins, defects are often closed perpendicular to the eyelid margin to prevent lagophthalmos. If lagophthalmos

is not going to be an issue, the following recommendations are made:

- In the upper eyelid, close wounds parallel to the eyelid margin and within pre-existing creases, especially the upper eyelid skin crease.
- In the lower eyelid, close wounds parallel to the eyelid margin. Subciliary wounds and wounds lying at the cheek/eyelid junction tend to heal very well.
- At the lateral canthus, close wounds within 'crow's feet'. These can be accentuated by asking the patient to smile.
- At the medial canthus, 'webbing' of skin over the concave surface should be avoided by closing wounds on the flat of the nose without undue tension.

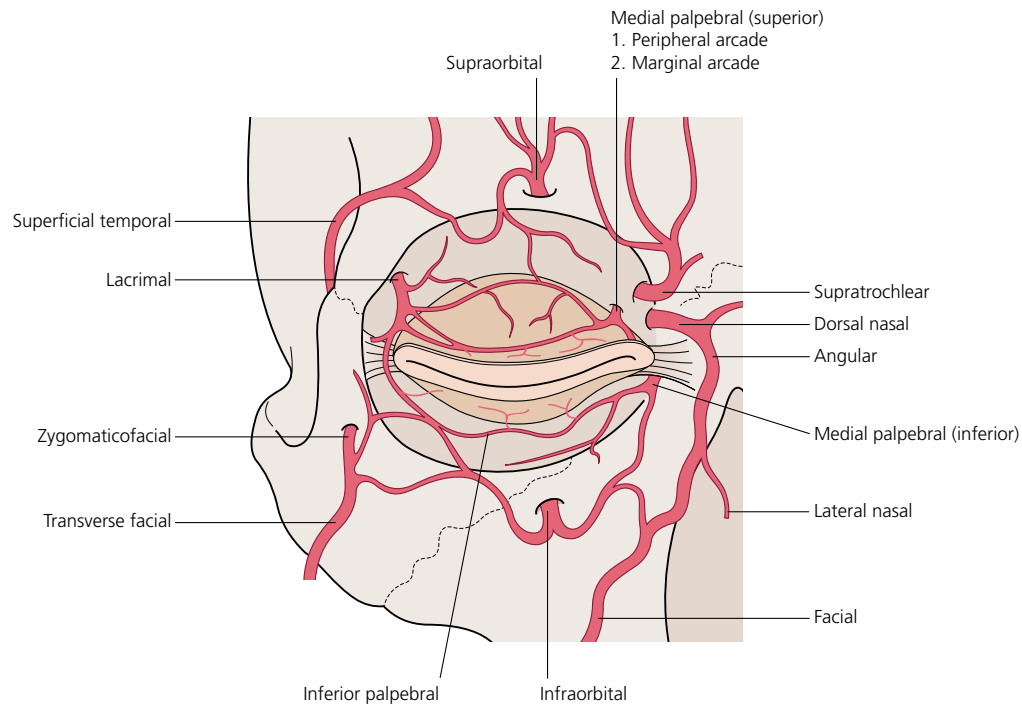


Figure 33.2 Arterial supply to the upper and lower eyelids. Source: Bauer & Margulis, 2004 [*Plast Reconstr Surg* 114: 98–106]. Reproduced with permission of Lippincott Williams & Wilkins.

- On the nasal bridge, wounds should be closed exactly horizontally.

In all instances careful marking of useful creases and lines before infiltration with local anaesthetic is important for accuracy.

Vector considerations

The periocular region is a complex, three-dimensional dynamic structure. There are a number of specific features that should be evaluated when considering reconstruction of this area.

- *The presence of a negative vector:* A negative vector describes the situation when the anterior surface of the cornea lies anterior to the malar prominence. This is an important feature to note when reconstructing the lower eyelid. A negative vector is a risk factor for lower eyelid retraction. This can be minimized by not overtightening the lower eyelid.
- *The circular orientation of the orbicularis oculi muscle complex:* The orbicularis oculi can be envisaged to run in a circular orientation around the eye with medial and lateral raphe. To repair a defect of the anterior lamella in this region a larger distance of the outer circumference should be closed compared to the inner circumference (i.e. a wedge shape should be closed).
- *The action of gravity and tissue restitution:* There is a tendency for tissue to return to its original position and there is a gravity effect. This is an important consideration when designing flaps to repair periocular defects. Flaps repairing defects below the canthi, where possible, should exert an upwards vector. This prevents problems with lower eyelid ectropion (e.g. a medial canthal rhomboid could be designed in a

number of ways – there are always four possible options). The recommendation is to design the flap so the tendency of the flap to return to the original site exerts an upward vector.

Patient factors

A periocular defect should be considered in the context of the patient as a whole. The patient's comorbidity, attitude to treatment, geographic location and support structure will all influence the choice of reconstructive technique.

Basic techniques

'Laissez faire'

'Laissez faire' is the principle of not interfering with the healing process of the body. It can be applied to a large range of defects, being favoured for defects in the medial canthal region and for full-thickness lower eyelid defects. The limitations of the approach should be understood. Alternative approaches should be sought if the defect:

- compromises the ocular surface (e.g. full-thickness upper eyelid defects);
- may result in eyelid malposition (e.g. lower eyelid anterior lamella defects in a lax eyelid).

Direct closure

Direct closure is an ideal technique for managing full-thickness upper and lower eyelid defects (Figure 33.3). There are no strict rules regarding the size of defect that can be closed; rather, the defect must

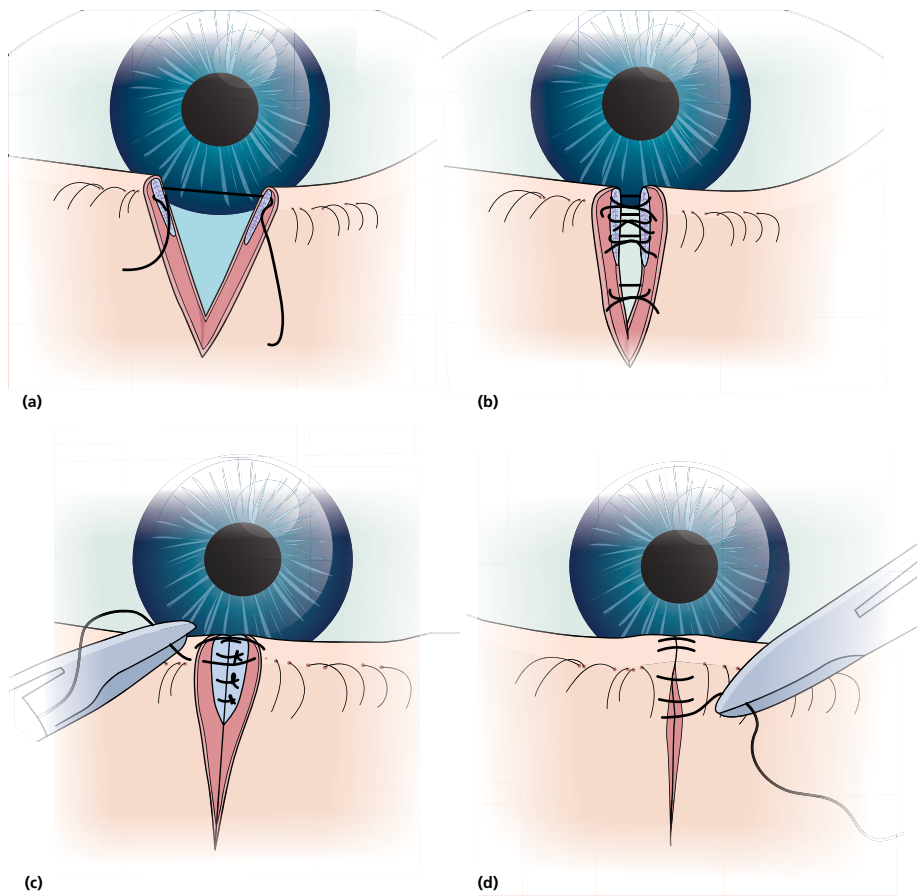


Figure 33.3 Direct lamellar closure of the eyelid. Source: *Oculoplastic Surgery*, 2nd edition, Brian Leatherbarrow. Reproduced with permission of Taylor & Francis.

be considered in the context of the surrounding tissue laxity. In the young patient a defect less than a third of the eyelid width may be reasonably closed, whereas in the older patient with significant laxity a defect measuring over half the eyelid width may close using this technique. The technique can be used on larger defects in the lower eyelid by carrying out a lateral cantholysis; although this risks the development of a rounded lateral canthal angle.

Surgical technique

- 1 The surgeon's preference of local anaesthetic is infiltrated throughout the region of the defect and the lateral canthus if a lateral cantholysis is being considered.
- 2 The full face is prepared with antiseptic solution and draped in a sterile fashion.
- 3 The margins of the defect are cleaned.
- 4 The defect margins are grasped with toothed forceps and approximated to each other to ensure the defect can be closed without undue tension.
- 5 If a lateral cantholysis is required it should be carried out now.
- 6 A lateral cantholysis is carried out through a conjunctival incision from the lateral canthus directed inferoposteriorly only a few millimetres.
- 7 The lateral cantholysis (if necessary) is performed in a graded fashion until adequate laxity has been generated.

- 8 Absorbable 5-0 braided suture is used to close the tarsal plate ($\times 2-3$ in the lower eyelid and $\times 3-4$ in the upper eyelid). Care is taken to ensure suture material is not visible through conjunctiva and that the lid margin and conjunctival surfaces are aligned.

- 9 Non-absorbable 6-0 monofilament suture is used to close the grey line and lash line. These sutures are placed in a vertical mattress fashion to produce a positive 'pucker' to minimize the risk of a lid margin notch. These sutures are left long so they can be secured to the skin to prevent corneal irritation.
- 10 The skin is closed with 6-0 monofilament, non-absorbable suture.

Postoperative management

A topical antibiotic ointment can be applied three times a day to both the eye and wound for up to two weeks. All sutures can be removed at 5–10 days and cool packs applied for 48 h.

The Tenzel flap

This flap is useful in the situation where there is a full-thickness marginal eyelid defect that will not close despite complete lateral cantholysis. Skin from beyond the lateral margin of the lateral commissure is recruited to form new lateral eyelid margins.¹

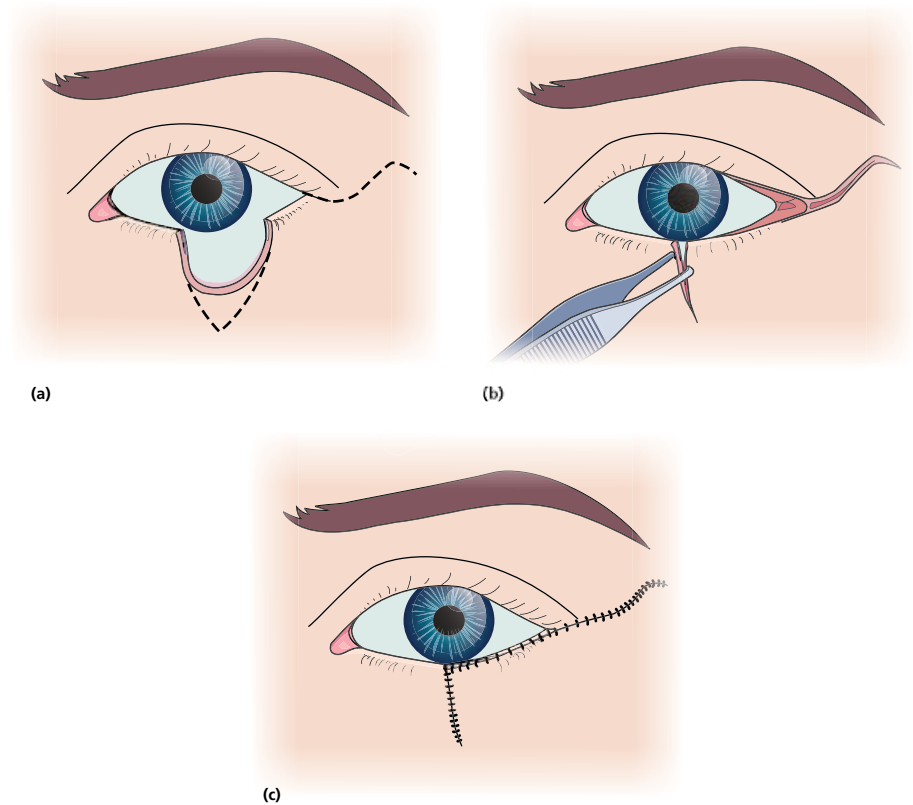


Figure 33.4 The Tenzel semicircular flap modified with a steeper initial incision. Source: *Oculoplastic Surgery*, 2nd edition, Brian Leatherbarrow. Reproduced with permission of Taylor & Francis.

The Tenzel flap was described to aid the closure of lower and upper eyelid defects. It is a semicircular rotation and advancement flap (Figure 33.4).

Surgical procedure

- 1 The Tenzel flap is designed by following the natural contour of the eyelid at the lateral canthus accentuating the natural curvature upwards then back down to form a semicircle. A relatively steep gradient at the lateral canthus is recommended, with some authors recommending an initial vertical cut.² The high point of the flap should remain below the level of the brow.
- 2 The flap marking is incised and extended to the plane of dissection, which should extend no deeper than the superficial temporal fascia, and should remain superficial when approaching the zygomatic arch to prevent damage to the temporal branch of the facial nerve.
- 3 A lateral canthotomy and complete cantholysis is carried out. The lateral lower eyelid should be free from all deep attachments and the margin in smooth continuation with the flap.
- 4 The flap is suspended a few millimetres superior to the intended position of the lateral canthus at the lateral orbital rim with a 5-0 braided, absorbable suture.
- 5 The original defect is closed in the manner described previously.
- 6 The lateral incision site is closed with 6-0 non-absorbable, monofilament suture.

Postoperative management

As described above.

The Rhomboid flap

The rhomboid flap is a versatile flap that is used to transpose tissue from one area to another. If designed appropriately it improves the vector of tension compared to a direct closure and recruits tissue from areas of laxity.

In periocular reconstruction the rhomboid flap is particularly useful at the medial canthus. The flap recruits lax tissue from the bridge of the nose leaving an insignificant horizontal scar across the bridge of the nose and a U-shaped scar at the medial canthus that is often well hidden by glasses.

The Hughes tarsoconjunctival flap

The Hughes flap recruits tarsus and conjunctiva from the upper eyelid to reconstruct the posterior lamella of the lower eyelid. The relatively avascular tarsus retains a blood supply from a conjunctival pedicle kept attached to the superior fornix.³ The conjunctival pedicle is divided at 2–6 weeks.^{4,5} The flap is ideally suited to reconstruct a shallow, wide full-thickness lower eyelid defect. If the whole width of the lower eyelid needs to be reconstructed the Hughes flap can be secured at its lateral and/or medial end with periosteal flaps.⁶

Surgical technique

- 1 The surgeon's preference of local anaesthetic is infiltrated throughout the region of the defect in the lower eyelid and subcutaneously in the upper eyelid.
- 2 The full face is prepared with antiseptic solution and draped in a sterile fashion.
- 3 The margins of the defect are cleaned (Figure 33.5).
- 4 A 4-0 silk traction suture is placed through the grey line of the upper eyelid and the eyelid is everted over a Desmarres retractor.
- 5 The width of the lower eyelid defect is measured.
- 6 The donor tarsus from the upper eyelid is marked – width is equal to the lower eyelid defect; height is equal to the height of the upper eyelid tarsus minus 3.5 mm. A minimum of a 3.5 mm marginal strip of tarsus should be left in the upper

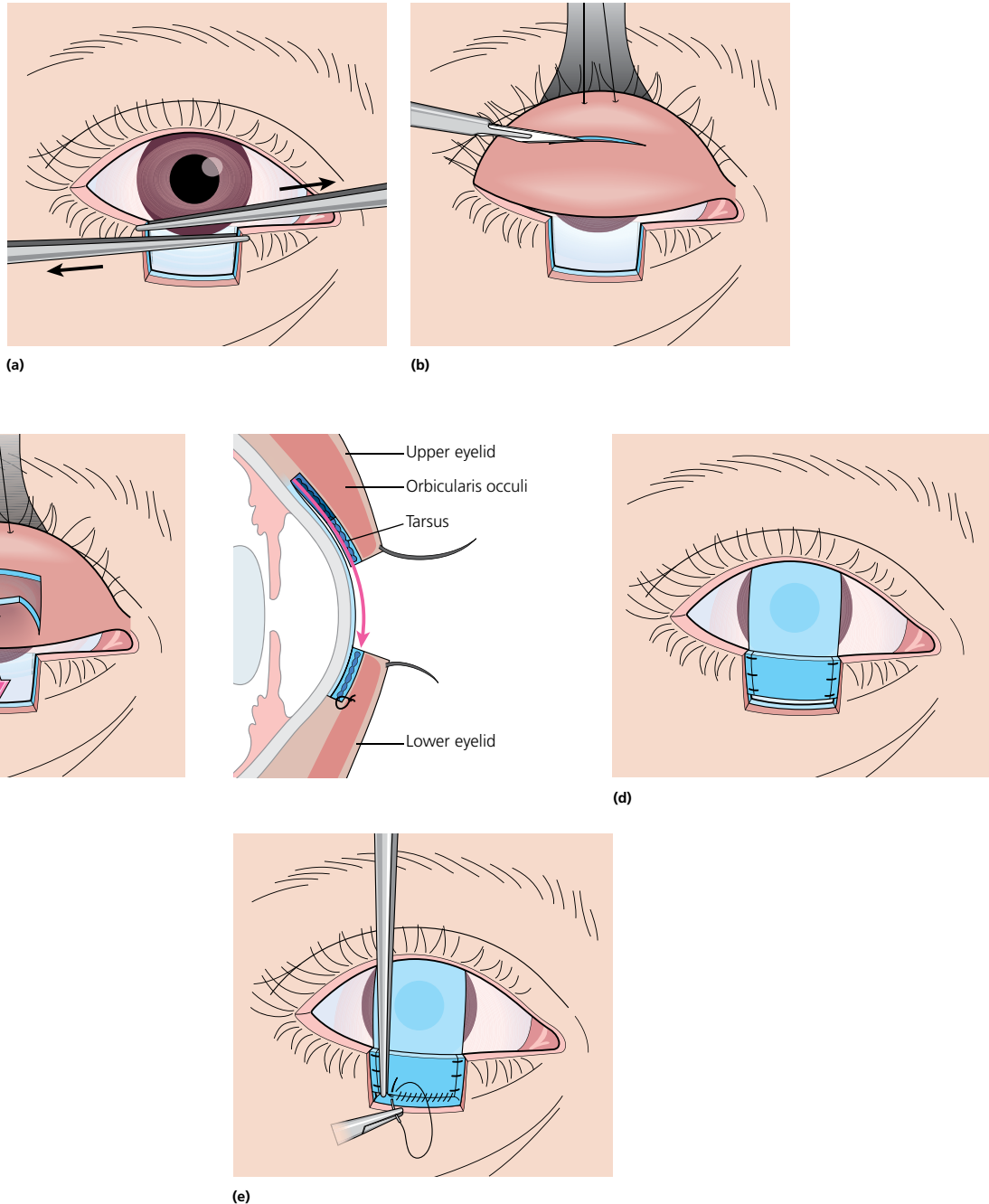


Figure 33.5 (a) The lower eyelid defect is cleaned. (b) The upper eyelid is everted over a Desmarres retractor and incised leaving at least 3.5 mm between the incision and the eyelid margin. (c) Vertical incisions are made through the tarsus towards the upper border of the tarsus and the tarsus dissected off underlying orbicularis. The dissection is continued between Müller's muscle and conjunctiva to create a tarsal flap suspended from the upper eyelid by conjunctiva. Right: eyelid anatomy. (d) The tarsal flap is secured medially and laterally to remnant lower eyelid tarsus or periosteal flaps if no tarsus remains. (e) The lower border of the tarsus is secured to conjunctiva and inferior retractors. Source: *Atlas of Oculoplastic and Orbital Surgery*, Jonathan Dutton, 2013. Reproduced with permission of Lippincott, Williams and Wilkins.

eyelid to ensure it remains stable so an upper eyelid entropion does not develop.

- 7 The donor tarsus is incised and dissected from the underlying orbicularis.
- 8 The dissection is continued to a subconjunctival plane.
- 9 Care should be taken dissecting the conjunctiva from Müller's muscle to prevent excessive bleeding and conjunctival buttonholes.
- 10 The donor tarsus should now be freely mobile on a wide-based conjunctival pedicle.
- 11 The donor tarsus is secured into the defect with 5-0 braided absorbable suture ($\times 2$ at each end). If no remanant tarsus exists, the Hughes flap can be secured to periosteal flaps reflected from the lateral orbital rim or the lateral wall of the nose.
- 12 The inferior border of the donor tarsus is secured to the subconjunctival tissue with a continuous 7-0 braided absorbable suture.
- 13 The posterior lamella has now been reconstructed.
- 14 A number of techniques can be employed to reconstruct the anterior lamella.

Postoperative management

A topical antibiotic ointment can be applied three times a day to both the eye and wound for up to two weeks. The conjunctival pedicle is divided under local anaesthetic in 2–6 weeks.

Tripier and hemi-Tripier flaps

The Tripier flap is a bipediced myocutaneous flap taken from the upper eyelid to reconstruct the anterior lamella of the lower eyelid (Figure 33.6).⁷ The flap is designed to maintain myocutaneous innervation. The excellent blood supply of the eyelids means that the flap can survive with only a single pedicle (the hemi-Tripier flap), usually lateral. The flap is useful in patients with significant upper eyelid dermatochalasis with a lower eyelid defect extending to either or both the medial and lateral canthi. The flap is for anterior lamella reconstruction only, hence, if the defect is full thickness an additional technique will need to be applied to address the posterior lamella. There have been descriptions of 'reverse' Tripier flaps,⁸ using lower eyelid anterior lamella to reconstruct an upper eyelid defect, however, we would caution readers to select patients carefully if this approach is being considered due to the high risk of lower eyelid retraction. The hemi-Tripier flap can be used to reconstruct the full width of the lower eyelid, however, tenuous vascularity of the distal segment of the flap may be inadequate support for a posterior lamella graft, therefore, we use this technique for full-width reconstructions only when the posterior lamella is not being grafted.

Surgical technique

- 1 The upper eyelid skin crease is marked and a maximum blepharoplasty mark made. At least 20 mm of upper eyelid skin must be preserved. The marking outlines the largest flap that can be harvested.

- 2 The medial and lateral ends of the blepharoplasty marking are swept superiorly to define the pedicles of the flap. Efforts should be made to blend these marks into pre-existing rhytids. The flap is designed in such a way that there is traction in a superolateral and superomedial direction at the lateral and medial ends of the flap respectively.
- 3 The surgeon's preference of local anaesthetic is infiltrated throughout the region of the defect in the lower eyelid and subcutaneously in the upper eyelid.
- 4 The full face is prepared with antiseptic solution and draped in a sterile fashion.
- 5 The margins of the defect are cleaned.
- 6 A 4-0 silk traction suture is placed through the grey line of the upper eyelid.
- 7 A template of the lower eyelid defect is made.
- 8 The template is transferred to the upper eyelid, the lower margin aligned with the upper eyelid skin crease marking.
- 9 A new superior blepharoplasty incision is marked including the template plus an additional 20% height to allow for contraction.
- 10 The lower eyelid defect may need to be enlarged at the canthi to accommodate the ends of the flap.

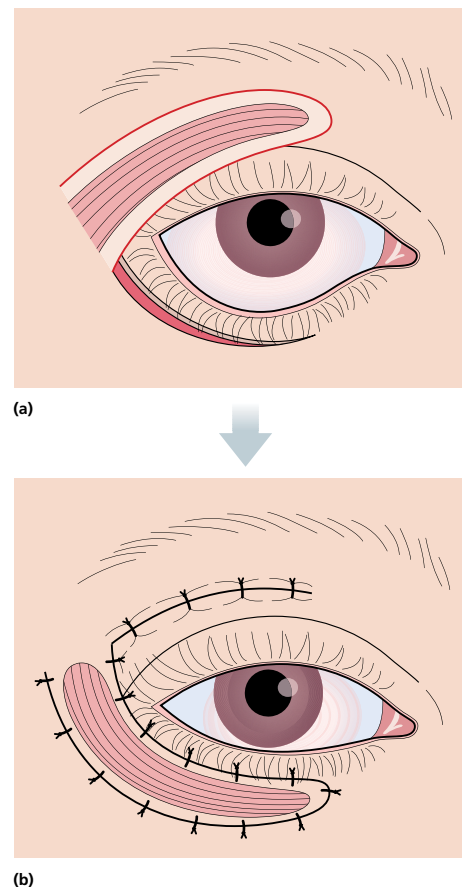


Figure 33.6 A laterally based hemi-Tripier flap to reconstruct the lateral lower eyelid anterior lamella. Source: *Reconstructive Facial Plastic Surgery: A Problem-solving Manual*, Hilko Weerda, 1999. Reproduced with permission of Thieme.

- 11 The upper eyelid flap is dissected in the suborbicularis plane leaving the septum intact. If the flap only has one pedicle, we recommend tapering the thickness of the flap from its base to its tip to reflect the increasingly tenuous blood supply along the flap.
- 12 The flap is secured into the defect with no tension using 6-0 non-absorbable monofilament suture and the upper eyelid defect closed with the same suture material.

Postoperative management

A topical antibiotic ointment can be applied three times a day to both the eye and wound for up to two weeks. All sutures can be removed at 5–10 days and cool packs applied for 48 h.

Cheek rotation flap

The cheek rotation flap is used for total reconstruction of the anterior lamella of the lower eyelid (Figure 33.7). The posterior lamella should be reconstructed separately, commonly using a graft (see below).

Surgical procedure

- 1 The cheek rotation flap is marked by following the natural contour of the lower lid upwards at the lateral canthus onto the temple then turning inferiorly in front of the ear. As with the Tenzel flap, height of the flap is important to prevent a downwardly displaced lateral canthus.
- 2 The flap marking is incised and extended to the plane of dissection, which should extend no deeper than the superficial temporal fascia, and should remain superficial when approaching the zygomatic arch to prevent damage to the zygomatic branch of the facial nerve. The cheek dissection should be extensive to allow mobility of the flap and carried out in the subcutaneous/fat plane.

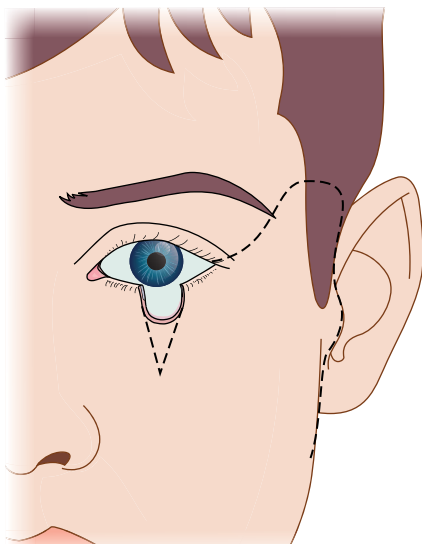


Figure 33.7 Marking for a cheek rotation flap. The 'standing cone' that would result from the flap rotation has been marked inferior to the defect – this is to be excised. Source: *Oculoplastic Surgery*, 2nd edition, Brian Leatherbarrow. Reproduced with permission of Taylor & Francis.

- 3 The flap is rotated into position.
- 4 Standing cone/dog-ear deformities may need to be excised both in the preauricular region and the cheek.
- 5 The highest point of the flap is suspended a few millimetres superior to the intended position of the lateral canthus at the lateral orbital rim with a 5-0 braided, absorbable suture.
- 6 The flap is secured with deep 5-0 braided, absorbable suture and 6-0 non-absorbable monofilament used to close the skin.

Postoperative Management

We recommend application of a firm dressing for 3–5 days to reduce the risk of haematoma formation. A topical antibiotic ointment can be applied three times a day to both the eye and wound for up to two weeks. All sutures can be removed at 10 days.

Full-thickness skin grafting

Full-thickness skin grafting is a versatile technique for anterior lamella reconstruction. There are some general principles that should be adhered to. Full-thickness skin grafts gain a tenuous blood supply from the tissue they are placed upon, therefore, to maximize the chance of viability they should be:

- *thin* – the graft should be thinned to the level of the dermal Rete pegs;
- *placed on a well-vascularized bed* – for example, when placing a skin graft on a Hughes flap, which is relatively avascular, a 'bucket handle' of orbicularis oculi muscle can be interposed to provide a more robust vascular base; in areas where there is no vascular base in the immediate vicinity, a pericranial flap can often be fashioned to provide one;
- *static* – to develop a blood supply the graft should initially be kept static on its vascular bed; this may necessitate a light compression dressing and/or immobilization of the eyelid with 'Frost' sutures.

Donor site choice

There are number of areas that skin can be harvested from; with pros and cons for each choice. All donor sites should be hair free.

- *The upper eyelid*: In patients with significant dermatochalasis, the upper eyelid can be a useful donor site. The skin here is usually a good colour match for the rest of the periocular region and is thin. Disadvantages include a relatively limited supply and the presence of sun damage.
- *The pre- and postauricular region*: Advantages include relatively insignificant donor scars and a reasonable colour match. Disadvantages include limited availability and thicker dermis.
- *The supraclavicular region*: Advantages include a donor scar that can usually be well hidden (however, counsel patients carefully as they may want to expose this region) and a relatively large reservoir. Disadvantages include occasional hairs in this area and thicker dermis.
- *The inner upper arm*: Advantages include a large reservoir and it is usually free from significant sun damage. Disadvantages include a colour mismatch and thicker dermis.

Surgical technique

- 1 The surgeon's preference of local anaesthetic is infiltrated throughout the region of the defect and the donor site.
- 2 The full face and donor site is prepared with antiseptic solution and draped in a sterile fashion.
- 3 The margins of the defect are cleaned.
- 4 A template is made of the defect using plastic drape or sterile paper.
- 5 The template is transferred to the donor site and traced around.
- 6 The skin graft is cut from the donor site – oversizing it slightly – with a combination of a blade and scissors. Cautery to the graft is not recommended.
- 7 Care is taken to thin the skin graft to the level of the Rete pegs.
- 8 The graft is sutured in place with interrupted 6-0 non-absorbable, monofilament suture.
- 9 The graft is immobilized with a light compression dressing and/or a Frost suture.

Postoperative management

The dressing can be removed 1–5 days post op depending on the size of the graft. Sutures should be removed on day 5–7. A topical antibiotic ointment can be applied three times a day to both the eye and wound for up to two weeks.

Key to the success of a skin graft is careful postoperative management. Massaging with or without a silicone dressing and steroid injection into thickened areas can be important in achieving a satisfactory outcome.

Posterior lamella grafting

The tarsus is an important structural element of the eyelids. It is responsible for maintaining a stable eyelid margin position. If there is significant loss of tarsus leading to a potentially unstable eyelid margin, thought should be given to replacement of this structure. The Hughes flap has already been discussed, however, this is not suitable for every case. Free grafts can be considered. In situations where there is no canthal tendon support either laterally or medially to secure the posterior lamella replacement, a periosteal flap can be reflected from the lateral orbital rim or side of the nose respectively.

Tarsal replacement graft donor sites

- *Contralateral upper eyelid:* The upper part of the tarsus of the upper eyelid can be harvested for use as a graft. At least 3.5 mm height of tarsal plate should be left behind to avoid destabilizing the donor eyelid. The advantage of this graft is that it is identical tissue and has a conjunctival covering. Disadvantages include possible donor site morbidity and a limited size graft.
- *Hard palate:* This has the advantage of being readily harvested in an adequate size with a mucosal covering. Disadvantages

include the potential to cause corneal abrasion as the mucosal surface tends to be rougher than conjunctiva, hence, caution should be exercised when using this in the mobile upper eyelid.

- *Auricular cartilage:* This can be used as a structural replacement for tarsus. Disadvantages include a lack of mucosal covering, therefore, this must be considered separately. The cartilage is also not flat, therefore, must be scored and sculpted to create an appropriate shape. Despite shaping, the graft often returns to its original shape that can distort the eyelid.
- *Nasal septal cartilage:* Similar to auricular cartilage, nasal septal cartilage can be used as a structural replacement for tarsus, however, it has no mucosal covering. An advantage is its relatively flat shape, disadvantages include possible donor site comorbidity (e.g. nasal strut collapse and septal perforation).

Discussion

Successful periocular reconstruction demands an intimate knowledge of the functional anatomy of the eyelids and an appreciation of aesthetics. The solution for managing a patient's periocular defect must be bespoke and often requires imagination.

This chapter does not provide a definitive guide to periocular reconstruction, but it does provide the basic tools required to manage the vast majority of situations.

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CHAPTER 34

Ear reconstruction

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A variety of traumatic ear defects are observed after amputation, whether partial, subtotal or total, all of which require complex contour reconstruction.

There are multiple causes of ear amputations and this summary aims to define some principles and guidelines that permit a precise surgical management plan.

Initial assessment concerning a posttraumatic amputation

Mechanism of ear amputation

Clean amputations

Clean amputations are usually secondary to trauma involving a knife, surgical resection or sharp bites (animals or human). Primary treatment consists of debridement and suturing of the edges of the wound.

If we exclude surgical amputation for cutaneous malignancy, it is tempting to replant the amputated segment of ear. Many different techniques have been proposed to ensure success of such replantation (burying, fibrocartilage fenestration, cartilage storage and secondary replantation).

Whatever the degree of success of such surgical approach, the final result will be disappointing. Even though it may be difficult to make the decision not to attempt replantation and to sacrifice the amputated ear segment, this will preserve the skin potential for a superior secondary reconstruction.

Replantation should only be considered when vascular anastomosis is possible and reliable.

Crush injury

A crush injury occurs after a fall, road traffic accident, blunt trauma or violent compression. In such circumstances the ear defect is generally only part of a complex injury involving the surrounding soft tissue.

Burn injury

Thermal, chemical or electrical burn injuries are a frequent cause of traumatic ear defects. Both ears are frequently affected and the burn area extends around the auricular area. Besides the zone directly injured by the burn, a secondary chondritis often results in severe resorption of the fibrocartilage.¹ When limited to the ear, the defect usually affects the scapha and helix, but often the retro-auricular skin and scalp are also affected with scars and alopecia.

Burn patients are generally managed in a burns unit where initial stabilization and grafting are performed. Reconstruction of the ear is a last step of management. Those patients who have undergone multiple surgical procedures may opt not to have an ear reconstruction. In some cases, an ear prosthesis is indicated.

Analysis of the defect

A precise analysis of the defect is critical in all reconstructive efforts. The missing contours must be appreciated and reconstructed in all dimensions.

To correctly appreciate the defect it is essential to draw the normal ear on a transparent template, and place it on the injured ear to appreciate the missing contours needing reconstruction. Skin alone is not adequate to reconstruct the contours of the ear, and depending upon the size of the defect, either fibrocartilage or rib cartilage is required as part of the reconstruction.

Analysis of the skin potential

The specific problem encountered in cases of posttraumatic amputation is the presence of scars. It is essential to analyse their location and the skin availability to cover the reconstructed cartilage construct. The following three factors need consideration:

- The quality and quantity of skin in the auricular area must be appreciated. The presence of lax and scar-less retro-auricular skin provides the best conditions for coverage of the cartilage framework.

- Scars. After inspection of scars a surgical approach is chosen to minimize the use of new scars and ensure vascularity of the skin to cover the cartilage construct.
- Deficit of skin. In cases where there is no skin available in the auricular area, surrounding fascia (temporoparietal or occipital) or the use of skin expansion will have to be considered to cover the cartilage framework.

Reconstruction in partial amputations

Partial traumatic defects of the ear are always located along the free border of the ear. In this discussion we will exclude defects of the helix that can be closed directly or using skin flaps or chondro-cutaneous flaps. The Antia Buch flap² can be used for small defects but its principle of moving the helix after a scaphoid resection always reduces the size of the ear.

In partial amputation, skin availability is usually adequate and the main concern is to choose the best support to reproduce the missing cartilage contours. Either fibrocartilage or rib cartilage may be used. It is essential to draw the normal ear on a template, and then transpose the template on the affected ear to work out what is missing and what contours need reconstructing.

The rule is that fibrocartilage can only be used to reconstruct a partial defect after trauma if the it is no more than a quarter of the border of the ear and includes no more than two adjacent planes. Harvesting all of the concha can only reproduce two adjacent planes and just a quarter of the ear. If the defect is less than a quarter but includes three planes, then rib cartilage should be harvested to correctly reproduce the missing contours (Figure 34.1).

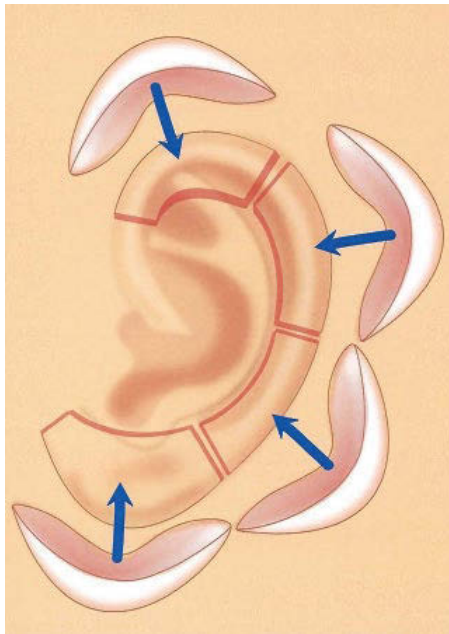


Figure 34.1 Fibrocartilagenous conchal graft can only be used when the defect is no more than a quarter of the ear and no more than two adjacent planes.

Reconstruction using fibrocartilage

Harvesting the fibrocartilage graft

The concha is the best donor site considering its size and the fact that it will not be visible if harvested correctly. The entire concha has to be harvested, taking care to preserve the root of the helix and enough posterior wall of the concha.

Depending on the case it can be harvested through an anterior or posterior approach from the ipsi- or contralateral ear.

The choice concerning the skin approach (anterior or posterior) depends on the defect and the skin vascularity. The choice concerning the harvesting on the ipsi- or contralateral side may be discussed with the patient, who sometimes is reluctant to have surgery performed on the normal ear.

Depending on the location of the defect and the skin potential the reconstruction will be performed in one or two stages.

The case in Figure 34.2 shows a reconstruction in one stage using an ipsilateral conchal graft. The defect is secondary to

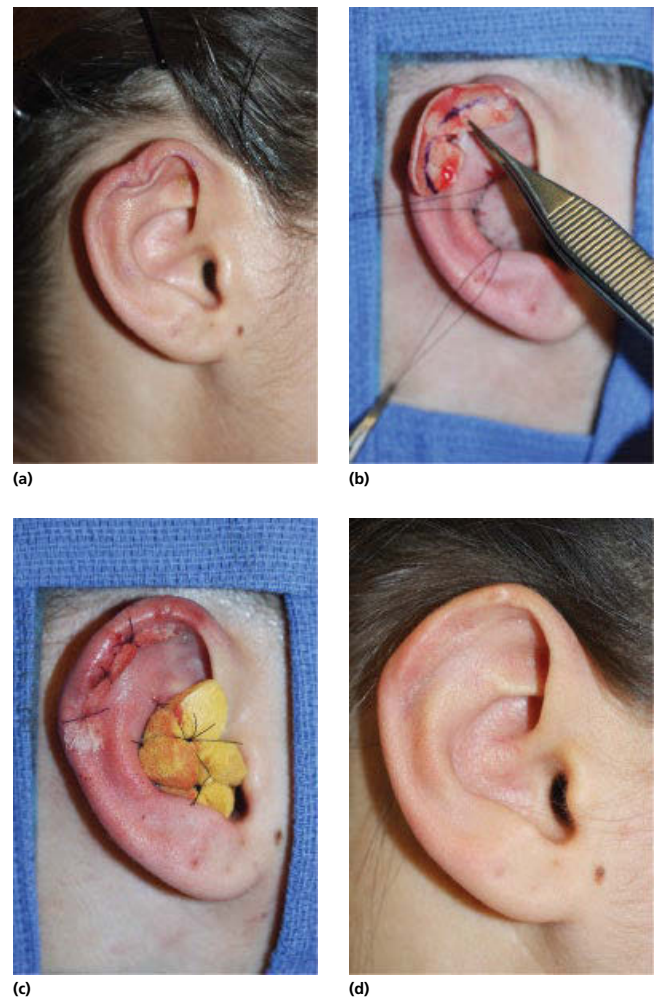


Figure 34.2 (a) The defect is a quarter of the ear and no more than two planes. (b) The conchal graft harvested on the same ear by an anterior approach will be reduced to reproduce the missing contours. (c) Bolster sutures apply the skin to the underlying contours. (d) Result.

chondritis after trauma. It is a quarter of the contour of the ear and no more than two adjacent planes.

In the case shown in Figure 34.3, because the defect is a quarter and no more than two planes the reconstruction was performed using a contralateral conchal graft covered with retro-auricular skin. Creation of the retro-auricular sulcus behind the reconstructed part of the ear will be performed in a second stage. Elevation of the ear is performed, respecting the soft tissues behind the ear, to cover the area with a small full-thickness skin graft harvested from the opposite retro-auricular sulcus.

Reconstruction using costal cartilage

There is an abundant amount of cartilage when harvesting from the thorax but when harvesting for reconstruction of partial defects, only one segment of cartilage is required. In such situations the scar on the thorax will be short.

Harvesting cartilage is a routine procedure with minimal risk. Pain is avoided by injecting bupivacaine (Naropin USA) into the muscles.

Using only a skin flap to reconstruct a partial amputation located on the periphery of the ear cannot reproduce complex contours and will give a poor result.

It is important to differentiate cases where the entire antihelix has been preserved from the ones where part of it is missing, particularly the posterior wall of the concha. If part of the antihelix is missing it has to be reconstructed respecting the anatomical unit of the antihelix and the base that supports it.

Cases with normal antihelix

Figure 34.4 shows a clean amputation with a defect that is no more than two adjacent planes but more than a quarter of the ear of the border of the ear, requiring the use of rib cartilage support. The

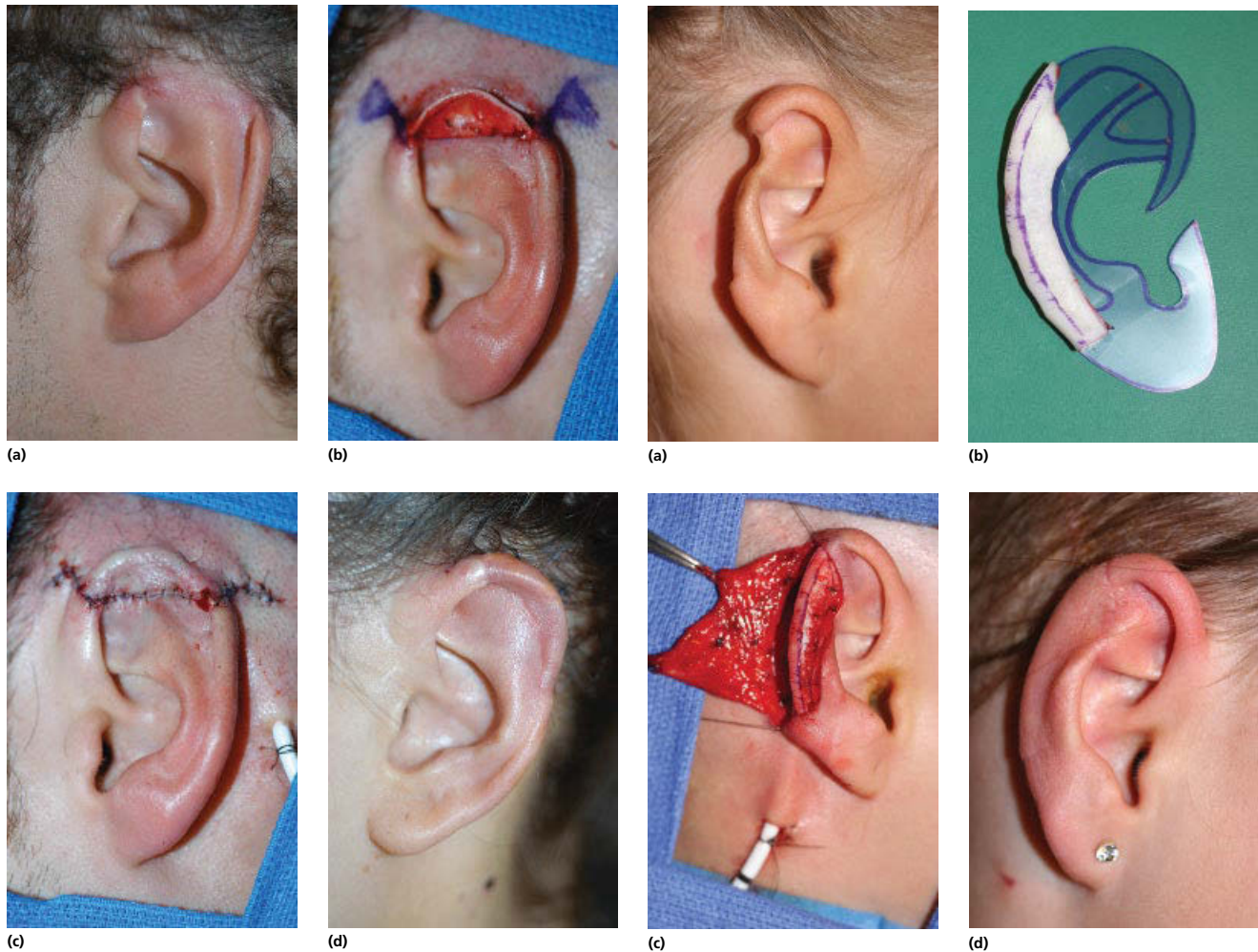


Figure 34.3 (a) The defect is a quarter and no more than two adjacent planes. (b) The conchal graft harvested on the contralateral ear is sutured to the fibrocartilage and the retro-auricular skin will cover it. (c) Appearance after the first stage. (d) Result after elevation of the reconstructed contours using a full-thickness skin graft harvested from the contralateral retro-auricular sulcus.

Figure 34.4 (a) Even though the defect is no more than two adjacent planes, it is more than a quarter of the border of the ear. (b) Perfect adaptation of the sculpted rib cartilagenous graft to the contours of the other ear drawn on a template. (c) Coverage of the reproduced contours by an advanced retro-auricular skin flap. (d) Result obtained in one stage.

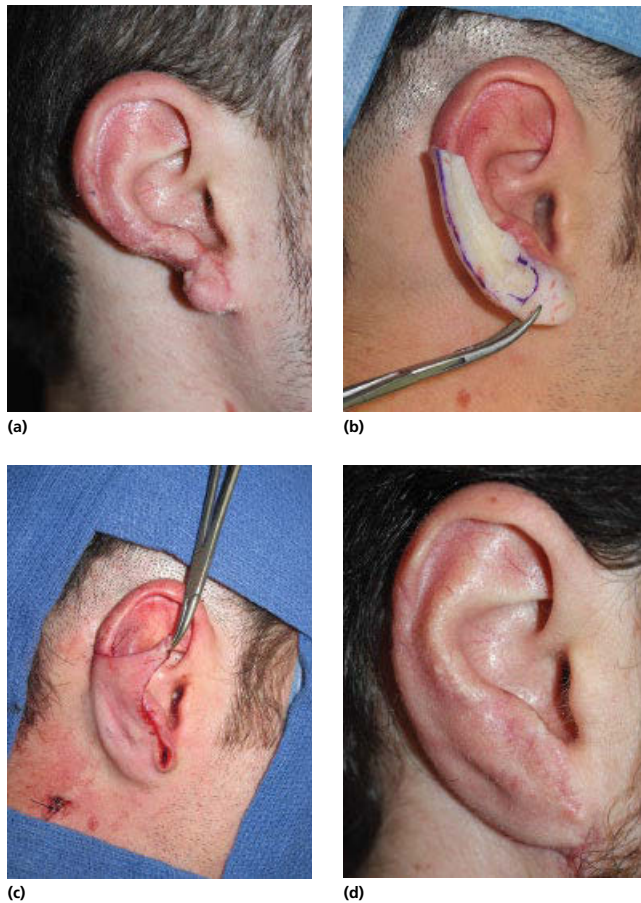


Figure 34.5 (a) Defect after unsuccessful replantation. (b) Rib cartilaginous graft reproducing the missing contours. (c) Skin flap covering the reconstructed contours. (d) Final result after elevation using a split-thickness skin graft harvested from the scalp.

reconstruction can be performed in one stage as there is enough retro-auricular skin to cover the narrow cartilaginous graft.

The amputation in Figure 34.5 is secondary to a human bite. An unsuccessful replantation has left a defect involving half of the inferior border of the ear, including the lobule. The reconstruction was performed in two stages with a very good result. The former scar was used to prepare the flap to cover the cartilage framework during the first stage. Advancement of the retro-auricular skin and grafting the posterior surface of the reconstructed ear was done four months later during the second stage.

In the case shown in Figure 34.6, using a rib cartilage graft on a 60-year-old patient was possible as there is no correlation between age and ossification. The texture and colour of the cartilage will just be different. The rib segments are thicker so the scapha and helix can be sculpted in one segment. Elevation of the reconstructed upper third of the ear can be performed four months later.

Cases with an injured antihelix

In cases where the antihelix has been destroyed, a framework including a base must be fabricated to be able to fix the antihelix to obtain correct projection of the antihelix and middle part of the ear.

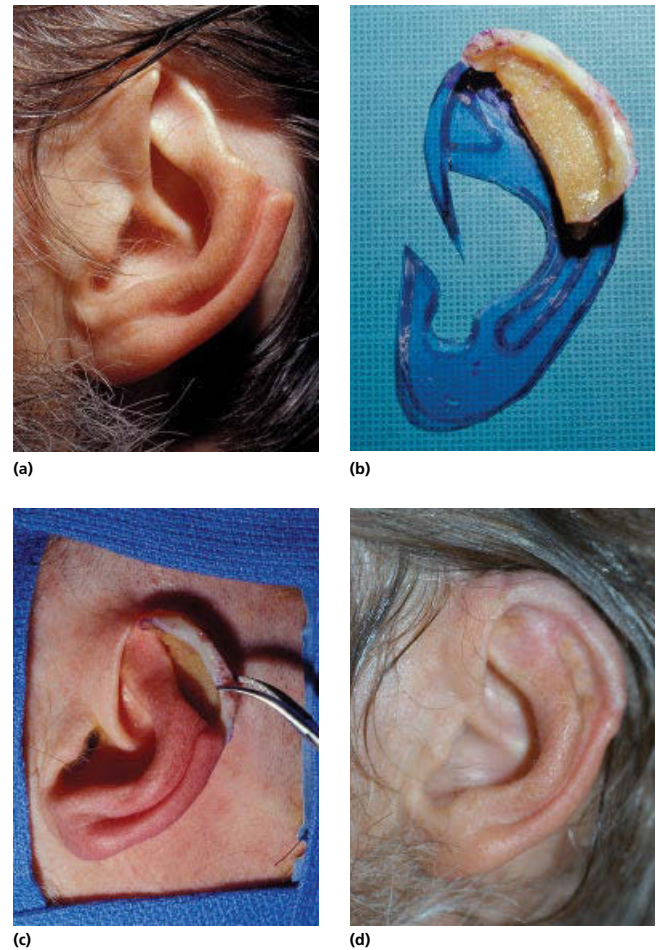


Figure 34.6 (a) The defect is more than a quarter and only two planes (helix and scapha). (b) Cartilaginous rib graft sculpted after appreciation of the missing contours using a transparent template. (c) Good match of the graft with the contours of the ear. (d) Result after elevation of the upper part of the ear using a skin graft.

Figure 34.7 shows amputation secondary to a dog bite. It is clear that part of the antihelix is missing and that the entire antihelix anatomical unit has to be reconstructed.

Even though this patient is 52, her cartilage is not ossified and presents with the usual yellow discolouration and is particularly dense.

Total and subtotal amputation

Total amputation does not always imply that there are no auricular remnants. Total or subtotal amputation implies that most of the contours are missing and that the reconstruction has to be done with a cartilage framework reproducing all of the ear's contours with rib cartilage.

A framework will be carved from several segments of ribs. Even though many synthetic materials have been proposed as an alternative to rib cartilage, autologous rib cartilage remains the best material.

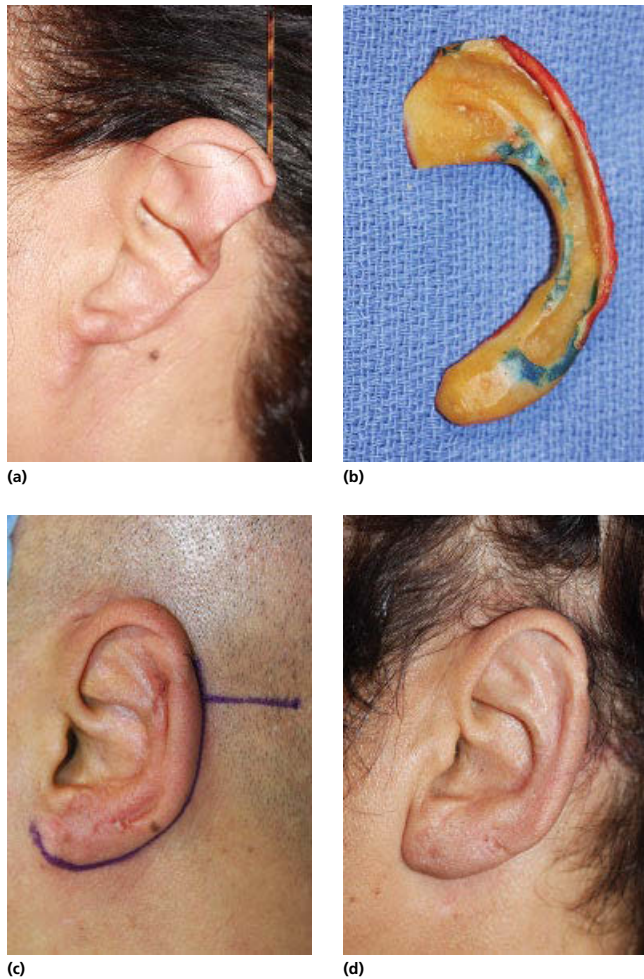


Figure 34.7 (a) The roots of the antihelix have been preserved but not the posterior wall of the concha and the inferior part of the antihelix. This case must be considered like a subtotal amputation. (b) Antihelix with its two roots is carved in one segment of cartilage and just the missing part of the helix is added on the base. This shows the typical colour of the non-ossified cartilage of a 52-year-old patient. (c) Reconstruction of the retro-auricular sulcus six months later by retro-auricular skin advancement and skin graft of the posterior surface of the ear elevated with soft tissues. (d) Result after one year.

In these cases two stages are required for ear reconstruction. The first stage involves creation of the cartilage construct with all the contours, followed by the second stage – elevation and creation of a postauricular sulcus – performed 4–6 months later.

Reconstruction technique

First stage: Reconstruction of the ear contours

The contours of the normal ear are drawn on a transparent template. Under general anaesthesia the operative field is prepared and the head is placed in a lateral position. The template is then transposed on the affected ear to correctly analyse the missing contours and to appreciate the symmetry at the end of the

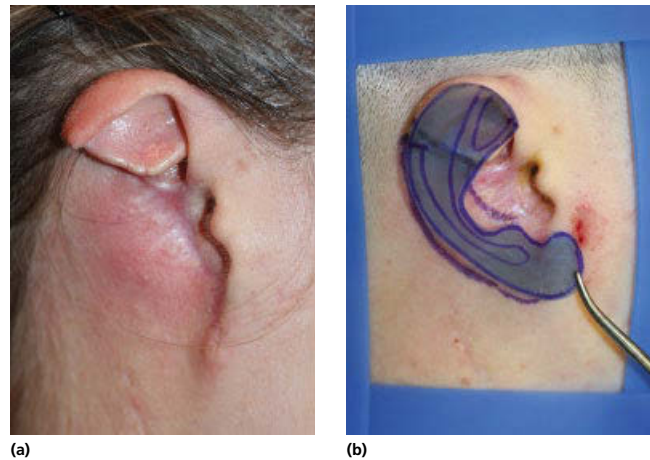


Figure 34.8 (a) Only the upper part of the helix and the tragus have been preserved. (b) After drawing a template of the other ear, the missing contours to be reproduced are drawn on another transparent template.

operation. Landmarks are taken on the normal side and then transposed on the affected side to reconstruct the ear in an ideal position and axis (Figure 34.8).

The first stage includes three steps: harvesting of the rib cartilage, carving the cartilagenous framework and preparation of the skin in the auricular area to insert the framework.³

Harvesting rib cartilage

Three or four segments of rib cartilage are harvested on the same side as the injured ear. The skin approach on the thorax is oblique and located close to the inferior border of the thoracic wall. This gives access to the anterior surface of the 6th, 7th, 8th and 9th rib segments.

A template of the other ear, having been sterilized, is used to select the adequate segments of cartilage. Once selected, these segments will be harvested, preserving the posterior perichondrium. When the framework includes the helix, the 7th or the 8th segment is chosen to obtain a piece of cartilage no shorter than 7 cm in length. The template is also used to select the two adjacent segments united by their synchondrosis that are the most appropriate for the base. Preservation of the posterior perichondrium reduces the risk of injury to the pleura. Before closing the different planes, bupivacaine (Naropin USA) is injected into the muscles to minimize postoperative pain. The muscle's edges are approximated by interrupted sutures, including the anterior and posterior edges of the aponeurosis. Care must be taken to reduce the prominent residual cartilage to avoid visible thoracic irregularities. There are no functional sequelae after harvesting these segments of cartilage.

Carving the framework

With experience, this is the most routine step of the reconstruction. A specific set of instruments (Storz, Germany) and double-ended wire sutures are used to construct the framework. Using a silicone board for stabilization is very helpful.

The posterior surface of the segments, free of perichondrium, becomes the anterior surface of the base and the segments of cartilage are cleaned, removing any perichondrial remnants. The template of the other ear is drawn on a blotting paper which is dipped into ink and applied on the cartilage. The base is placed on two adjacent ribs.

The eighth rib is prepared to reproduce the helix; it can be lengthened with an additional piece if needed. It is then carved with a gouge to increase pliability. The lobule is lowered and shaped, as well as the area of the intertragal notch and antitragus. The posterior edge of the base is cut obliquely to look thinner from behind. The base is carved and a segment reproducing the antihelix with its two crus is added to give more projection to the antihelix. The antihelix is fixed on the base using double-ended wire sutures. Once added to the base, the antihelix is reshaped. The helix is then added. Its root is fixed onto an extra piece of cartilage to give more projection and stability. The overall size and shape of the framework is checked with the template.

All the double-wire sutures used to fix the construct are tightened, cut short and buried in the cartilage. In cases of trauma there is usually no need to reconstruct the tragus.

The final touch accentuates the curve of the junction between the antihelix and the antitragus. An additional piece is sometimes added behind the base to deepen the concha and increase projection.

Framework classification

We have established a classification for the different types of cartilagenous frameworks.³

- Type I is a complete framework including the tragus and the antitragus.
- Type II includes the antitragus but not the tragus.
- Type III does not include the tragus and the antitragus.

In cases of traumatic amputation the tragus is often preserved and the most frequent frameworks used are types II and III (Figure 34.9).

Insertion of the framework under the auricular skin

A correct analysis of the skin potential in the auricular area is essential to choose the best skin coverage for the framework. When the retro-auricular skin has been preserved, it represents an ideal coverage and the retro-auricular sulcus will be reconstructed during a second stage. When it has been damaged it is necessary to use temporoparietal fascia and a skin graft. If the skin is too tense to cover the tridimensional framework it is possible to use indirect expansion.

The following scenarios represent different skin conditions.

Good skin availability in the auricular area

If the retro-auricular skin has not been damaged and has not been sutured under tension it can be used to cover the framework. The skin approach to prepare the skin pocket and to insert the framework must be through a previous scar. Dissection can be performed respecting the subdermal plexus.

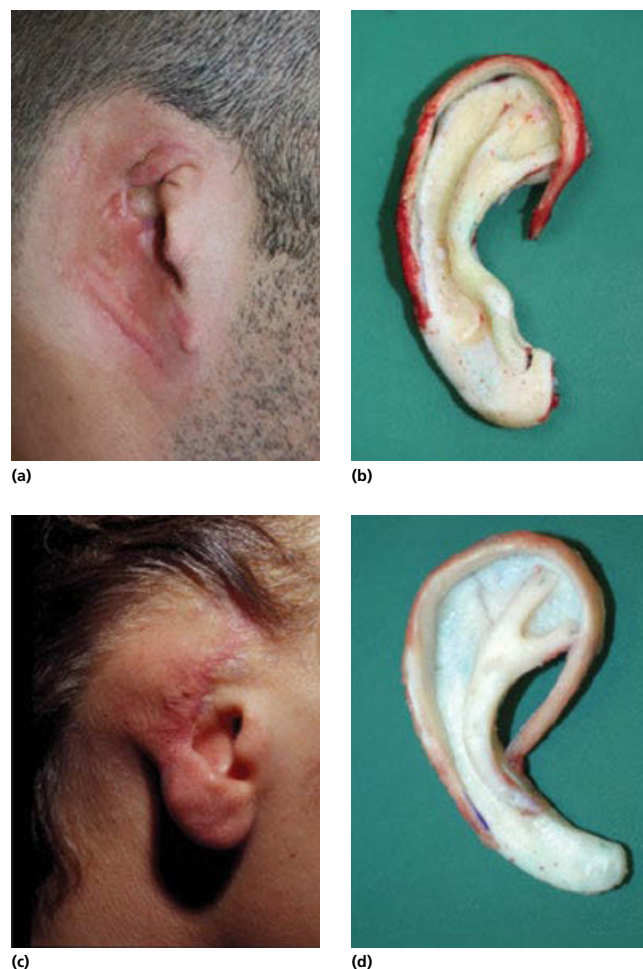


Figure 34.9 The most frequent types of cartilage in cases of total amputation: (a, b) all the contours except tragus must be reconstructed: type II cartilage; (c, d) all the contours except tragus and antitragus must be reconstructed: type III cartilage.

The case shown in Figure 34.10 was total amputation preserving the tragus on an adult patient 55 years old. A preoperative CT scan with 3D coloured printing is very useful to appreciate the degree of ossification of the cartilage at that age. Because the non-ossified segments of cartilage are larger and thicker, it is sometimes possible to carve the base of the framework and the antihelix in one piece and just add the helix, generally carved from the eighth costal cartilage segment. There is just enough skin to cover the 3D framework. Six months later, elevation of the reconstructed ear was performed taking into account the projection of the other ear. Here a temporoparietal fascia flap was used to cover the posterior surface of the framework after adding a crescent-shaped piece of cartilage behind the antihelix, stored since the first stage under the thoracic skin.

Good, scar-less skin but under tension

It is very important to appreciate the elasticity of the retro-auricular skin to decide if it can adapt to a 3D framework without too much tension, particularly on the helix. If not, an expander may

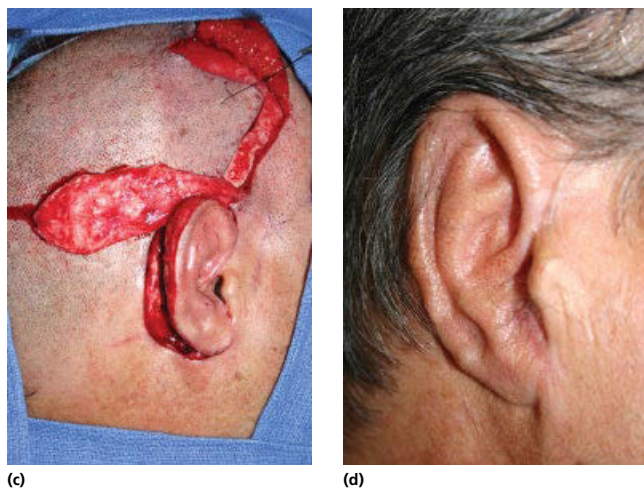
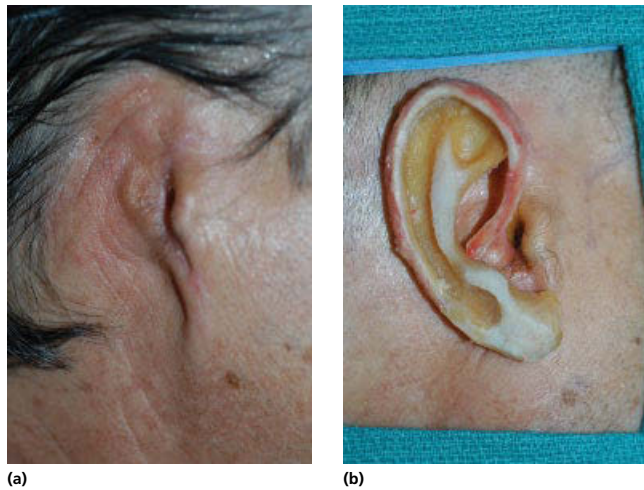


Figure 34.10 (a) Only the tragus has been preserved. (b) Framework type II, reproducing all the contours except the tragus. Only two segments of ribs (seventh and sixth) have been harvested. The antihelix has been carved in the base. (c) After the first stage all the contours are present but there is no retro-auricular sulcus. (d) Elevation of the ear is performed using a temporal fascia which covers the posterior surface of the ear and an additional cartilage graft placed behind the antihelix to give projection to the ear and deepen the concha. Some hair-bearing skin on the helix can also be excised.

be placed under the adjacent scalp prior to the formal first stage. This expansion is indirect expansion because it will not expand the non-hair-bearing skin in the auricular area but will increase the skin potential, which can be redistributed during placement of the framework, reducing the skin tension and avoiding the risk of skin necrosis and cartilage exposure.

Crescent-shaped expanders are used (150 cm^3) and filled over a period of 6–8 weeks.⁴

In the case shown in Figure 34.11 a type III framework was carved, as tragus and antitragus have been preserved. To avoid excessive tension and allow good adaptation of the skin to the contours, indirect expansion was used. Reconstruction of the retro-auricular sulcus can be performed six months later.

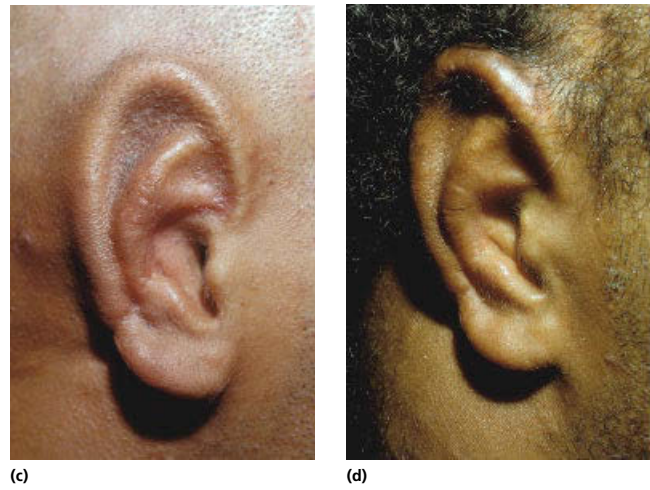


Figure 34.11 Indirect skin expansion. (a) Primary closure has been performed under some tension. (b) A crescent-shaped expander (150 cm^3) has been placed under the skin behind the non-hair-bearing skin of the auricular area, and filled. (c) The 3D framework has been inserted without excessive tension. (d) Result after elevation of the ear.

No available skin

When there are scars in the retro-auricular area that impair skin vascularity, making it impossible to cover the framework without a high risk of skin necrosis, other options are needed. A temporoparietal fascia flap based on the superficial temporal artery, if not damaged, is planned to cover the framework. This is then covered with a scalp split-skin graft. When the superficial temporal artery has been damaged it is still possible to use a random pattern fascial flap.

After shaving of the temporal area, the course of the superficial temporal artery is mapped with Doppler. A zig-zag incision is then performed on the scalp to expose the fascia. To facilitate the rotation of the fascial flap a narrow pedicle including the artery is designed and raised. Once inset, the fascial flap is covered with a scalp split-thickness skin graft, which is a superior donor site for colour match and donor site visibility.

Two or three suction Blake drains are placed during the first stage. The aims of the drains are to minimize haematoma formation and to apply the skin onto the underlying cartilage contours. The drains are removed after 3 days during the first dressing change.

Definition of the contours and the quality of the result can only be judged after a year to allow the scarring to have minimized. When elevation of the ear is indicated to create the retro-auricular sulcus, it is usually performed after one year.

Figure 34.12 shows grafting of the auricular region following a road traffic accident. A temporoparietal fascia was indicated. The temporoparietal fascia was covered with a scalp split-thickness skin graft placed on the upper two thirds of the framework. Elevation of the upper part of the ear was performed one year later.

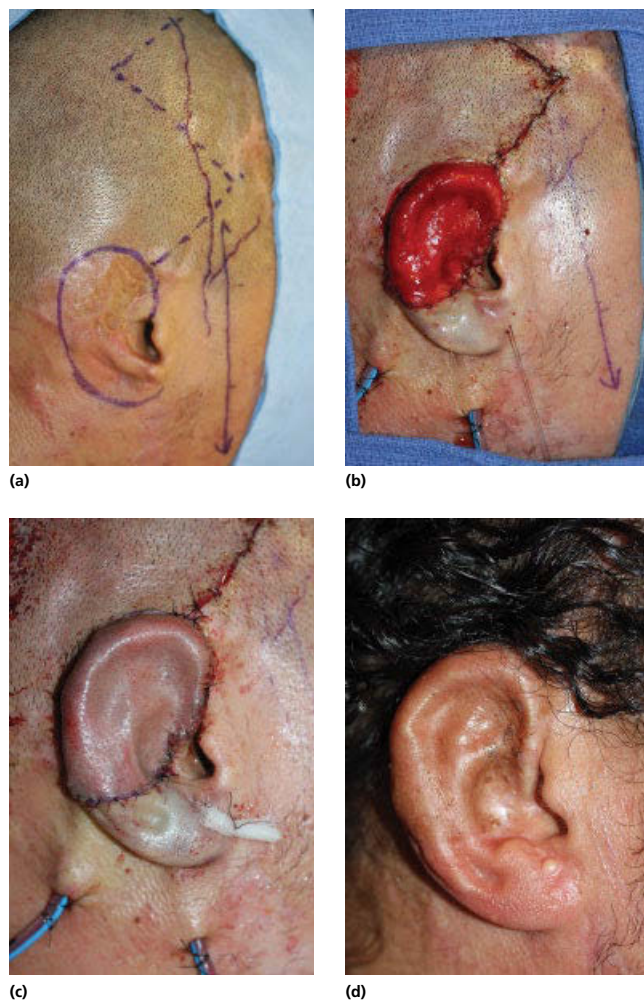


Figure 34.12 (a) The skin of the auricular area cannot be used. The route of the superficial temporal artery has been drawn using a Doppler. A zig-zag incision allows good exposure of the fascia and reduces the risk of a noticeable scar. (b) The well-vascularized fascia flap covers the framework. (c) A split-thickness skin graft taken from the scalp with a dermatome covers the fascia. Suction drainage will avoid haematoma. (d) Result after one year and before elevation.

Second stage: Reconstruction of the retro-auricular sulcus

There are different techniques to elevate the reconstructed ear.

- Elevation of the ear, preserving soft tissues on the posterior surface of the framework. A split-thickness skin graft is used to cover the posterior surface of the ear. The posterior edge of the incision around the ear is brought forward after undermining the skin. This is the technique advocated by Brent, except that he was not using a split-thickness skin graft harvested from the calvarium⁵ (see Figure 34.6c).
- Use of a temporoparietal fascia covering a piece of cartilage placed behind the antihelix to deepen the concha and give more projection to the ear. This procedure was advocated by Nagata in cases of microtia⁶ (see Figure 34.10d).
- Improving the projection of the ear after its elevation by inserting a piece of cartilage behind the framework, into a

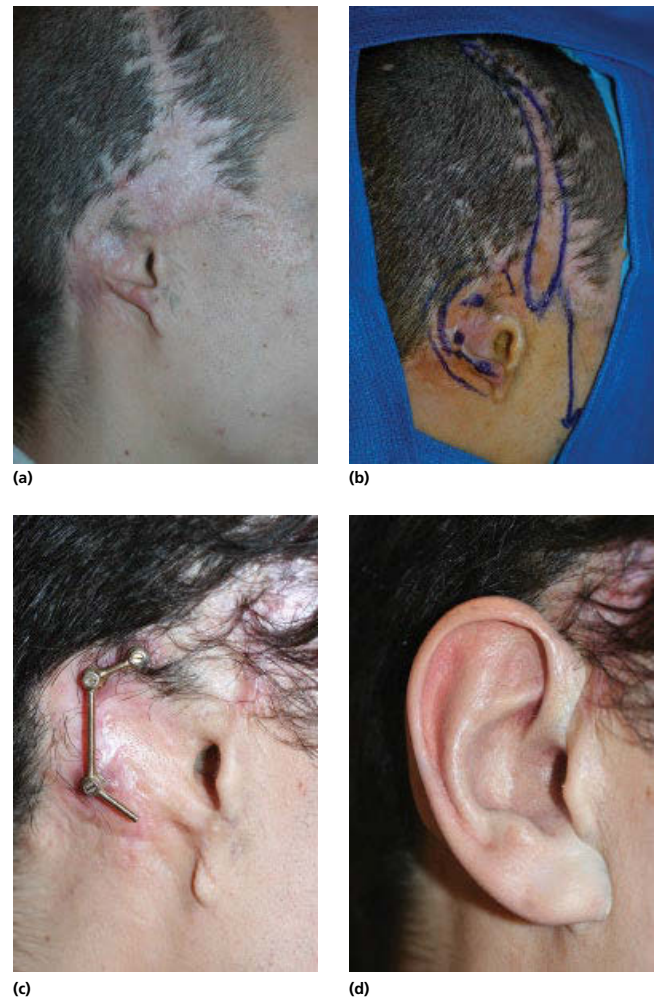


Figure 34.13 Prosthesis. (a) Temporal fascia cannot be used and there is no skin available in the auricular area. (b) Revision of the temporal scar and correct location of the implants. (c) Brenemark implants. (d) Prosthesis.

tunnel, which will be placed behind the anterior root of the antihelix or the antihelix or the antitragus.³

The criteria used to select the most appropriate procedure is the need to obtain symmetry concerning the projection of the ears, taking into account the local conditions. Analysis of the projection of the contralateral ear is essential before performing the second stage.

Prosthesis

In cases of poor local condition, including a severed superficial temporal and occipital arteries, the prosthetic solution should be considered. It may be indicated for older patients when rib cartilage may be ossified or when a patient has undergone multiple surgical procedures and opts for this solution.

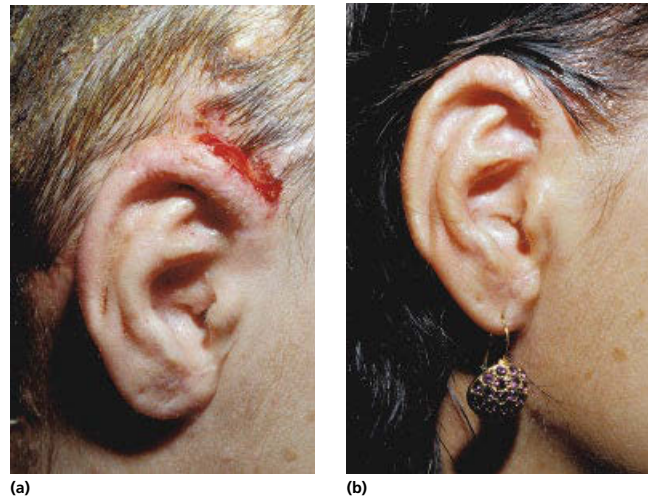


Figure 34.14 Exposure treated by spontaneous healing. (a) Exposure of the root of the helix after elevation of the reconstructed ear. (b) Granulation of the skin edges will cover the exposed cartilage, followed by epithelialization.

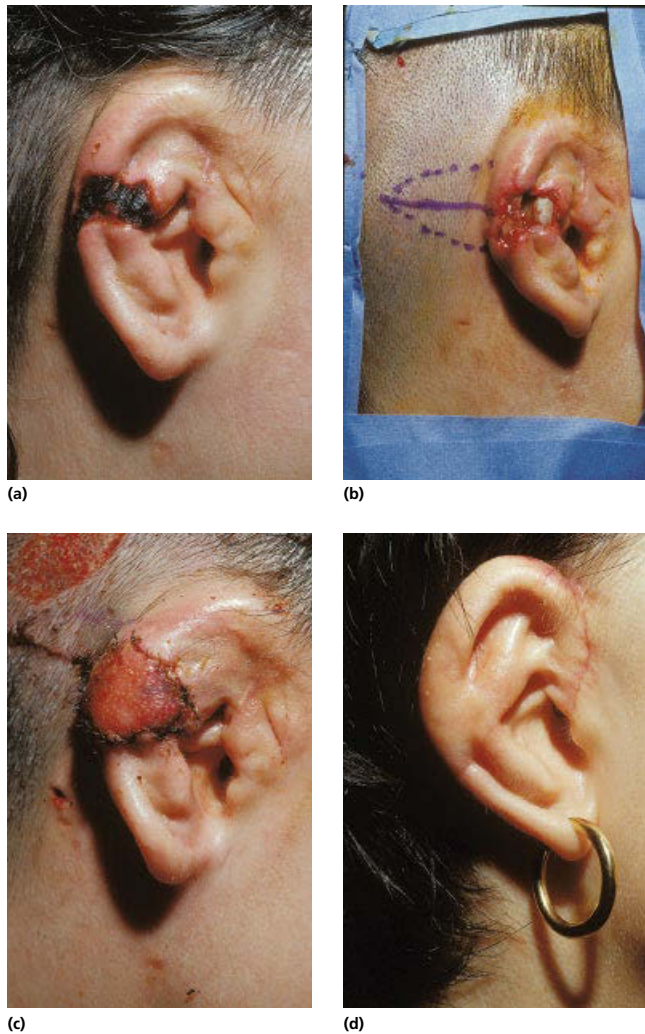


Figure 34.15 (a) Large skin necrosis at the level of the previous sutures. (b) Clean exposure of the cartilage framework and drawing of the skin approach to dissect a random fascial flap (dotted line). (c) Fascial flap covered with a split-thickness skin graft taken from the scalp. (d) Final result with good colour match of the skin graft.

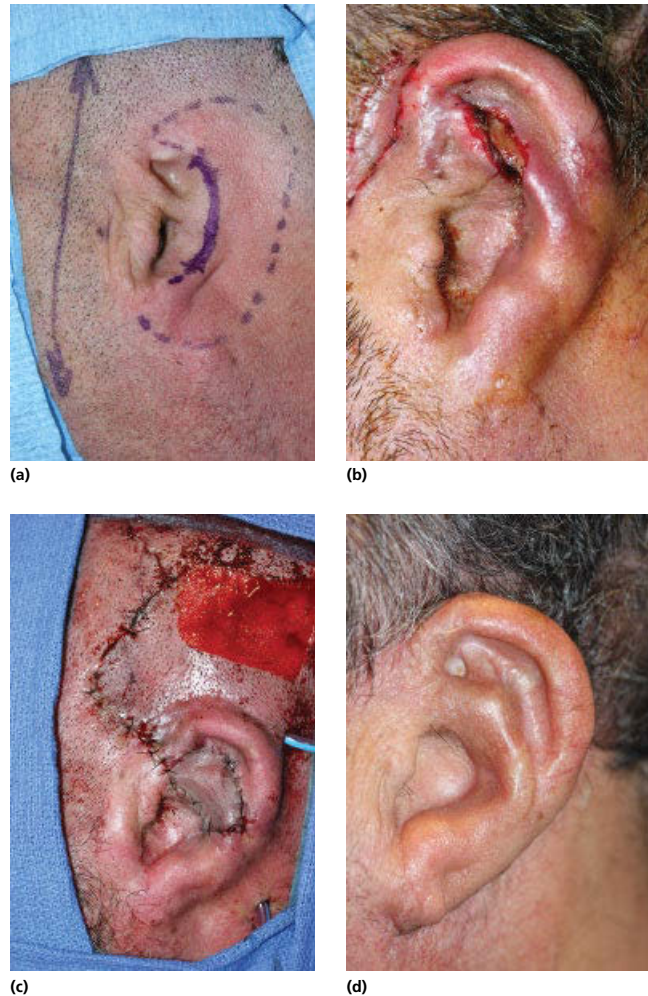


Figure 34.16 (a) Total amputation after human bite. The former scar in the concha is used as a skin approach to prepare the skin pocket. (b) Skin necrosis and exposure of the cartilage. (c) The temporal fascia flap has been placed in front of the exposed cartilage, avoiding any tension of the skin, and is covered with a split-thickness skin graft from the scalp. (d) Final result.

Placement of Brenemark implants is performed in one or two stages depending on local condition and auricular remnants. The result, of course, relies on the quality of the prosthesis and the good adaptation of the patient to the management of the prosthesis and the percutaneous abutments.

A patient with total amputation of the ear after an automobile accident who needed primary coverage of the temporal bone by a scalp flap is shown in Figure 34.13. The temporal artery and fascia have been damaged and there is no skin available in the scarred auricular area. Three Brenemark implants are placed in one stage, and a prosthesis by an experienced anaplastologist (Dr. Pillet) give the patient great satisfaction.

Complications

The use of suction drains correctly located and removed after 3 days helps to avoid haematomas. Unsatisfactory results may result from inappropriate planning in carving the framework or selecting the wrong skin approach. Extrusion of wire sutures, retraction of the sulcus and hypertrophic scarring may occur.

Infection and skin necrosis, with exposure of the cartilaginous framework, are the most serious complications.

Infection

Infection is the most severe complication as it will be responsible for cartilage resorption. It should be prevented by a very vigorous preparation of the operating field (shaving around the ear, cleaning

the skin invaginations, cleaning of the auditory canal and exposed face and scalp). Cleaning the auditory canal and prescription of antibiotic drops some days before surgery is also helpful. Antibiotics during the operation and postoperative are essential.

When diagnosed, infection must be treated by repeated drainage and appropriate antibiotic therapy. If the infection is serious enough it is usually responsible for resorption of the cartilage, which may in some cases necessitate the carving of another framework.

Skin necrosis

Secondary to a poor blood supply of the skin flaps or excess skin tension over the contours of the cartilaginous framework, skin necrosis is generally due to poor surgical planning. A small exposure less than 2 mm may close by spontaneous healing from the skin edges, which must be cleaned to induce granulation and epithelialization over the exposed cartilage. If a larger exposure needs to be covered, random fascial flaps all around the ear are available, as well as temporal fascia when possible and needed.

Figure 34.14 shows a small exposure that can be managed with correct local treatment, inducing spontaneous healing. When skin necrosis is responsible for a large exposure of the framework (Figure 34.15), a fascial flap can save the situation and must be performed once the necrosis is confirmed, which means some days after the first dressing. Antibiotics must be prescribed after a bacteriologic exam. A random flap of fascia covered with a skin graft harvested on the scalp will save the situation with a good colour match.

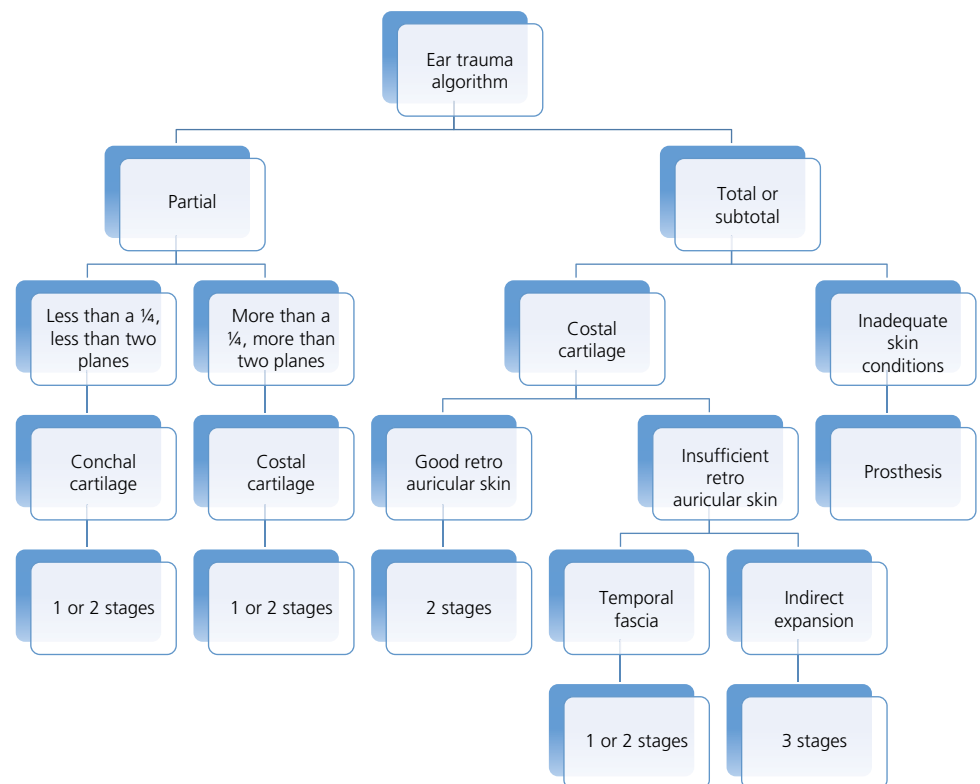


Figure 34.17 Ear trauma algorithm.

Too much tension of the skin over the framework can lead to skin necrosis on the antihelix (Figure 34.16). This situation cannot be solved by trying to approximate the skin edges with sutures. Only a short temporal fascia with axial vascularization (ATS) can solve the problem and avoid secondary infection and resorption of the cartilage.

Algorithm

It is essential when reconstructing an ear after trauma to have in mind the principles and guidelines of this surgery. It can be useful to follow a precise algorithm, such as the one shown in Figure 34.17, established by the senior author after three decades of experience in ear reconstruction, including 654 reconstructions after traumatic amputation.

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CHAPTER 35

Scalp and calvarial reconstruction

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Introduction

The position of the scalp makes it susceptible to environmental and traumatic injury. It is a common location for skin cancers, as it is normally exposed to solar radiation. The scalp is also a common target of assault with blunt or sharp weapons aimed at inflicting head injury. Hair, particularly if long and untied, is susceptible to entanglement in unguarded machinery, leading to devastating scalp avulsion. Composite defects spanning the calvarium to expose the dura and its content, such as from neurosurgical resections, generally require microsurgical free tissue transfer. The scalp, therefore, deserves special mention with regard to its reconstructive principles, underscored by a clear understanding of its anatomy and aesthetic requirements, including hair restoration. The forehead is considered separately.

Anatomy of the scalp

Borders

Classically, the scalp comprises the forehead as well as the hair-bearing scalp. The borders of the scalp, so defined, are the supraorbital ridge anteriorly, the nuchal line posteriorly and, bilaterally, a line passing through the mastoid process, zygomatic arch and zygomatic frontal process. The frontal hairline separates the forehead from the hair-bearing scalp. Surgically and aesthetically, the scalp refers to the hair-bearing scalp, which is the focus of this chapter.

Layers

These can be remembered by the mnemonic, 'SCALP': skin, connective tissue (or, alternatively, subcutaneous tissue), aponeurosis, loose areolar tissue and pericranium. The skin is thick, hair-bearing and sebaceous. The connective tissue comprises fat and fibrous septa that anchor the skin firmly to the underlying aponeurosis. Traversing the scalp, predominantly within the connective tissue layer, are sensory nerves and a network of arteries and veins. Aponeurosis refers to the galea aponeurotica

(or epicranial aponeurosis), which is a thin, tendinous sheet that unites the paired frontalis and paired occipitalis muscles, and extends bilaterally to the extrinsic auricular muscles. The galea is continuous with the temporoparietal fascia bilaterally and the superficial musculoaponeurotic system (SMAS) of the face.¹ The loose areolar tissue connects the galea to the pericranium and transmits valveless emissary veins between the superficial venous system of the scalp, the diploic veins of the cranial bones and the intracranial venous sinuses. The loose areolar tissue allows the skin, connective tissue and aponeurosis layers to move freely as one unit over the pericranium, such as under the influence of the occipitofrontalis musculature. The pericranium refers to the periosteum of the cranial bones; this is continuous with the endosteum, which lines the inner surface of the cranial bones, at the cranial sutures. The pericranium fuses with the deep temporal fascia bilaterally at its origin from the superior temporal crest.

Arterial supply

The scalp is supplied by the paired supratrochlear, supraorbital, superficial temporal, occipital and posterior auricular arteries; all terminally anastomose to form a rich vascular plexus that connects the internal and external carotid systems and bleeds profusely when injured. These arteries ultimately course predominantly within the connective tissue layer.

The supratrochlear and supraorbital arteries are terminal branches of the internal carotid artery. The supratrochlear artery exits the orbit at its medial angle by piercing the orbital septum and continues vertically over the supraorbital ridge, via its notch (in more than 97%) or foramen,² up the forehead and into the scalp. Within the forehead, it pierces the loose connective tissue and aponeurosis layers to reach the connective tissue layer. It can be located at the supraorbital ridge within 3 mm (mean 0.8 mm; standard deviation ± 0.7 mm) either side of a vertical line passing through the medial canthus; it tends to lie medial to the medial canthus in females and lateral to the medial canthus in males.³ An alternative surface landmark is the glabellar frown line on each side, which the artery passes directly

under in 50% of cases and an average of 3.2 mm lateral to in the remainder.⁴ The supraorbital artery exits the orbit lateral to the supratrochlear artery by piercing the orbital septum and passes over the supraorbital ridge, via its notch or foramen,² up the forehead and into the scalp. Within the forehead it pierces the loose areolar tissue and aponeurosis (through the frontalis muscle) layers to reach the connective tissue layer. It is located by palpating its pulse immediately superior to its notch/foramen, which is usually also palpable.

The superficial temporal, occipital and posterior auricular arteries are branches of the external carotid artery. The superficial temporal artery ascends vertically immediately anterior to the auricle enveloped by the temporoparietal fascia. It divides approximately 3–5 cm superior to the zygomatic arch into its parietal and frontal branches. The pulse of the superficial temporal artery (and its main branches) is readily palpable. The occipital artery ascends from the apex of the posterior triangle of the neck towards the vertex of the skull. Its pulse is readily palpable approximately 3–4 cm lateral to the external occipital protuberance. The posterior auricular artery ascends in the groove between the auricle and the mastoid process. It branches to form anastomoses mainly with the superficial temporal artery.

Venous drainage

The supraorbital and supratrochlear veins accompany their arteries before uniting, medial to the orbit, to form the facial vein. The superficial temporal vein accompanies its artery until it unites with the maxillary vein within the parotid to form the retromandibular vein. The occipital vein usually drains into the suboccipital venous plexus, which continues into the vertebral veins; less often it drains directly into the internal jugular vein. The posterior auricular vein unites with the retromandibular vein as it exits from the parotid to form the external jugular vein. These veins anastomose with each other and with the emissary veins.⁵

Lymphatic drainage

Knowledge of lymphatic drainage patterns is important when treating cancers of the scalp. The lymphatic system is located principally within the subdermal and subcutaneous levels, with drainage to the parotid glands, the preauricular, postauricular, upper cervical and occipital lymph nodes.

Motor nerves

The frontal (also termed temporal) branch of the facial nerve exits the parotid gland approximately 2.5 cm anterior to the tragus, ascends on the periosteum of the zygomatic arch, then continues adherent to the deep surface of, and eventually within, the temporoparietal fascia towards a point approximately 1.5 cm lateral to the orbital rim. It terminates within the ipsilateral frontalis muscle, which it innervates. Injury to the frontal/temporal branch causes ipsilateral frontalis muscle paralysis and progressive brow ptosis. Typically the orbicularis oculi continues to function due to secondary innervation from the

zygomatic branches. According to Seckel, the frontal/temporal branch of the facial nerve lies within Facial Danger Zone 2 on the immediate undersurface of the temporoparietal fascia/SMAS layer. This zone is located within a triangle bound by three lines: (a) a line drawn from a point 0.5 cm below the tragus to a point 2 cm above the lateral eyebrow; (b) a line drawn along the zygomatic arch to the lateral orbit; (c) a line dropped from the first line, to the lateral limit of the eyebrow, to the second line.⁶ Dissection, especially within this zone, should be either superficial to the temporoparietal fascia/SMAS layer or purposefully deep to it, but not immediately beneath the layer. Finally, the occipitalis and extrinsic auricular muscles are supplied by the posterior auricular branch of the facial nerve, which leaves the facial nerve before it enters the parotid.

Sensory nerves

The paired supratrochlear, supraorbital, zygomaticotemporal, auriculotemporal and lesser occipital nerves provide sensation to the scalp and can be blocked with local anaesthetic.^{7,8} The supratrochlear nerve accompanies its artery, providing overlying sensation to conjunctiva, upper eyelid, nasal sidewall, medial forehead and scalp back to the vertex. It can be blocked with local anaesthetic by field-injecting just lateral to the root of the nose at the medial portion of the orbital rim. The supraorbital nerve divides into superficial and deep branches as it exits its foramen to provide sensation, respectively, to the forehead and frontoparietal scalp. It is effectively blocked with local anaesthesia after it exits its palpable notch/foramen. Alternatively, both the supraorbital and supratrochlear nerves can be blocked with transverse infiltration at or slightly above the eyebrow. The zygomaticotemporal nerve provides sensation to the temple and can be blocked by infiltrating just inferoposterior to the zygomaticofrontal suture, which is palpable. The auriculotemporal nerve accompanies the superficial temporal artery anterior to the auricle and supplies the lateral scalp and part of the anterior surface of the auricle. It is effectively blocked with infiltration of local anaesthetic anterior to the auricle. The lesser occipital nerve provides sensation to the lateral occipital scalp and part of the posterior surface of the auricle. It is included within the superficial cervical plexus block, which is performed with field infiltration at the midpoint of the posterior border of the sternocleidomastoid muscle.⁹

Reconstructive principles

The principles of scalp reconstruction are to provide stable and durable coverage (sensate if possible) with aesthetically pleasing contour and hair growth. Although the entire hair-bearing scalp is grossly considered as one aesthetic unit, closer inspection reveals subdivisions in the pattern, density and direction of hair growth. Displacement of aesthetically important neighbouring structures (particularly the anterior hairline, sideburns, eyebrows, upper eyelids and auricles) should be avoided.

Healing by secondary intention

Not every patient with a scalp defect requires surgery. The scalp's good blood supply and high density of skin appendages, when they are intact, contribute to its dependable wound healing. Limited partial-thickness wounds, such as superficial burns or abrasions, may therefore be treated simply with cleaning and dressings. It may be necessary to shave some of the scalp in order to reduce the risk of wound colonization/infection and to improve the adherence of dressings. Deeper partial-thickness burns may still heal with dressings alone, but scar contracture and sparse hair growth may result; these issues can be addressed secondarily if necessary. Full-thickness defects generally require surgical intervention, as discussed below. However, in frail patients, limited exposure even of the outer table of the calvarium can be allowed to granulate secondarily under appropriate moist conditions, owing to its membranous bony origin.

Primary closure

Acute scalp wounds without loss of tissue can generally be debrided and closed primarily. Whenever possible, the galea should be approximated as a separate layer to reduce the wound tension endured by the overlying skin. The abundant blood supply to the scalp allows somewhat tensioned closures for small (up to 2–3 cm width) areas of tissue loss, particularly in the parietal region where scalp mobility is greater. Scoring of the galea aponeurotica perpendicular to the desired direction of skin advancement (without damaging the vasculature lying superficial to the galea) can help, but wide undermining may still be necessary to recruit tissues adequately. It should be noted, however, that if a tension-free closure is not achieved, the scar will likely widen over time and suffer scar alopecia. Even if such approaches render suboptimal aesthetics, they may still be used to achieve wound healing first before definitive aesthetic reconstruction is planned.

Skin grafting and adjunctive techniques

Skin grafts can take on any exposed layer of the scalp that is vascularized and clean. They are particularly useful for resurfacing soft tissue defects such as from resection of skin cancers, as monitoring for evidence of local recurrence over the subsequent years is made easier by the adherence of the graft to the native wound bed. A counterargument is that skin grafts are unable to restore the contour or hair of the scalp, which can be aesthetically displeasing.

Skin grafts can take on freshly burrered calvarial outer table as long as the conditions are appropriate for wound healing to progress. However, such reconstructions are fragile and tend to undergo repetitive wound breakdowns and regranulations; they are also aesthetically poor. Skin grafting directly onto calvarial bone should therefore be reserved for patients who are unsuitable for, or refuse, anything more complex. Prior to application of the skin graft, the calvarial bone should be seen to produce pinpoint bleeding points. Alternatively, the outer table can be

removed to reveal the well-vascularized diploic space,¹⁰ or a pericranial flap designed to cover the bone before grafting; however, these options are more morbid and still provide poor aesthetics, thus inviting other options to be considered instead.

Negative pressure dressings can be applied to improve graft take or encourage granulation,¹¹ and dermal substitutes can be useful to improve the quality of skin graft reconstructions.¹⁰ It is also possible to transplant hair to skin-grafted scalp that has appropriate underlying soft tissues. Ultimately, however, a successful hair-bearing reconstruction would be an aesthetic improvement on most skin-grafting techniques. Like a tensioned primary closure, skin grafting can sometimes be useful to achieve wound healing, and a definitive aesthetic reconstruction can be performed at a later stage, if necessary, once the wound is quiescent.

Local flaps

Local hair-bearing flaps are useful when recruitment and advancement of neighbouring tissue cannot achieve a tension-free closure. Although a plethora of locoregional flap techniques have been described for use on the scalp, the most dependable options are variations of rotation and transposition scalp flaps. Single transposition flaps generally require a skin graft for the secondary defect, so rotation scalp flaps should be preferred when available. If the donor site of a rotation flap is predicted to require grafting, consideration should instead be given to tissue expansion first if a multistaged reconstruction is feasible and acceptable.

Pinwheel flaps are useful for small circular defects. They incorporate a series of random pattern rhomboid flaps bordering the defect, each designed to reconstruct one sector of the defect and achieve donor site primary closure. They are an alternative that avoids the long curvilinear incisions required for scalp rotation flaps and are able to replace hair-bearing scalp with like tissue.

A recipient defect in an advantageous location on the scalp (i.e. placed laterally rather than centrally) of up to a maximum of approximately 5–6 cm diameter can be closed with a primarily closed large scalp rotation flap that is both scored then back-cut. These need to be designed with an axial pattern blood supply, incorporating at least one of the named arteries of the scalp. Ideally, the flap should be at least 5 times wider than the diameter of the defect to allow wound closure with acceptable tension; hence even a 5 cm circular defect can stretch the limits of a scalp rotation flap with a primarily closed donor site. Meticulous reverse planning with careful attention to pivot point, blood supply, sensory supply and defect/flap dimensions are critical. The long curvilinear incision is performed with a scalpel, employing electrocautery only sparingly for specific bleeding points to reduce damage to hair follicles; the subgaleal plane is used to raise the flap widely. If skin grafting is to be employed, the donor site should preferably be left with undamaged pericranium and located inconspicuously or so that it can be camouflaged with hairstyling.

Although the pericranial, pericranial–galeal and temporalis flaps can be useful for small defects with exposed dura or exposed dural repair, in the setting of scalp reconstruction, such problems may be better addressed instead with free tissue transfer. It should also be noted that Orticochea's oft-referred-to descriptions of three-flap or four-flap scalp reconstructions are of historical interest only. They were useful for defects over the occipital scalp and involved extensive scoring of the galea to maximize flap advancement. They brought high risks of postoperative alopecia, unnatural direction of hair growth, wound-healing problems and the requirement for skin grafting, but were an ingenious solution to difficult scalp defects prior to the wider availability of tissue expansion and free tissue transfer. Likewise, free tissue transfer has replaced many of the regional flap options, such as the pedicled latissimus dorsi and splenius capitis muscle/myocutaneous flaps, in the remit of scalp reconstruction. The trapezius myocutaneous flap, however, is a reasonable option for moderately sized composite occipital scalp ablative defects for which tissue expansion would be contraindicated by the presence of tumour.

Tissue expansion

Immediate expansion can be achieved intraoperatively with traction applied to the skin edge. The biological principles and application of tissue expansion is covered in Chapter 6 in this book. Briefly, removal of the traction causes the skin to relax to its original dimensions. If the applied load is excessive, however, the skin stretches without subsequent relaxation. This can be exploited when advancing local tissues as long as tissue perfusion is maintained under the applied tension; otherwise the sutured leading edge will become ischaemic, necrose and the wound will dehisce. Tissue creep instead occurs over time under constant loading, such as from placement and expansion of a

prosthetic tissue expander. Mechanical creep involves the stretching and alignment of collagen fibres, microfragmentation of elastin, and the displacement of interstitial fluid, whereas biological creep involves the generation of new tissue. With progressive tissue expansion, the epidermis thickens due to cellular hyperplasia, the dermis thins, appendages spread, and fat and muscle layers thin. These properties can also be exploited for serial excision of moderate-sized benign lesions of the skin.

Tissue expansion of the scalp allows the replacement of like with like tissue that is both hair-bearing and potentially sensate (Figure 35.1). The intention is generally to expand a scalp rotation-advancement flap in order to allow defect coverage as well as donor site primary closure. Judicious planning, especially the placement of expander pockets, ports and incisions, is key to reducing morbidity. General guidelines for tissue expansion in the head and neck region include the following:

- Choose a tissue expander with a length and breadth that is at least as large as the defect.
- Design the incision perpendicular to the direction of intended expansion, make it as small as possible, and locate it away from the defect, the pocket and the future flap incisions.
- Place the valve in a healthy area above and/or lateral to the expander, and at least 7 cm away.
- Fill the expander at the conclusion of the operation in order to reduce seroma/haematoma and the need for drains.
- Wait two weeks for healing before beginning expansion, and continue weekly thereafter.
- The capsule can be incised to increase stretch, but avoid a full capsulectomy.
- Elasticity and contractility of the expanded flap depend on individual factors, but an estimation of gained flap length is the distance over the dome of the expander minus the corresponding measurement of its base.



Figure 35.1 (a) Alopecia in the occipital region treated with a tissue expander. (b) Postoperative appearance at two months. Source: Dr Shiow-Shuh Chuang. Reproduced with permission.

- With this measurement in mind, overexpand by 30–50% to account for flap contraction, as the theoretical flap gain is rarely, if ever, yielded *in vivo*.¹²

Multiple tissue expanders can be employed when more than one expanded flap is necessary or when a large flap requires expansion in different areas. Although the direction of hair growth on a rotation-advancement scalp flap is unlikely to match perfectly the neighbouring uninvolved scalp, every effort should be made to inset the flap in such a way as to minimize this disparity.

Microsurgical free tissue transfer

Free flaps are useful when there is an absence of healthy local tissues (for example, due to irradiation, extensive scarring from multiple previous reconstructions, neighbouring trauma), and when the surface area of the defect is too large for local flap reconstruction or is composite (spanning the calvarium or more deeply). Microsurgical free tissue transfer can offer a single-stage solution for extensive scalp defects, which is particularly important in the ablative oncologic setting. Free flaps also import fresh, vascularized and healthy tissues from a distant site, which can be the critical factor that allows wound healing in irradiated or previously infected fields. The principle drawbacks of free tissue transfer to the scalp are that these flaps cannot bring the same density of hair and they are generally thicker. The former can be addressed at a later stage with hair transplantation if necessary, and the latter either by primarily or secondarily thinning a cutaneous/fasciocutaneous flap or by importing a muscle flap and sheet skin grafting its surface. The muscle will atrophy and conform over time, producing aesthetic outcomes that can be surprisingly acceptable, particularly if the patient had preexisting alopecia. If, however, the patient were planned for postoperative irradiation, a fasciocutaneous free flap would provide more robust healing of the skin. Sometimes the ablative composite defect spares the hair-bearing skin of the native scalp; in such circumstances, an adipofascial free flap with or without muscle can be de-epithelialized and buried to obliterate the deadspace and restore contour. A small monitoring paddle can be incorporated into the design and later removed under local anaesthetic once its purpose has been fulfilled.

Many free flap donor sites have been described for scalp/calvarial resurfacing, including the latissimus dorsi, rectus abdominis and gracilis muscles or omentum with skin graft, and the cutaneous/fasciocutaneous anterolateral thigh, parascapular/scapular and radial forearm flaps. When the defect is composite, involving the calvarium and/or dura and/or brain, the free tissue transfer can be tailored to incorporate fascia and muscle as required. The anterolateral thigh is probably the most versatile donor site for composite ablative defects (Figure 35.2), allowing vascularized fascia lata to be brought with the flap as a replacement for the dura, voluminous vastus lateralis muscle for deadspace obliteration in cases of neurosurgical ablations, and a long pedicle that is sizeable and dependable. It is also located far from the scalp, which allows a two-team approach during ablative surgery or recipient site preparation.

However, if the requirement is for subtotal or total cranial resurfacing, the latissimus dorsi muscle with skin grafting provides the greatest area for coverage while allowing primary closure of the donor site; the omentum is an alternative large resurfacing flap but requires the abdominal cavity to be breached. An extensive fasciocutaneous free flap would be possible but a less desirable option since donor site skin grafting would be necessary if the subtotal scalp was to be resurfaced. The nearby superficial temporal vessels on either side are dependable recipient vessels for free tissue transfer. Free flaps with long pedicles are necessary to reach alternative recipient vessels in the neck. Eccentric design of the vascular hilum of the flap can effectively lengthen the pedicle but vein grafts may still be necessary when reconstructing defects near the vertex.

Replantation

Scalp avulsion classically occurs from entanglement of loose hair in machinery, but animal bites and 'scalping' assault injuries are other causes. It is important that the patient is aggressively resuscitated with fluid and blood products as necessary and pressure applied to bleeding points as scalp injuries can bleed catastrophically. No reconstructive alternative can provide superior results to scalp replantation and thus it should be attempted whenever possible (Figure 35.3). Mechanical scalp avulsions predictably cleave along the path of least resistance, which is the loose areolar tissue plane between the galea aponeurotica and the pericranium. This means that the axial vascular network supplying the amputated scalp (residing superficial to the galea) may well be intact and allow replantation. Indeed, extensive collateralization of vessels means the entire scalp can be revascularized on one set of anastomoses.

The few contraindications to an attempt at replantation include devastating multitrauma precluding attempted microsurgical salvage and a severely crushed and/or macerated amputate with multiple-level segmental injuries. Even if the scalp is avulsed in more than one part, each sizeable scalp island should be attempted for replantation unless clear contraindications exist.

Preoperative shaving of the entire scalp and amputate leaving approximately 1 mm of hair length allows better appreciation of lacerations/contusions and correct alignment of wound edges. Accurate identification and resection of traumatized vasculature and replacement with vein grafts as necessary is critical to success. Usually the superficial temporal vessels are the chosen recipient vessels, often proximally within the parotid owing to the avulsive segmental vascular injury, however the supraorbital, occipital and postauricular vessels have also been used. The arterial inflow should be restored first both to reduce ischaemia time and to declare at least two high-outflow veins for anastomoses; other veins should be ligated to reduce blood loss. The scalp should then be carefully inspected for regions of threshold perfusion once vascularity is restored as additional anastomoses may be necessary. The use of *hirudo medicinalis* with antibiotic coverage, or alternative chemical/mechanical leeching methods,¹³ should be considered if venous congestion



Figure 35.2 (a) Scalp defect after skin cancer excision. (b, c) Postoperative appearance after anterolateral thigh flap reconstruction.

persists despite multiple flowing venous anastomoses. Failed replantation can be salvaged by negative pressure dressing assisted wound closure,¹¹ skin grafting or microsurgical free tissue transfer, but each of these options provides comparatively poorer aesthetics.

Reducing the risk of scar alopecia

If at all possible, the potential for hair regrowth should be preserved at the index operation. Employing simple measures such as avoiding wound tension and not using electrocautery or crushing instruments at the cut edges of the scalp help reduce the risk of follicular damage and scar alopecia. When performing the incision, some authors report aligning the scalpel parallel with the follicle, whereas others prefer to transect the follicle obliquely. The trichophytic method used by hair transplantation surgeons for strip harvest donor sites involves a combination of these concepts. First, the surgeon takes careful note of the direction of hair growth on either side of a planned incision. In any location on the scalp, there will be one side (Side A) of the incision where the hair grows toward the incision so that the hair shaft overlies it, whereas on the other side (Side B) the hair correspondingly

grows away from the incision and the shaft does not overlie it. Second, the incision is placed with a scalpel blade aligned with the direction of hair growth. As the incision is deepened, it should follow the curvature of the hair shafts so that the follicular components underlying the dermis and bordering the incision are not cut. Third, a trichotomy is performed as follows: the leading edge of Side A is de-epithelialized by 1 mm, resulting in the subepithelial transection of hair shafts bordering the incision on Side A. This leaves the dermal/subdermal components of the hair follicles intact, preserving their ability to continue producing hair. Classically, a trichotomy is not performed on Side B, but some authors believe that doing so further improves scar concealment.¹⁴ Fourth, the incision is closed with fine sutures so that the epithelial edges accurately appose. Accordingly, the hair shafts transected during the third step are directed to grow through the incision and thus within the scar, disguising its presence. Proponents of the technique suggest that the suture used for wound closure, whether single or double layered,¹⁴ should not bite deeper than approximately 1 mm because the bulge of the hair follicle extends between about 1.0 mm and 1.8 mm below the skin surface.¹⁵ These principles can be applied



Figure 35.3 (a) Avulsed scalp. (b) Appearance three months after microsurgical replantation. (c, d) Appearance one year after replantation.

to any incisions and wound closures on the scalp to reduce the risk of distressingly conspicuous scar alopecia.

Hair restoration

Alterations in hairstyling, psychological adjustment and acceptance, preference for wearing hats, or tolerance of wigs, toupées and other camouflages for alopecia, can mean that autologous hair restoration is not always necessary or requested. For those patients who would prefer hair restoration to proceed, the principal techniques used are surgical: tissue expansion, scalp reduction and hair transplantation. For aesthetic restoration purposes, however, tissue expansion and scalp reduction have largely been superseded by follicular unit transplantation. The two medical treatments that are approved by the US Food and Drug Administration for treating alopecia (minoxidil and finasteride) are indicated for generic rather than site-specific alopecia, such as due to male pattern androgenic alopecia.

Hair restoration can dramatically improve aesthetic and psychosocial outcomes for the patient following surgery or other

trauma that has left an alopecic patch, disrupted hairline, scar alopecia or a poorly designed hair-bearing flap inset. Previous punch-graft techniques, which produced unnatural 'plugged' or 'doll-like' appearances, have largely been replaced by highly sophisticated methods of follicular unit harvest, selection, preparation and transplantation into considered patterns and orientations of hair. An in-depth appreciation of the finer points of technique required to produce pleasing outcomes are important; these will only be covered broadly. The reader is directed to a selection of comprehensive review articles covering the meticulous approach necessary at every step for successful follicular unit transplantation.^{16–19} As stated by Rousso and Presti, 'the technique is only as effective as the technician, and results are heavily dependent on the forethought of the architect'.¹⁹ Accordingly, subspecialty training is important, both for the surgeons and their teams, before embarking on this technique.

Broadly, the goal of hair transplantation is to mimic the patient's natural hair growth pattern in an area of alopecia so

that it aligns and blends with its surrounds. This requires a careful analysis of the undisrupted hair, including its colour, density and direction of growth, bordering the area of concern so that its features can be mimicked as closely as possible. The patient's expectations must be appropriately managed and an evaluation of potential donor sites performed and explained to the patient. It should be emphasized to the patient that the donor hair density and calibre are critical determinants of the fullness of the transplanted recipient site. Standardized photography with a light blue background is important for documentation;¹⁸ video is also used to demonstrate the dynamic movement of native and transplanted hair, such as when being combed through.

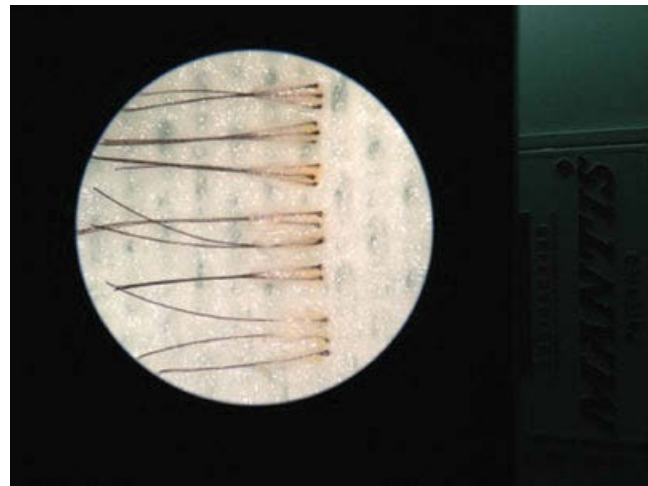
The donor site for follicular unit transplantation is usually either an occipital strip followed by microdissection of follicular units, or multiple follicular unit extractions performed manually or, more recently, robotically (Figure 35.4).²⁰ The occipital strip should be located horizontally in a region between the

occipital protuberance and 1 cm above the top of the auricles, and trichophytic closure used. Its size is determined by the follicular density in the region, as calculated by hair densitometry, and the predicted number of follicular unit transplantations to be performed. It is critical that follicular units are kept viable once harvested; chilled isotonic saline is one medium that is often used. Single follicular units are microdissected from the occipital strip by skilled technicians and arranged according to how many follicles the unit contains (one to four) so that the surgeon can inset them in an organized and expedient fashion.

The surgeon designs and plans the placement of the follicular units according to the aesthetic requirements of the recipient site. If a hairline is to be restored, this is perhaps the single most important determinant of the final aesthetic outcome. It must be located accurately – broken up by irregularly irregular gaps and triangles rather than linear – and transition naturally and progressively from the non-hair-bearing skin to reach the



(a)



(b)



(c)

Figure 35.4 (a) A 45-year-old male with frontal alopecia. (b) Follicular hair unit preparation. (c) Appearance one year after 2049 follicular hair transplantation. Source: Dr Yau-Li Huang. Reproduced with permission.

density of the native scalp.¹⁸ Each part of the scalp, such as the anterior hairline, crown and temporal regions, has its own follicular aesthetics that need to be restored for a good outcome. Generally, hypodermic needles, mindi knives or micro-punches are used to create recipient ports for the transplants. These need to be placed with the intended direction of hair growth in mind as well as the correct depth, so that the follicular unit skin sits flush with the recipient site. An evaluation of the transplant result can be performed at approximately eight months when the follicular units have appropriately cycled through their expected telogenic and anogenic phases.

Future considerations

The current array of autologous reconstructive techniques can provide wound closure and healing for the vast majority of scalp defects. However, the outcomes of extensive reconstructions, such as following failed total scalp replantations or full-thickness subtotal scalp burns, are generally not aesthetically pleasing.

Although hair transplantation can improve regions of alopecia, the lack of donor material in total scalp reconstruction is currently an unsolved problem. Efforts continue in basic research and biotechnology company laboratories to find methods of cloning hair follicles for transplantation to be possible with minimal donor material, but results so far have been disappointing.¹⁸

Total facial vascularized composite allotransplantations (VCAs) with scalp extensions have been performed with hair regrowth, facial functional return and excellent aesthetic results.²¹ Cadaveric work allows opportunities for planning total facial VCAs that incorporate total scalp and avoid facial cutaneous scars.²² Additionally, clinical cadaveric scalp VCA, allotransplantation of muscle in an already immunosuppressed patient, and scalp flap transplantation between identical twins have all been reported for scalp reconstruction.^{23–25} For the moment, however, scalp VCA is only justified when performed as an extension of a facial VCA, given the inherent lifelong risks of immunosuppression.

Conclusions

The scalp commands an important influence on the overall aesthetics of the face and skull and considerable psychosocial morbidity can result from suboptimal scalp reconstructions and/or alopecia. The presented techniques offer acceptable reconstructions for the vast majority of scalp defects and non-hair-bearing reconstructions can be improved with follicular unit transplantations or well-designed pre-expanded scalp flaps. Wound healing can be achieved for total scalp defects, but hair restoration remains a problem when there is no donor material present.

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CHAPTER 36

Facial trauma

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Frontal bone and naso-orbital fractures

The frontal bone and naso-orbital region constitute the upper third of the face. Trauma to this region of the face is often accompanied by brain injury and damage to the cranial nerves. The impacting forces may be transmitted to the base of the skull, producing a range of effects from transient loss of consciousness to loss of life. The injuring force may impact directly on the frontal bone, causing blunt or penetrating brain injury. With this in mind, it becomes obvious that the treating surgeon should be part of a team capable of assessing and treating such injuries.¹ The injuries therefore fall into three categories: injuries to the bone without cerebral involvement, externally compound fractures or penetrating wounds with underlying dural and cerebral injury, and internally compound fractures often of the cribriform plate region resulting in a cranionasal fistula with or without cerebrospinal fluid (CSF) leak.

Some of the causes of craniofacial trauma include falls, assaults, sporting injuries, industrial accidents and missile injuries. The epidemiology varies with the community so when organizing multidisciplinary care and planning preventative strategies, an understanding of the causes and incidences in a particular community is essential.²

Anatomy

The anterior cranial fossa is formed in the front and at the sides by the *frontal bones*, and they are attached to the facial skeleton at the front nasal, frontomaxillary and frontozygomatic sutures (Figure 36.1). The frontal bone also articulates with the ethmoid bone in the anterior cranial fossa, which bone forms part of the orbit and its vertical plate forms part of the nasal septum and indeed is said to play a part in facial development. Laterally, the anterior fossa forms the thin superior orbital plates; they dip down medially to articulate with the cribriform plate of the ethmoid, the posterior articulation of which is the body of the sphenoid bone. The lesser wings of the sphenoid bone form the

crescentic posterior borders of the anterior fossa. The optic canal is formed by the two roots of the lesser wing of the sphenoid and runs forward and laterally in the superolateral wall of the sphenoid sinus to the orbital apex.

The complex sphenoid bone is the keystone of the craniofacial skeleton; it forms part of the anterior cranial fossa, the middle cranial fossa, the lateral orbital wall, where its articulation with the zygoma is an important landmark in the reduction of orbitozygomatic fractures, and the infratemporal fossa.

The rest of the facial skeleton is composed of the lacrimal bones, the nasal bones, the ethmoids, the zygomas, the maxillae and the mandible. It is customary to describe the face as being divided into three thirds. The upper third is formed mostly by the frontal bone, which forms the orbital rooves. The middle third contains the maxillae, zygomas and the nasoseptal complex with a contribution from the lacrimal bone and the ethmoid. These elements articulate laterally with the zygomatic processes of the temporal bones, and serve as a good measure of the anterior projection of the face. Posteriorly they articulate with the sphenoid to form the orbit and centrally and laterally with the frontal bone. The lower third is said to be formed by the mandible, however this is somewhat of a convention as the body only is in the lower third, the ramus in the middle third and the temporomandibular joint has a major component formed by the base of skull.

The midfacial skeleton is designed to transmit the powerful forces of mastication to the base of skull. The horizontal elements in the maxilla, the alveolus and hard palate support three paired vertical buttresses, at the pyriform margins at the zygomatic maxillary junction and more posteriorly at the pterygo maxillary junction. The two pairs of anterior buttresses provide the best places for internal fixation with plates and screws.

The *orbital cavity* is formed of seven bones in the shape of a four-sided pyramid. There is a relatively strong rim anteriorly, four more flimsy walls and a thick apex. The zygoma contributes to the lateral and inferior orbital margins, the lateral orbital wall and the orbital floor. The maxillary component of the orbital floor medial to the zygoma is thin and delicate (lamina papyracea).

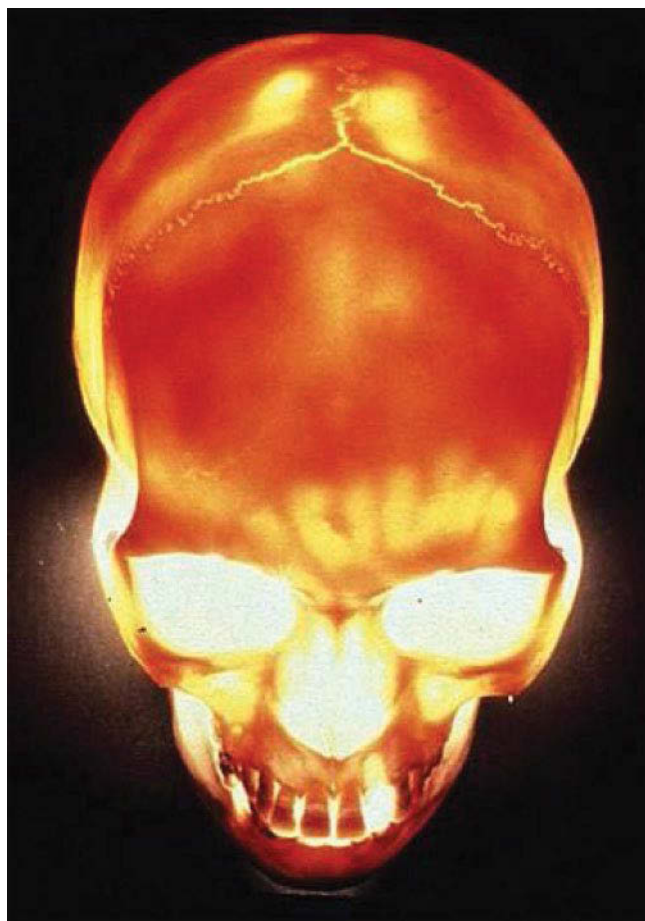


Figure 36.1 A transilluminated adult skull demonstrates the paranasal sinuses and the supporting buttresses of the face.

The anterior orbital floor medial to the infraorbital canal is concave downwards, but posterior to the globe it becomes convex upward. A point to be noted for successful restoration of the orbital floor after injury: the medial wall is composed of the lacrimal bone anteriorly and the thin lateral plate of the ethmoid posteriorly.

The *paranasal air sinuses* are rudimentary at birth and do not reach adult proportions until puberty. The degree of pneumatization varies, particularly that of the *frontal sinus*. They range from being barely pneumatized to extending superiorly in the frontal bone for a variable distance; laterally they can go as far as the lateral margins of the orbit and also extend into the orbital roof.

The frontonasal ducts open inferomedially and drain into the frontal recess of the nasal cavity.

The *ethmoid sinuses* constitute an interconnected series of thin-walled cells that lie between the upper lateral nasal cavity and the orbit and are separated from the orbit by the thin lateral wall. Their roof forms part of the anterior fossa lateral to the cribriform plate. The ethmoid sinuses are related to the maxillary sinuses inferiorly, the frontal sinuses superiorly, and the sphenoid sinuses posteriorly.

The *sphenoid sinuses* are within the body of the sphenoid and may sometimes spread into the lesser wing of the bone; they are divided by a septum and may be asymmetrical. They lie under the central part of the floor of the anterior fossa and are closely related to the internal carotid artery, the pituitary gland and the optic nerve. They connect through their anterior wall with the upper nasal cavity.

The *maxillary sinuses* usually fill the entire body of the maxilla and may extend into the zygoma and below the floor of the nose; their walls are fragile and easily broken. They also are rudimentary at birth.

Management principles

Management involves investigation and treatment and should be based on a thorough knowledge of the *pathophysiology*.

With respect to the *cranial cavity* the dura is firmly attached around the cribriform plate through which the filaments of the olfactory nerve pass sheathed by arachnoid. A dural arachnoid tear with a fracture communicating with the nose or paranasal sinuses will lead to CSF leak.

The frontal lobes of the *brain* are particularly vulnerable to the effects of frontal impact, and by anterior fossa fractures when the orbital rooves are shattered or buckled. Bilateral frontal lobe injury can result in long-term neuropsychological problems.

The *olfactory nerve* filaments may be damaged by fractures crossing the cribriform plates resulting in anosmia as well as CFS leak.

The *optic nerve* may be compressed by a displaced fracture through the optic canal, by shearing forces that devascularize the nerve, by contusion or haematoma within the canal without a fracture, or by direct injury to the orbit.

The *orbit* itself may be fractured directly or indirectly as part of frontal, nasoethmoid and midface or zygomatic fractures.

In addition to the pathophysiology, it is essential to have knowledge of the effects resulting from the various *mechanisms of injury*.

Frontal impact is the most common mechanism of craniofacial injury; it may result in linear fractures or depressed fractures, and the forces may be transmitted to the cranial base and cause fracturing there.

Fractures of the skull base are rarely significant in themselves, however the force required producing such a fracture is considerable and is often associated with injuries of the soft tissues, brain, cranial nerves, major vessels, etc.

The pattern of fracture lines resulting from blunt trauma follows the points of weakness and in the frontal bone is influenced by the extent of the frontal sinuses and the particular honeycomb structure of the particular sinus (Figure 36.2). The age of the patient is a significant influence, younger bones being more pliant and less brittle. Fracture patterns, however, rarely conform to a predictable formula.

Penetrating and missile wounds have no particular pattern. The tissue damage resulting from low-velocity penetration is confined to the pathway of the penetrating agent.^{3,4}

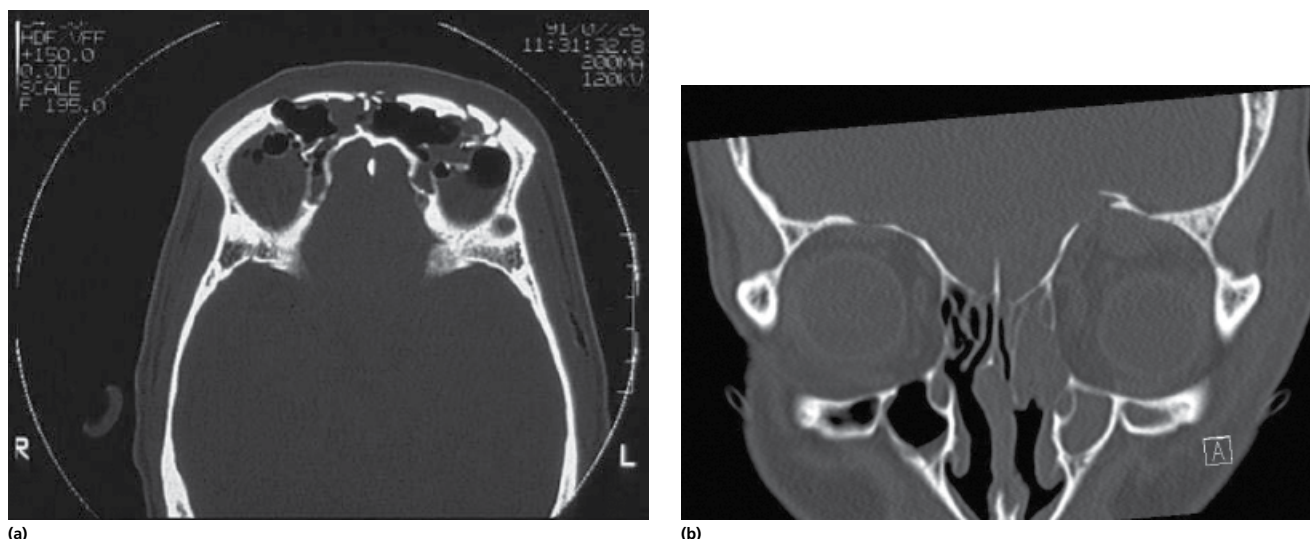


Figure 36.2 (a) CT scan showing a depressed comminuted fracture of the frontal sinus affecting both inner and outer tables resulting from blunt trauma. (b) A fracture of the orbital roof resulting from a billiard cue entering the orbit, sparing the globe, and penetrating the orbital part of the frontal bone and brain.

Gunshot wounds may be penetrating, perforating or ablative depending on the velocity and nature of the projectile, the higher the velocity, the greater the damage. High-velocity missiles cause massive avulsion with loss of bone and soft tissue, which is often more extensive than immediately apparent because tissues may be devitalised or injured by the traction of avulsion. The eyes and brain may be injured directly or by vascular injury, which can be delayed.⁵

Crushing injuries may result in severe fracturing of the frontal bone and skull base; however in infants and children the pliability of the bones means that the pattern of fracture is different with greenstick fractures.⁶

Initial management

Remember to save the patient! Fractures of the frontal bone and nasoethmoid complex can occur in isolation but more commonly are associated with other facial and systemic trauma. Severe craniofacial injuries often pose an immediate threat to life. Airway obstruction, displaced dentures and collapse of the mandibular arch with retro displacement of the tongue may cause severe hypoxia. Particular care should be taken with cases of frontofacial fractures, where the possibility of a breach in the anterior cranial fossa exists, not to forcibly insufflate the patient or to pass tubes via the nose.

Emergency assessment and resuscitation

The primary survey, setting the priorities for management, follows resuscitation, secondary survey and stabilization, and in the craniofacial region these are: airway, breathing and circulation.

Airway

The following is a list of problems that may be encountered when trying to secure the airway in severe frontofacial fractures. Subsequent chapters deal with fracturing of other regions of the face, and these same problems potentially occur.

- Distortion of the bony facial skeleton
- Swelling of the lips, tongue and floor of mouth
- Broken teeth and dentures
- Multiple fractures of the mandible with loss of tongue support
- Haemorrhage into the soft tissues of the neck hindering cannulation of the airway
- Disruption of the anterior cranial fossa
- Trismus
- Blood and vomit in the airway
- Fracture of the larynx.

Breathing

Associated chest injuries must be excluded. A brain injury may cause central problems with breathing as can the overzealous or inappropriate use of pain relief.

It therefore behoves the treating team to assess and secure the airway safely and soon. This may be difficult and intubation requires a skilled and experienced anaesthetist. Nasal intubation in patients with fractures potentially involving the anterior base of skull should be undertaken with great care. There are various anaesthetic strategies to minimize the risks.

Tracheostomy in an awake patient under local anaesthesia may be considered, however if the neck tissues are suffused, cricothyroidotomy can be used.

Circulation

Craniofacial injuries, especially those of the midface, can involve severe bleeding from the branches of the external carotid artery that supply the region. Superficial arterial bleeding may be controlled

by pressure or by wound exploration and ligation of the bleeding vessel. Packing may control bleeding from the nasal cavity or oropharynx. In the rare case when this cannot be achieved, arterial ligation may be considered, the maxillary artery via a transantral approach and/or ligation of the anterior ethmoidal artery, which is a branch of the internal carotid system and enters the orbit through the medial orbital wall. Interventional radiological techniques are emerging as useful tools in controlling deep bleeding in this area.

Clinical assessment

This assessment applies to all fractures of the craniofacial skeleton and precedes the special investigations.

The *neurological state* should be determined early using the Glasgow Coma Scale to record consciousness. It is necessary to examine the cranial nerves, and careful ophthalmological assessment may lead to urgent consultation with an ophthalmologist. Testing for light touch in the various zones identifies facial sensory nerve function. The facial nerve is tested by observation and by response to painful stimuli.

The *face* should be examined for lacerations and contour deformities. Bruising around the orbits may indicate an anterior fossa fracture, and bruising around the mastoid process indicates a temporal bone fracture. CSF leakage should be suspected, but in the presence of blood and mucus it is very hard to identify with certainty at this early stage.

Jaw movements and the muscles of facial expression are tested; the cervical spine is palpated for tenderness or deformity and the scalp for lacerations and haematomas.

Each facial region should be palpated systematically from the frontal bone to the mandible, looking for evidence of deformity or abnormal movement. Cooter⁷ has described a systematic sequence of examination by palpation.⁸ Palpation of the inferior orbital margin may help differentiate isolated fractures of the zygoma where the lateral rim is depressed from nasomaxillary fractures where the medial rim is depressed.

The mandible is examined with the patient's mouth slightly open; the margins of the bones are palpated externally. Intraorally, with a gloved finger the alveolar ridges and the hard palate are palpated. Grasping the maxillary alveolar ridge or pressing on the anterior hard palate behind the teeth and applying a rocking movement while palpating the face with the other hand can elicit abnormal movements of the midface.

Imaging

Early imaging

- *Computed tomography:* A cranial CT is performed after the initial resuscitation to assess for possible brain injury. It may be extended to include other areas of suspected injury. Fractures of the anterior fossa may warrant fine-cut bone windows at this stage, but it is the later investigations that will give the definitive information.

- *Angiography:* At this stage, angiography is indicated for penetrating injuries in the region of major vessels or blunt injuries where there is suspicion of vascular damage.

Later imaging

The detailed diagnostic imaging is usually planned after full clinical investigation is complete. It has become regular practice for the first and principal investigation to be a CT scan, however some of the standard radiographic projections can be most useful and in some circumstances essential (e.g. the panoramic radiograph (orthopantomogram, OPG) that shows the mandible, temporomandibular joints and teeth).

Axial CT scanning is vital in managing severe craniofacial trauma of the frontal and nasoethmoid region. Fine-cut two-dimensional CT is the best method for assessing craniofacial fractures around the fronto-orbito-ethmoid region and the fractures are seen most clearly on the scan at right angles to the bone that is fractured. The acquired CT slices, usually axial, can be used to produce a three-dimensional image of the skeleton and/or soft tissues. These images can be rotated and viewed from all angles and are indispensable for surgical planning. These data can be used to create solid nylon models of the injured skull, which can be sterilized and used for reference at surgery or for mock surgical planning.⁹

MRI is often useful later in tracking the origins of CFS leakage. It is used to assess ocular injuries and also to identify non-metallic foreign material, especially wood.

Definitive management

Having resuscitated the patient, established the presence of brain injury, established an infection control regimen and made a clinical and radiological diagnosis, the next step is to establish the priorities in managing the craniofacial injury, in this case the frontal and naso-orbital fractures. Urgent surgical intervention within hours is indicated by the presence of acute neurosurgical problems of intracranial bleeding, penetrating brain injuries or externally compound fractures. Otherwise delayed treatment is preferred by an appropriate team after full investigation and planning in the 'cold light of day'.

Urgent operative intervention may be necessary for injuries to other body systems, and appropriate discussions will determine what parts of the management can be done simultaneously.

Operative surgical principles

Modern craniofacial surgical technique is based on wide exposure of the craniofacial skeleton, the capacity to reduced bony fragments precisely and implement rigid inter fragmentary fixation with titanium mini- and microplates. Where there is absence of bone from the trauma, primary bone grafting of defects is undertaken at this stage.

The most effective and commonly used approach to the naso-orbito-frontal region is the *coronal scalp flap* (Figure 36.3). The incision is placed well behind the hairline and is made in a wavy or zigzag fashion to better disguise the scar. The flap is raised in

the subgaleal plane down to a level about 2 cm above the orbital margins or the site of any fractures of the frontal bones. The dissection then proceeds subpericranially, scraping the bone and extends over the orbital rim into the orbit. The supraorbital neurovascular bundle is preserved and, if necessary, chiselled out of its canal. More medially, the dissection disconnects the trochlea without obvious deleterious effect.

Laterally, it is important to dissect strictly against the temporal fascia beneath the fatty layer directly superficial to it in order to preserve the frontalis branch of the facial nerve. Within the orbit the periorbita may be safely dissected to within 1 cm of the apex, however in traumatic cases the walls may be breached, the periorbita torn and orbital fat trapped in fracture lines, making dissection more difficult.

Medially, in the region of the glabella, the dissection can be taken down over the nose, anteriorly and posteriorly to the canthal ligaments and nasolacrimal ducts. These structures, so often injured, must be identified and preserved.

Access to the anterior part of the medial orbital wall can be obtained via a transcaruncular approach. The floor is exposed by subciliary eyelid incisions or best by transconjunctival incisions with or without lateral canthotomy.

Bone grafting

Bone loss demanding early replacement can occur over the frontal sinus, in the orbital walls, especially medial, and the floor, and after comminuted depressed fractures of the nasal pyramid. In the middle third of the face the vertical bony pillars may need to be grafted to secure midfacial height.¹⁰

The common donor sites for harvesting bone are the iliac crest and medial surface of the ilium, the ribs (both bone and cartilage) and the calvaria. The most important factors

for the survival of bone grafts are: rigid fixation, good soft tissue coverage, a well-vascularized bed and absence of haematoma.

There is a wide selection of all plastic materials to reconstruct the midface and orbits, none of which are favoured by this author. In particular their use in relation to open sinuses should be avoided.

Donor sites

Hip grafts provide strong malleable bone (Figure 36.4). The inner table can be harvested, and additional cancellous bone scooped out for packing (e.g. the nasofrontal duct). The full thickness of the ilium can be used but it is important to preserve the crest in both adults and children.

Rib grafts are harvested through a submammary incision, cartilage can be taken in the form of the costochondral junction for use in the nose and the temporomandibular joint. Ribs can be used whole or more usually split for calvarial or orbital reconstruction (Figure 36.5).



Figure 36.3 A zigzag or wavy coronal incision gives a better final scar. The flap is raised over the pericranium and superficial temporal fascia. The pericranium is incised 1 cm superficial to the superior orbital margin and the dissection continued subperiosteally into the orbit. Staying deep to the fat pad over the superficial temporal fascia will protect the frontalis branch of the facial nerve.



Figure 36.4 A graft taken from the inner table of the right hip.



Figure 36.5 Rib with attached costal cartilage can be used to support the crushed nasal dorsum.

Calvarial grafts have the disadvantage of being less malleable than hip or rib grafts. Small portions can be taken from the outer table where the skull is thick enough, obviating a need for extra visible scars, however contour defects or the skull are often a problem as are complications arising from damage to the underlying dura and brain arising from inexperienced operators. The inner table can be harvested when a craniotomy is being performed and the bone is thick enough. Good quantities of high-quality bone can be obtained in this fashion (Figure 36.6).

Operative management

This depends on the nature of the fracture pattern. Each case must be managed on its merits but there are a number of consistent and significant variations, such as:

- a fracture of the outer table of the frontal sinus with skin intact and a contour defect of the forehead;
- a compound fracture with the outer table involvement as above;
- a compound depressed fracture involving the anterior and posterior walls with suspected involvement of dura and brain; and
- depressed fractures of the frontal bone lateral to the sinus with or without involvement of the orbital roof.

Penetrating wounds need separate consideration due to the extreme risk of contamination.

The *simple depressed fracture* with a contour defect can be approached via a coronal flap; the fragments can be repositioned and secured with microplates or fine wires. If there is an overlying or adjacent wound this can be debrided and the same procedure undertaken.

A *compound depressed fracture involving both walls* is also approached through a coronal scalp flap (Figure 36.7). A small frontal craniotomy is made above the sinus area to expose the posterior wall, the depressed fracture being approached extradurally. If the dura is torn, the posterior

wall of the sinus is nibbled and drilled away (cranialized) and the sinus mucosa meticulously stripped. The dura is repaired in a watertight manner by primary suture and may be augmented by a graft of temporalis fascia or pericranium (Figures 36.8, 36.9 and 36.10).

The *frontonasal duct* can be plugged with muscle and/or bone chips. A pericranial flap pedicled inferiorly is turned in and sutured to the dura in order to seal the frontal sinus. The anterior wall of the sinus is preserved and, if fragmented, treated as in the case of a simple depressed fracture.

Closed anterior and posterior wall fractures associated with CSF leakage are treated expectantly and if the leak persists for more than a week dural repair is performed.

Depressed fractures lateral to the sinuses are usually managed by the neurosurgical team by elevation and if necessary repair of any underlying dural or cerebral damage; however, if the orbital rim and roof is involved, a combined craniofacial approach is often used to reposition the orbital margins and graft the roof.

Penetrating injuries are likely to be associated with contamination along the wound track and consideration must be given to opening the wound track and removing foreign material. Remember that wood is often best visualized by a MRI scan. A frontal craniotomy is performed and if the wound is orbitocranial, the frontal crown can be removed for greater access and replaced. Where there is dural repair under an orbital roof defect, a bone graft will add additional security.

Central depressed fractures of the frontal region may be associated with naso-orbito-ethmoid fractures that extend to the floor of the anterior cranial fossa, hence often complicated by CSF rhinorrhoea. A combined neurosurgical/craniofacial approach is needed to deal with this situation. The anterior fossa is explored and the fistula repaired intradurally and the naso-orbito-ethmoid fractures are repaired in sequence from posteriorly to anteriorly with grafts, fixation plates and canthopexies (Figure 36.11).

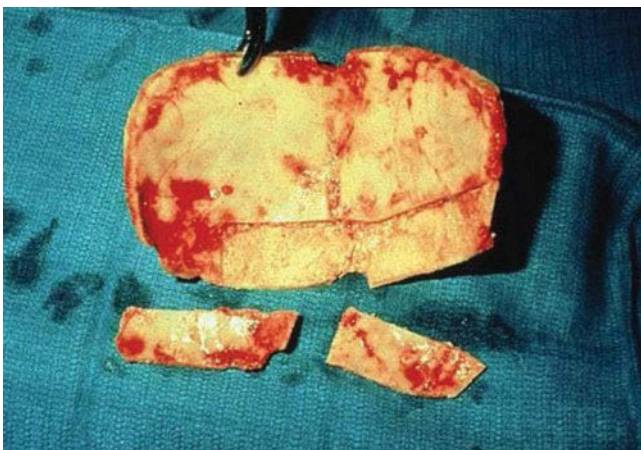


Figure 36.6 Bone can be taken from the inner table of the craniotomy bone flap if the skull is thick enough.

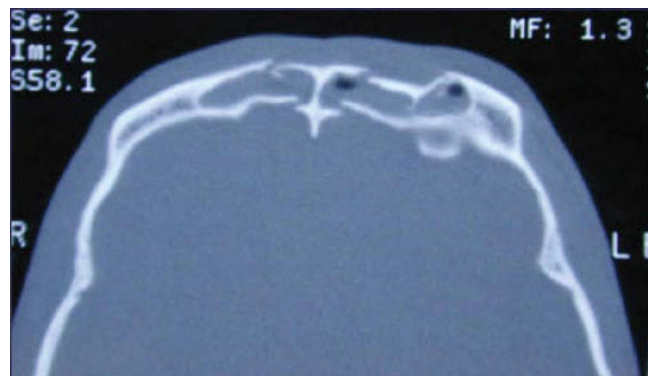


Figure 36.7 CT scan showing fractures of the anterior and posterior walls of the frontal sinus.

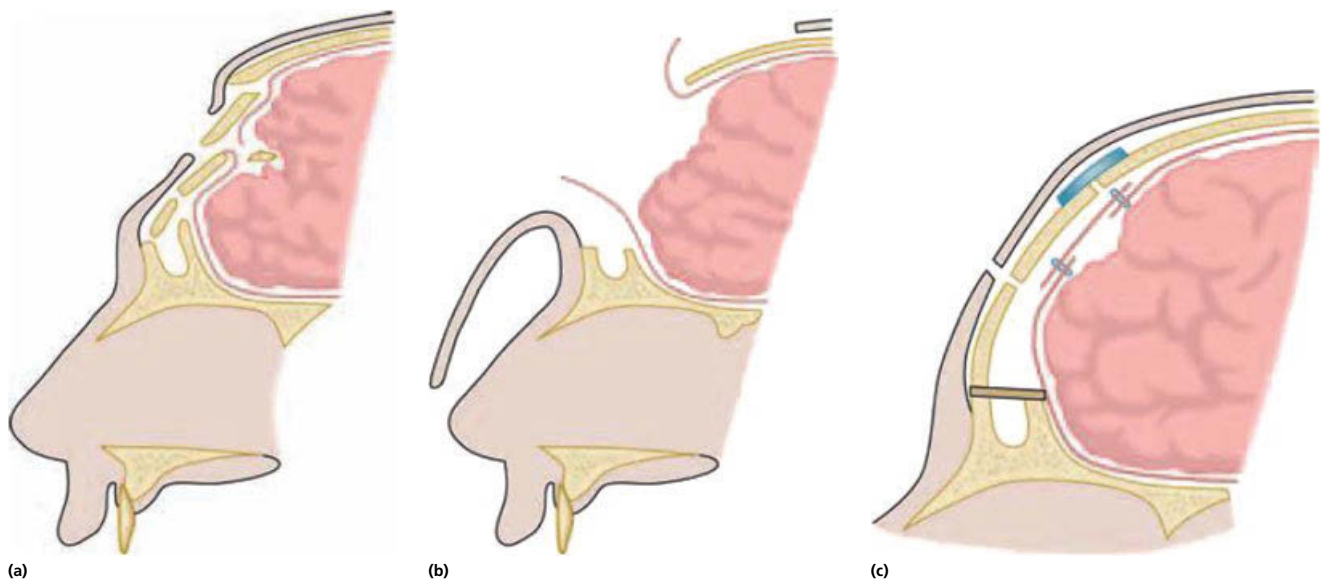
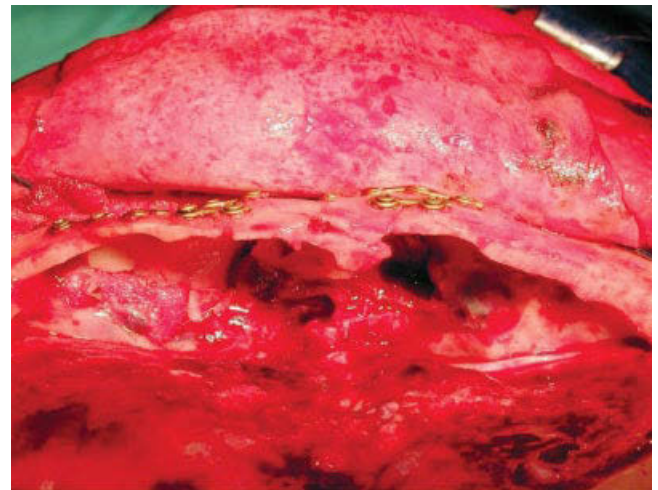


Figure 36.8 When there is a compound comminuted depression of the frontal bone involving both tables of the sinus and tearing dura and/or brain, the coronal flap exposes the area, the depressed fragments are removed, the area is gently cleansed, the dura repaired, the posterior sinus wall removed, the sinus ostia blocked and the frontal bone reconstructed with microplates or fine wires.



(a)

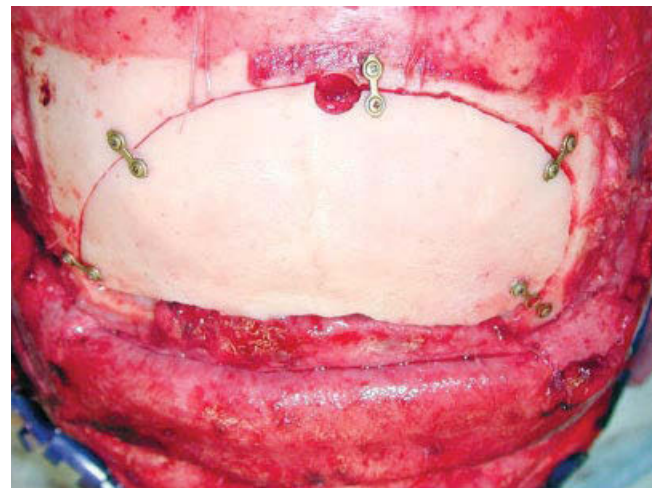


(b)

Figure 36.9 (a) The fracture exposed via the coronal flap. (b) The sinus has been cranialized, the dura repaired and the outer table fragments plated.



(a)



(b)

Figure 36.10 (a) An anteriorly based galeal or pericranial flap can be raised, and used to help obliterate the sinus and close off the ducts. (b) The flap is attached to the dura and coats the floor and walls of the anterior fossa; it is seen here 'posted' between the frontal bone flap and the reconstructed frontal crown.

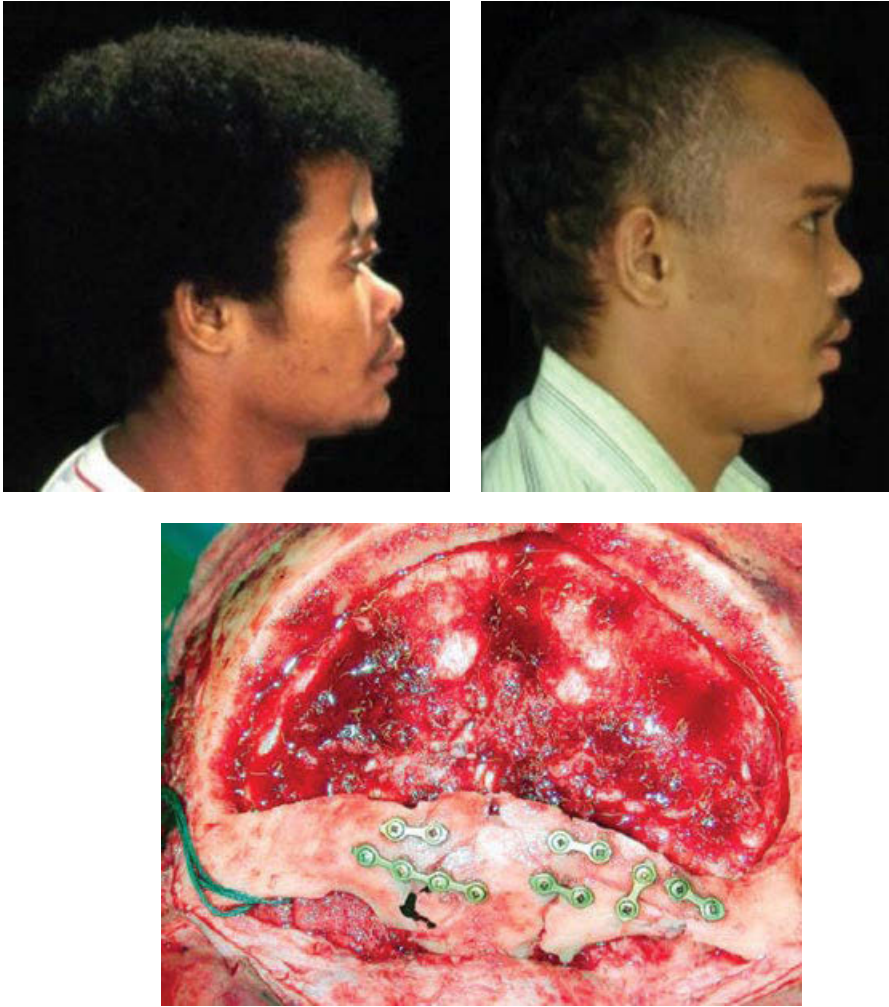


Figure 36.11 A completed frontal reconstruction after a severe frontal crush.

Naso-orbito-ethmoid fractures

These fractures occur as a result of direct trauma to the mid-face and, although they can occur in isolation, are frequently associated with other craniofacial fractures. Management is challenging because of the intricate and complex skeletal anatomy in close juxtaposition with the canthal ligaments, the nasolacrimal apparatus and the cribriform plate and brain above. Exposure, reduction and maintaining fixation have always been and still remain difficult.

Classification

These fractures may be unilateral or bilateral, simple or comminuted, closed or compound; they may occur in isolation or in association with more extensive damage to the craniofacial skeleton.

Multiple classifications have been proposed, Markowitz *et al.*¹¹ proposed Types I–III with increasing measure of complexity in each group. Gruss¹² attempts to relate to the fractures in adjacent bones. The problem with classifications in this area is the complexity and the difficulty of finding a language that reflects the

reality of each clinical situation.¹³ The author's view is that the closest method that is useful for measurement and comparison is one of alphanumeric scoring which quantifies the extent and pattern of bony damage in a global fashion.¹⁴

Principles of treatment

Historically, these fractures were treated by closed reduction and external splintage; the results were not only poor but rendered the secondary corrective surgery very difficult. Credit must be given to Mustardé,¹⁵ Dingman and Natvig¹⁶ and Stranc¹⁷ for identifying the need for open reduction and internal fixation, at the time, with wire. However restricted access and limited radiological assistance with diagnosis meant that the results remained suboptimal.

The modern approach is much aided by CT scanning, involves wide exposure using the coronal approach, accurate anatomical reduction becomes possible and fixation with micro- and mini-plates and screws ensures stability. In addition, primary bone grafting to delicate medial walls and graft support for the depressed comminuted nasal pyramid has vastly improved patient outcomes (Figure 36.12).¹⁸

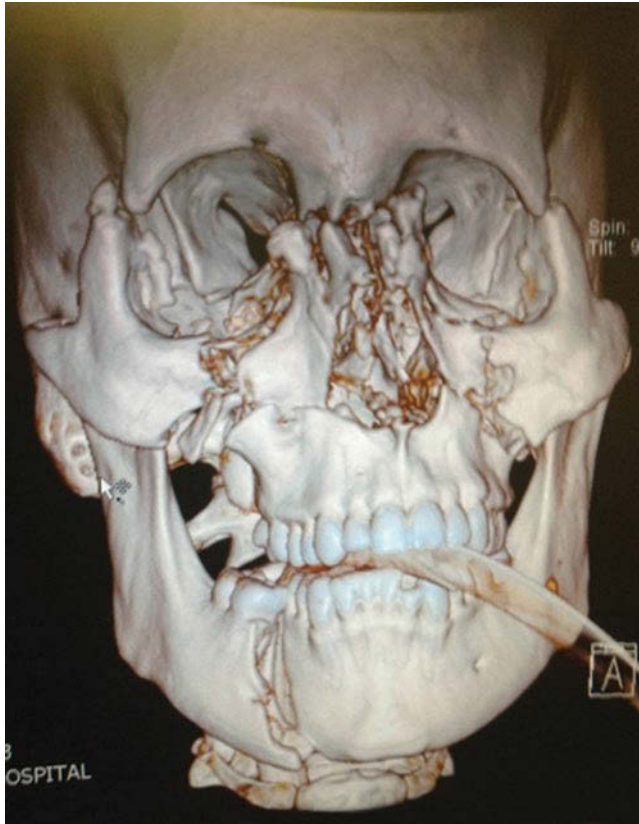


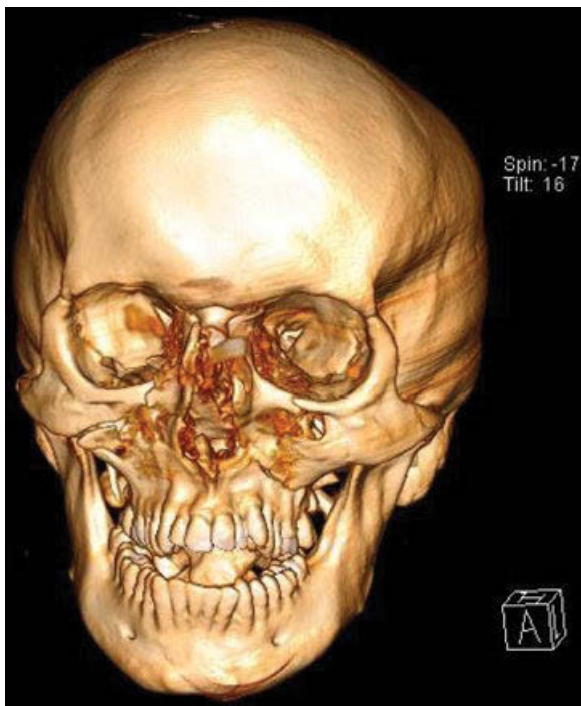
Figure 36.12 CT fracture.

Operative management

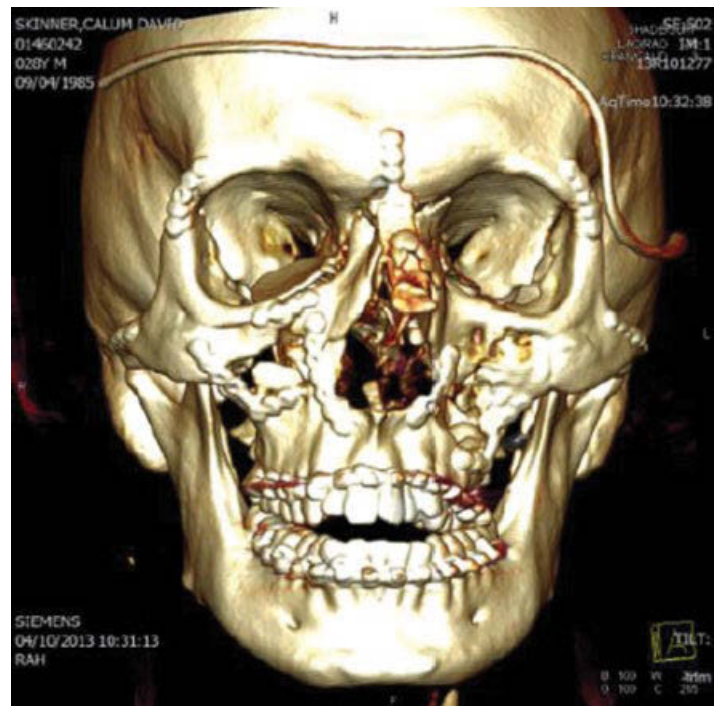
The coronal scalp flap extended to the supra orbital margin then subperiosteally into the orbit will give tension-free exposure of the medial orbital walls, nasal bones and frontal processes of the maxillae as well as the orbitofrontal region. Care must be taken to preserve whatever bony attachment there is of the medial canthal ligament.

Transconjunctival incisions or subciliary lower eyelid incisions are used to expose the inferomedial orbital wall and orbital floor. An upper buccal sulcus incision exposes the nasomaxillary buttress and the margins of the pyriform aperture. In the case of compound fractures, local lacerations may be useful for access.

When the naso-orbito-ethmoid complex is fractured and displaced en bloc the fragments can be reduced and controlled by plate and screw fixation to the adjacent stable skeleton. In these cases the canthal attachments in continuity with the major bony fragment and its correct position is restored. In the more severe comminuted cases it is necessary to determine if the canthal ligament is attached to a fragment of bone. If this is so, this attachment is preserved and may be used to maintain the correct intercanthal distance. Transnasal wiring of the canthi is achieved by placing the wires posterior and superior to the lacrimal fossa. The wires are tightened; if there is much loss of bone the wire that is attached to the ligament can be passed through a small bone graft on one or both sides. Reconstruction of the medial walls is usually necessary; the author prefers thinned out inner table of ilium for this purpose. It is important to remember to reconstruct this area before tightening the intercanthal wires thus denying you access! The repair proceeds from back to front (Figure 36.13).



(a)



(b)

Figure 36.13 (a, b) A panfacial fracture where the naso-orbito-ethmoid complex has been repaired from posterior to anterior: the medial orbital walls grafted, the nasomaxillary processes plated, hence restoring the canthal ligaments, and a cantilever bone graft to the dorsum of nose to support the integument against collapse and contracture.

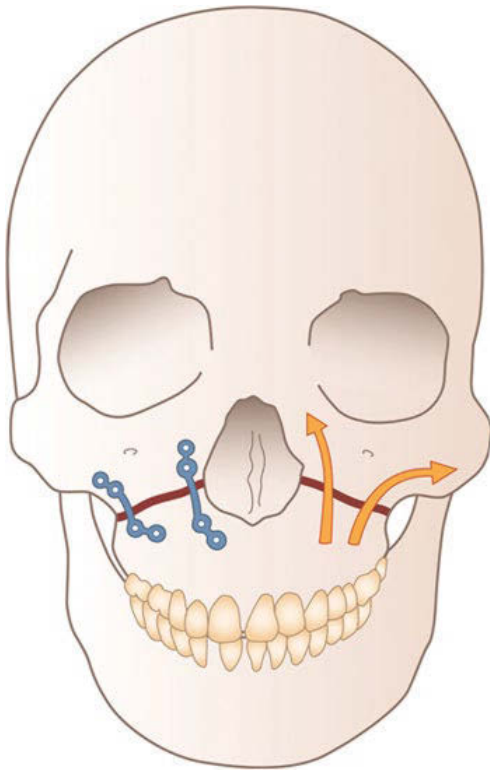


Figure 36.14 This shows the load-bearing direction of the buttresses, and on the right how plates are positioned on these lines.

In the comminuted cases, use of a costochondral graft fixed as a cantilever restores the nasal bridge projection and maintains the soft tissue expansion, which aids and minimizes secondary reconstruction, which is so often necessary in this condition.

With respect to the nasal septum, primary open reduction is not attempted beyond centralization by closed manipulation.

The management of the associated frontal sinus and cranial fractures are repaired at the same time. This implies a team working in harmony. It is the author's experience that a written preoperative plan setting out the sequence of events from beginning to end of the procedure facilitates harmony.

Management of nasolacrimal injuries does not usually involve early exploration, except where there is an obvious lacrimal system transection in an external laceration. Repairing this over a silicone tube is the preferred option. Despite its intimate proximity to the fractures, the nasolacrimal system is not often damaged, even with severe injuries.¹⁹ When late obstruction does occur, it is usually in the bony nasolacrimal canal. The more accurate the initial reduction, the less need for later dacryocystorhinostomy²⁰ (Figure 36.14).

Zygomatico-maxillary fractures

The face is generally described as having an upper, middle and lower third. The middle third is formed mostly by the maxillae, zygomatic bones and the nasoseptal complex. This section deals

with fractures of the zygoma and maxilla. As these bones articulate with each other, form part of the orbit, and the tooth-bearing maxillae form the upper jaw which articulates with the base of skull and occludes with the mandible, fractures of this complex may not be simple. Trauma to the middle third of the face has the potential for severe functional, psychosocial and aesthetic consequences. Whereas fractures of the mandible remain the most common form of single facial fracture, the zygoma is the commonest when multiple facial fractures occur. The ratio of males to females remains high, 3–4:1.^{2,21}

Anatomy

The zygoma and maxillae are part of the face between the lower jaw and the base of skull that together with the nasoethmoid complex compose the majority of the bones generally described as the middle third. It is important to understand the blood and nerve supply to the maxilla and upper dental arch. This assists with planning, safe incisions and for understanding the patterns of numbness of the skin and palate associated with maxillary fractures. The greater palatine canal transmits the greater palatine artery and nerve which supplies all of the bone and mucosa of the hard palate. Branches of the maxillary artery and maxillary nerve pass into the posterior maxilla through small foramina to become the posterior superior alveolar artery and nerve. This nerve supplies the molar teeth with a dental plexus. Branches from the infraorbital artery and nerve enter the anterior maxillary bone from the orbital floor and supply the anterior teeth. Fractures of the orbital floor and the anterior maxilla may result in numbness of the anterior teeth, whereas fractures extending low in the maxilla posterior to the first molar region may result in numbness of the posterior teeth. It is important to know that the maxilla gains blood supply from the gingival attachments to the alveolar bone and through its attachments to the soft palate from the pharyngeal palatine branches of the facial artery and the ascending pharyngeal branches of the external carotid artery. Both the trauma and the surgical exposure may significantly compromise the blood supply to fracture fragments.

There are strong horizontal elements in the maxillae, alveoli and hard palate which make up a foundation to support the three paired vertical bony condensations or buttresses (Figure 36.15).

The most anterior of these extends from the dentoalveolar arch in the region of the lateral incisor/canine region superiorly along the pyriform margin to the medial orbital rim and frontomaxillary suture. The middle buttress extends from the first molar tooth to the body of the zygoma and through this bone as the lateral orbital wall to the frontozygomatic suture. The posterior buttress is represented by the attachment of the maxillary tuberosity to the pterygoid plates and hence to the sphenoid bone.

The biomechanical stresses in the midface have been harder to assess than those in the mandible; however, the load paths for distribution of force appear to be the buttresses described above.²



Figure 36.15 This 3D image of the skull shows a fractured zygoma on the left and a normal bone on the right. The articulation with the sphenoid bone forms the lateral orbital wall, with the frontal bone at the fronto-zygomatic suture, with the temporal bone to form the zygomatic arch (visibly deformed in this image), with the maxilla in two places to form the inferior orbital rim and the zygomatico-maxillary buttress.

The zygoma makes a key contribution to the orbital contour, facial width and projection. Comprising as it does the inferolateral orbital rim, the zygoma articulates with the frontal bone superiorly, the maxilla inferiorly and medially and with both sphenoid and temporal bones posteriorly and laterally.

Four bony processes radiate from the zygomatic bone, providing strong articulations with the frontal, maxillary and temporal bones as well as the greater wing of the sphenoid bone. The bony junction between the temporal process of the zygoma and the zygomatic process of the temporal bone constitutes the zygomatic arch. The temporal fascia and masseter muscle are attached to the arch. These muscles are usually considered to be the major deforming force in maintaining displacement of a depressed zygomatic fracture. The superficial temporal vessels and frontal branch of the facial nerve lie immediately superficial to the arch. Deep to it pass the internal maxillary vessels, temporalis muscle and coronoid process. It is important to understand the value of maintaining the zygomatic arch when locating the zygoma appropriately in relation to the skull base in an antero-posterior, vertical and transverse dimension.²¹

Classification

In classifying occlusal fractures of the maxilla we seem to be stuck with Le Fort's original description. The Le Fort I pattern is a low horizontal fracture through the pyriform aperture and the zygomatic buttresses and pterygoid plate. The Le Fort II fracture has the upper detachment at a fracture line running across the nasal bones, through the inferior orbital rims and through the zygomatic buttresses and pterygoid plates. The Le Fort III pattern has the upper jaw detached from the base of skull at a

fracture line running through the nasofrontal suture, ethmoid bone, frontozygomatic suture and arches of the zygomas and the pterygoid plates.

The language associated with this form of description is now so embedded that it ceases to be scientifically meaningful; it does not take into account the degrees of comminution and displacement of fragments, nor does it mention the parasagittal fractures of the hard palate and the smaller dentoalveolar segment fractures of the upper jaw. It certainly does not relate the severity of the maxillary fracture to other areas of the craniofacial skeleton. It is the author's belief that alphanumeric classifications are superior in this respect.¹⁴

Classifications of zygomatic fractures, are many and varied.^{22–24} With the advent of CT scanning together with improved methods of operative exposure and fixation of the orbital zygomatic region, the extent rather than the pattern of fracturing was seen to be of greater significance. Gruss²² recognized the role of the zygomatic arch in accurate reduction and classified these injuries according to the level of bony damage to the zygomatic body and zygomatic arches. Cooter and David¹⁴ quantify the degree of bony comminution of each element of the zygoma as well as the level of displacement of the skeletal articulations.

Clinical assessment

With respect to fractures of the *maxilla*, the signs depend on the degree of displacement and comminution and indeed whether the fracture extends into the orbits and skull base and whether the zygoma's are involved as well. Intraoral examination may show the upper dental arch to be intact or it may be fragmented. There may be mobile dental alveolar segments as well. Malocclusion may be evident: the patient may notice it or it may be obvious on inspection. It is important to be mindful of pre-existing occlusal disharmony. In the typical fracture of the upper jaw with an intact alveolar arch, posterior and inferior displacement the occlusal surface will result in an open bite.

The external examination shows cheeks that are typically swollen, and there may be bleeding from the nose and bruising. There may also be emphysema from the air forced out of the paranasal sinuses. If the fracture extends into the orbit, swelling and bruising of the eyelids may be a dominant feature. A gloved figure on the hard palate exerting an upward and rocking force may elicit pain, crepitis and abnormal mobility. Diastases at the fracture sites may be felt at the nasofrontal and frontal zygomatic sutures.

When managing fractures of the zygoma, it is important to record the mechanism, direction and particularly the force of the impact, as this will assist in understanding the pattern of fracture. These observations may be very important in later medico-legal proceedings, particularly in cases of interpersonal violence. The association of zygomatic fractures with other

midface injuries, in particular fractures of the maxilla, has been noted above (see Figure 36.15).

The symptomatology typically relates to the effect of the displaced zygomatic bone on the surrounding soft tissues. The orbital soft tissues manifest swelling, ecchymosed and subconjunctival haemorrhages, particularly laterally. Double vision or blurred vision may be complained of due to orbital soft tissue swelling, ocular dystopia or primary ocular injury. Numbness or altered sensation in the distribution of the infraorbital nerve indicates a fracture in the region of the orbital rim. If the zygoma is medially displaced, trismus and complaints of malocclusion are noted when the arch fractures impinge on the underlying temporalis muscle. Patients sometimes complain of altered occlusion and in this situation occlusal disturbance by extension of the zygomatic fracture into the maxillary alveolar process must be excluded by careful intraoral examination.

Physical examination and especially palpation will readily identify displacement. Observation from above the patient provides an impression of loss of the zygomatic prominence.

The examining fingers are placed on the prominent part of the zygomatic body and will give a comparison with the non-injured side. When there is displacement of the inferior rim, there may be a palpable step at the infraorbital margin and this is usually painful. In isolated fractures of the zygomatic arch there may be a depression which is palpable. When the zygomatic fractures are part of extensive midface fracturing, for example in conjunction with maxillary fractures, the zygomatic arches bow outwards and the anteroposterior projection of the zygomatic prominence is diminished.

Intraoral examination shows bruising in the upper buccal sulcus. Alveolar extensions of the fracture or malocclusion caused by trismus or associated with midfacial and mandibular fractures can be noted at this time.

It is important to have a detailed ophthalmological examination similar to that necessary in isolated fractures of the orbital walls.

Imaging

Fractures of the middle third of the face were once investigated by plain radiology. This has almost completely disappeared and the most useful radiological investigation has become the CT scan with axial, coronal and three-dimensional reformatting. This is the standard radiological examination for fractures of the upper jaw and orbit and all but the most simple fractures of the zygoma.

Axial cuts show fractures of the posterior wall of the maxillary antrum and of the pterygoid plates. It is possible to identify midline fractures of the hard palate as well as dentoalveolar segmental fractures. Coronal CT scans of the midface give detailed information of the anterior maxilla and the relationships with nasoethmoid and orbitozygomatic fractures. When the fractures are primarily of the orbitozygomatic region, then the radiological analysis is similar to that described in the section on Orbital fractures.

Principles of management

The mainstay of surgical treatment for zygomatic fractures was for many years the Gillies technique of elevating by leverage from an incision in the hair-bearing scalp of the temple.²⁵ The Dingman variation¹⁶ or elevation from a lateral eyebrow incision is a more useful and elegant method as it also affords access to the lateral orbital wall and allows for frontozygomatic inter-fragment fixation.

An intraoral upper buccal sulcus approach gives access to the arch and body of the bone which can be elevated and fixation can be made at the zygomatico-maxillary buttress. Because of the pull of the masseter muscle, it became common practice, in order to prevent relapse, to add fixation using a wire at the frontozygomatic suture. It is now standard practice to employ miniplate fixation. Miniplate or microplate fixation together with wide operative exposure of the orbitozygomatic skeleton allows anatomical restoration of the zygomatic prominence with fewer positional disturbances of the globe, such as enophthalmos and vertical global dystopia.

Following clinical examination and CT confirmation of the fracture pattern, it becomes important to identify fracturing and comminution in and around the zygomatico-maxillary buttress and the inferior orbital rim, disruptions of the arch and at the zygomatico-frontal suture. Because of the deforming and displacing effects of the masseter muscle, the placement of adequate mini- or microplate fixation is necessary. The techniques of exposure, reduction and fixation are now well described. It becomes logical to subdivide fractures of the zygoma to those where there is no orbital wall involvement (the isolated zygomatic arch fracture) and the more common orbitozygomatic fracture and also those associated with other midfacial fractures, or the panfacial fracture.

Isolated zygomatic arch fractures

These result in the main from low-velocity injuries and the characteristic symptoms are those of trismus and a depression at the site of impact. This injury is usually best treated by the Gillies technique of closed reduction. A short oblique incision in the hair-bearing temple allows passage of an elevator deep to the temporalis fascia behind the depressed arch. This technique produces a stable reduction in majority of cases. If the arch fracture is not corrected, the cosmetic deformity remains and in extreme cases there is some long-term disturbance of jaw opening.

Orbitozygomatic fractures

An undisplaced fracture does not need operative intervention.¹⁶ Displaced orbitozygomatic fractures require careful open reduction and internal fixation on an individualized basis. Exposure via an eyebrow incision, a lower eyelid subciliary

skin muscle flap or transconjunctival incision reveals the zygomatico-frontal suture, lateral orbital wall inferior orbital rim, orbital floor and interior zygomatic arch. An upper buccal vestibular incision permits exposure of the zygomatico-maxillary articulation and anterior attachment of the masseter muscle to the zygoma. Following fracture mobilization and elevation, either by the intraoral approach or by using the elevator through the eyebrow approach, an anatomical restoration is achieved by visualizing the zygomatic articulations and checking the alignment of the zygoma with the greater wing of sphenoid. Miniplate fixation in the zygomatico-maxillary buttress is effective, however there may be comminution in this area and sometimes the bony attachments of the plate are thin. The zygomatico-frontal suture on its own is often not enough but it does provide the strongest bone for miniplate fixation. Two-site fixation will provide sufficient stability in the majority of fractures. When the orbital floor needs exploring then the orbital rim can be plated as well through the lower lid or transconjunctival incision.

Comminuted and grossly displaced orbitozygomatic fractures seldom occur in isolation and are almost always the result of high-velocity impacts producing major midface fractures involving the maxillae. In those cases, recognizing the disruption and restoring the arch is key to restoration of zygomatic projection and hence a very good marker for fixation of adjacent fractured bones.²⁶ In these cases a coronal scalp flap facilitates exposure, mobilization and reduction of the arch fractures.

Comminution and loss of bone at the major articulations, the anterior maxillary wall, the orbital floor and the lateral orbital wall demands careful operative exposure, reduction and reconstruction by primary bone grafting to avoid enophthalmos and orbito-ocular dystopia.

When dealing with *maxillary fractures*, it is important to have a preoperative dental assessment. Impressions of the dental arches and teeth can be used to cast a plaster model which is then cut through the site of the fractures. Restoration of the pre-morbid occlusion can be planned and an acrylic bite wafer constructed, which can be used intraoperatively and postoperatively to maintain correct occlusal relationships.

There is some discussion in the literature about the timing of definitive treatment of maxillary fractures. The desirability of performing surgical correction of midface fracture as early as possible after injuring has been championed by Gruss and Mackinnon²⁷ and Manson *et al.*,²⁸ their argument being that early accurate bony reconstruction will prevent the development of soft tissue contractures and hence post-traumatic deformity. It is the author's belief that there are other factors that must be considered when deciding on the correct timing of surgery. The foremost of these is consideration of the priorities of management of other injuries, such as associated brain injuries and other associated limb and intra-abdominal problems. It is also useful to have time to properly plan and assemble an operative team to be able to give the appropriate time and expertise to the restoration of the facial injury.²

Airway management may be compromised with other jaw fractures and great care needs to be taken when considering placement of endotracheal tubes at the time of surgery. Particular care should be taken with panfacial fractures, which may extend through the cranial base. Where there is massive craniofacial disruption with haemorrhage then there is a role for tracheotomy.

Fracture reduction by disimpaction used to be carried out using somewhat brutal forceps inserted with one prong in the nasal cavity, the other in the oral cavity so as to grip the palate bilaterally. It is possible to inflict further damage with this instrument. With the advent of wider exposure through coronal, buccal and conjunctival approaches, more delicate instrumentation can be used to diss-impact the fracture sites.

Stabilization of fractures is of course now achieved with miniplate fixation and when there is comminution and loss of bone at the buttresses, primary bone grafting is performed.

Once the impacted displaced maxilla has been reduced, the upper and lower jaws are stabilized in the pretraumatic occlusion using arch bars, the wafer and intermaxillary wiring. Where the zygomas are also fractured, these should be reduced as described above. They then provide a reference point for re-establishing the vertical buttresses.

In the case of an uncomplicated Le Fort I fracture, the vertical height can be determined easily by opposing the vertical buttresses at the pyriform margin and zygomatico-maxillary region, which can then be plated. Even when there are other fractures of the midface, at least one of the four anterior vertical midface buttresses will usually not be severely comminuted and so provide a guide for the correct vertical relationship. Once this has been established, miniplates are used to stabilize the reduced fracture using at least two screws on either side of the fracture. If there is loss of bone then bone grafting may be needed in one or more of these vertical buttresses. Once the vertical height has been established and stabilized, with or without bone graft, the maxillary fixation can be released, the surgical fields irrigated and the wounds closed. The jaw opening is controlled with light elastics and the wafer removed after several days when the patient is able to bite in the correct position (Figures 36.16, 36.17 and 36.18).

Management of complex midfacial fractures when the zygomas, maxillae and indeed nasal ethmoid fractures are involved (that is, fractures above the Le Fort I level) need different treatment sequences. The occlusal complex (i.e. mandible and maxilla) is stabilized and brought together in occlusion with the help of a preplanned occlusal wafer. The zygomatic orbital complexes are stabilized usually via a coronal scalp flap to give the forward projection offered by accurate reduction of the zygomatic arch. The occlusal complex can then be united with the orbital complex via the four vertical buttresses. Fixation of the buttresses can be with miniplates and/or bone grafting if necessary. Finally, the nasal ethmoid complex can be reconstructed by meticulous interfragmentary miniplate fixation and the nasoethmoid area as described earlier.

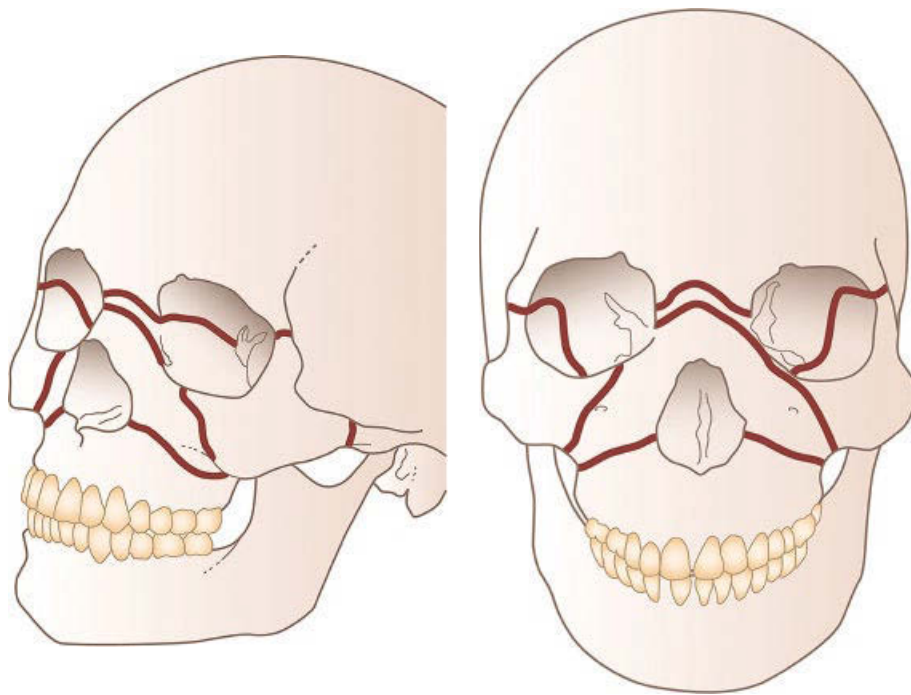


Figure 36.16 Maxillary fracture classification of Le Fort: The classical fracture lines – horizontal (I), pyramidal (II), total disjunction (III).

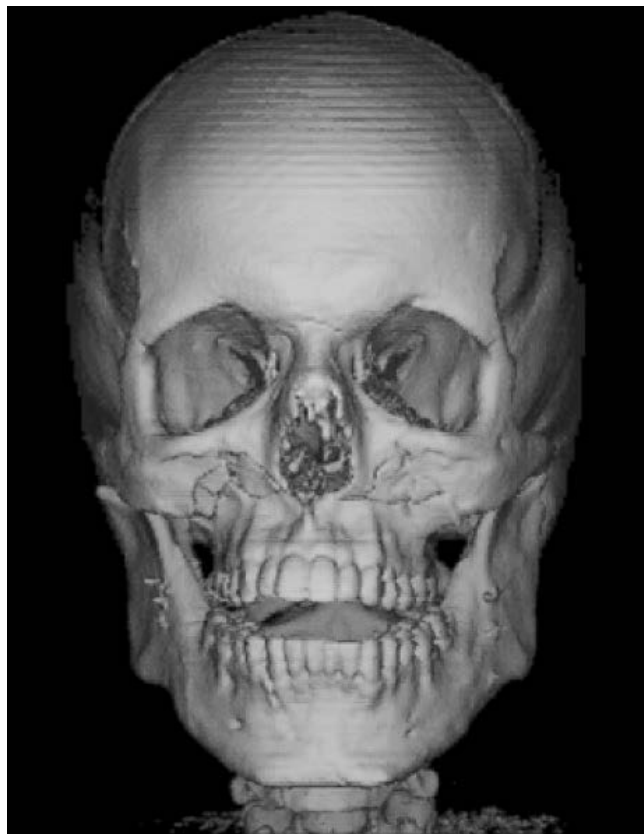


Figure 36.17 A typical Le Fort I fracture of the upper jaw – the fracture line is horizontal; there is an intact alveolar arch. The posterior and inferior displacement of the arch and palate result in the open bite seen clearly on this 3D image.

Complications

Zygoma

These include:

- deformity
- prominent plates
- disturbance of globe position
- ocular injury
- nerve injury.

Deformity was more common with older techniques of management where there was inadequate exposure and fixation. Soft tissue deformities, including sagging of the eyelid and cheek, after zygomatic dissections require meticulous repositioning of soft tissues (Figure 36.19–36.22).

Prominent plates are a common complication which may require plate removal on a subsequent occasion.

Disturbance of globe position in both the anteroposterior and vertical dimensions remains a major long-term complication. Enophthalmus and vertical globe dystopia are cosmetically and functionally disturbing. They are very hard to correct and in some cases may require osteotomies of the orbit to reposition part or the entire orbital box.

With regard to ocular injury, direct globe injuries are common in association with these fractures. The fracture of the lateral wall of the zygoma that produces the ‘cigar cutter technique’ can injure the optic nerve. Ophthalmological investigation will determine whether or not there are any retinal injuries as a result of the trauma.

Nerve injuries most commonly involved are those of the infraorbital nerve, causing persistent numbness, paraesthesia or

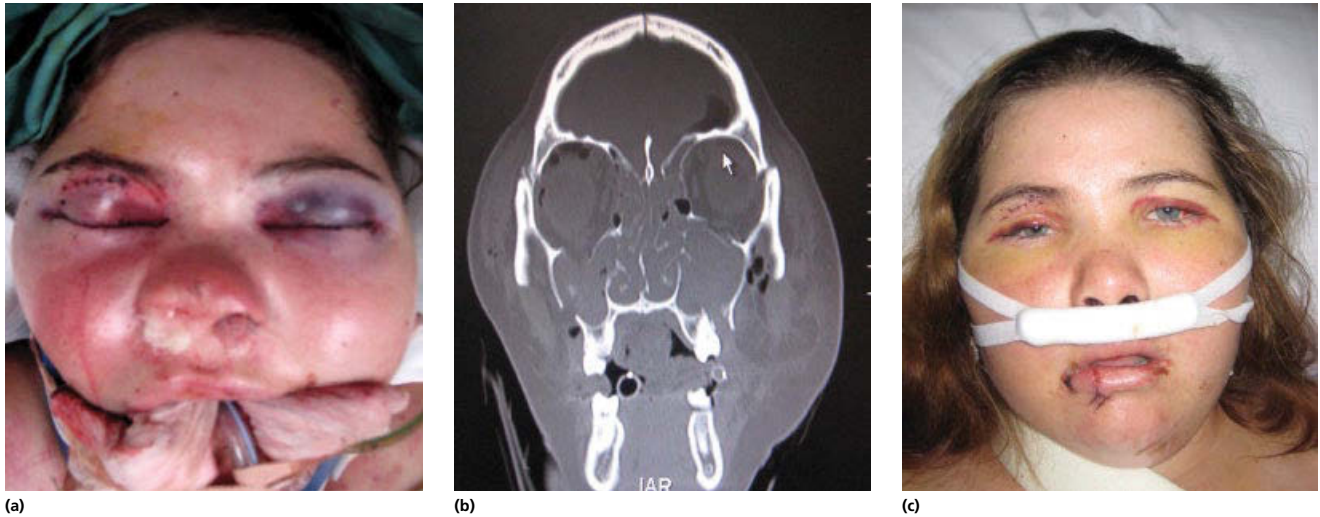


Figure 36.18 A patient with fractures at all three Le Fort levels. (a) Swollen, bruised, nasal and oral packing to control haemorrhage, and intubated to protect her airway. (b) The coronal CT slice shows the endotracheal tube, the oral packing and the fractures. (c) After 7 days, extubated, lacerations sutured, swelling abating, clinically there is right enophthalmos, flattening of the cheeks, flattening and widening of the nasoethmoid region and an anterior open bite.

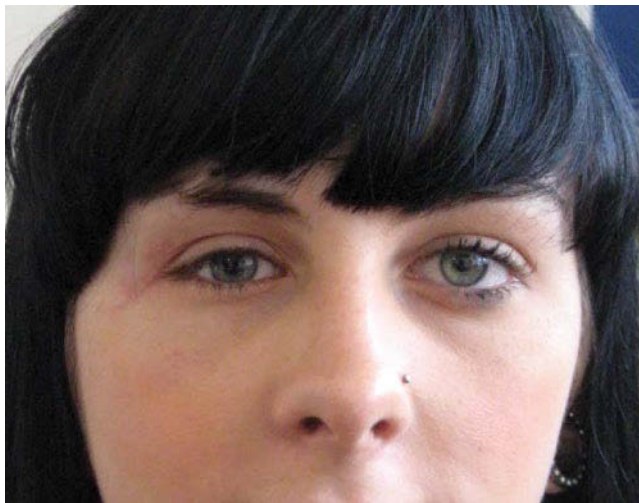


Figure 36.19 Flattened right zygoma, the arch is bowed out, and the palpebral fissure is narrowed due to the enophthalmos.

hyperaesthesia in the distribution of this nerve. Most patients experience significant recovery of function early but occasional patients experience progressive improvement over a longer period of time, up to 18 months or more. A very small number go on to have disaesthesia, which can be distressing.

The maxillae

These include:

- malunion and malocclusion
- non-union
- facial deformity
- infection
- nasal obstruction
- lacrimal obstruction.

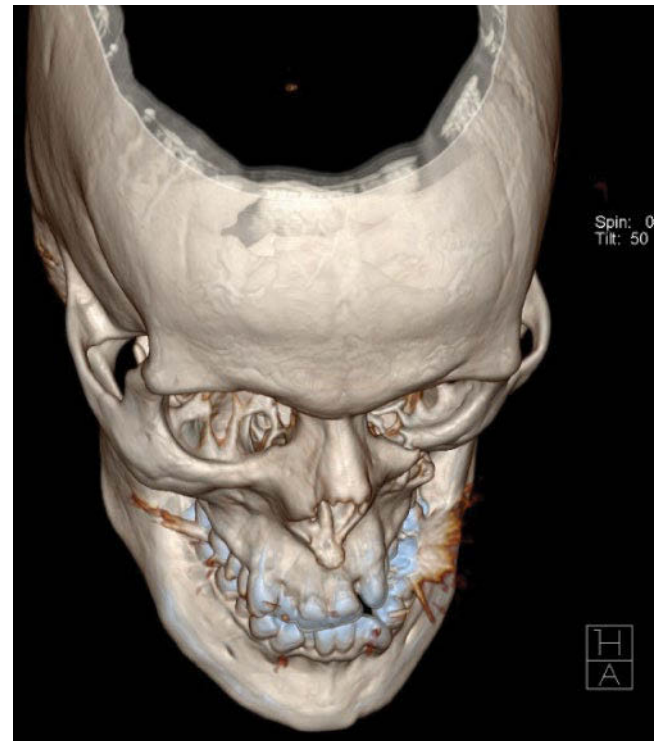


Figure 36.20 Depression of the zygoma which is both visible and palpable.

Malunion with associated malocclusion and loss of normal facial form are most significant complications of upper jaw fractures, particularly where there has been severe comminution in and around the midfacial buttresses. Deformity may require early intervention if there has been some relapse; however, long-term assessment leading to osteotomies and onlay bone grafts may be the answer in complex cases.

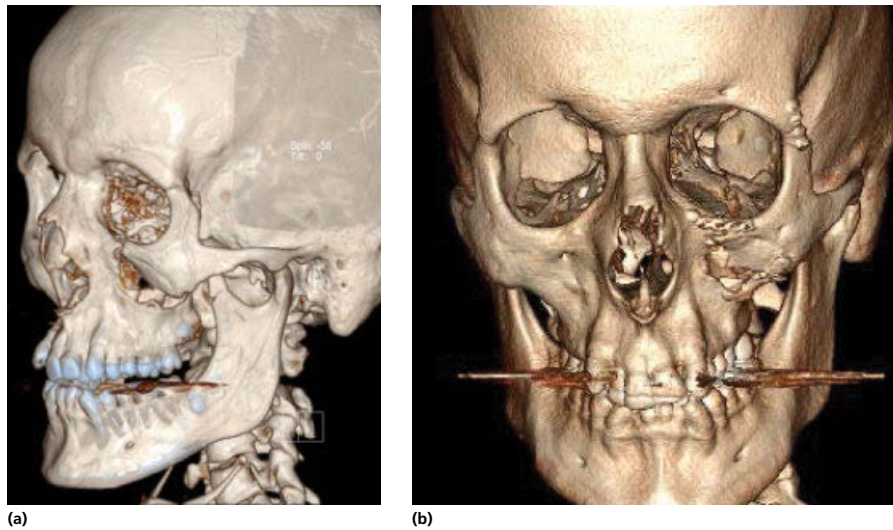


Figure 36.21 (a, b) Open reduction and internal fixation of a fracture of the left zygoma. Access was gained via a buccal sulcus incision, an eyebrow incision and a transconjunctival incision. Plates are seen at the frontozygomatic suture, the inferior orbital rim and the zygomatico-maxillary buttress. The orbital floor has been reconstructed with iliac bone.

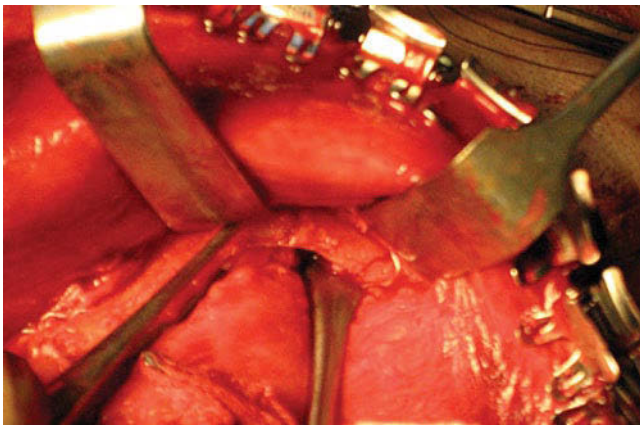


Figure 36.22 Right zygomatic arch fracture exposed via a coronal scalp flap.

Facial deformity can also result from an alteration to the facial contour, for example where the anterior wall of the maxilla has been fragmented and there is scarring between the maxillary fragment and the overlying integument. The wide dissection necessary to secure adequate reduction is itself a cause of scarring at the periosteal level, which produces an ageing effect.

Infection tends to occur in and around plates and screws but in the long term, patients can come back possibly many years later with sinusitis resulting from obstruction to the sinus drainage or indeed screws that have eroded into the sinus cavities.

Nasal obstructive symptoms may follow upper jaw fractures through fracture dislocations of the nasal central cartilage bone. Correction is usually performed once the mucosa has soundly healed, at which time a septorhinoplasty may be performed.

Lacrimal obstruction is common in those fractures that involve the nasal ethmoid complex (Figures 36.23, 36.24, 36.25, 36.26, 36.27, 36.28 and 36.29).

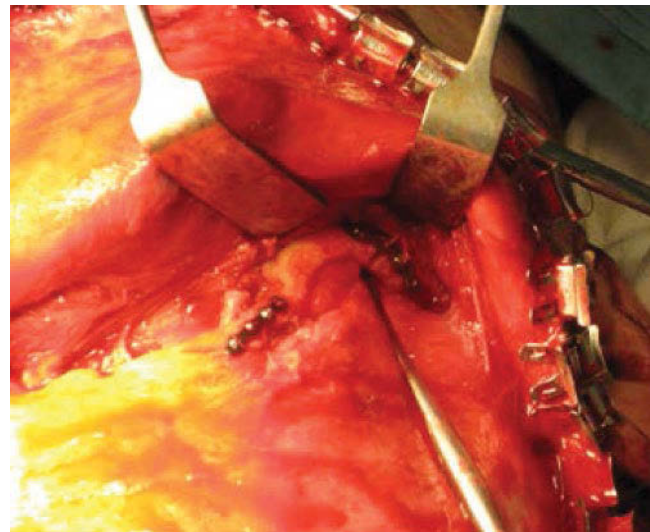


Figure 36.23 The forward projection of the zygoma is assured by accurate reduction and plating of the zygomatic arch. Access is via the coronal flap and the fronto-zygomatic diastasis has been reduced and plated.

Orbital fractures

Because the orbit is situated between the cranium above and the midface below, it is frequently involved in the other fractures described in this chapter, that is the zygoma and midface, the naso-orbito-ethmoid, frontal and orbito-zygomatic fractures. Advances in imaging have led to a greater understanding of the anatomy of fractures; this in turn has led to more meaningful correlation between observed pathology and symptoms. A good example of this is the ability to image the medial orbital wall and understand the relationship between these fractures and persisting enophthalmos when they go uncorrected (Figure 36.30).²

Advances in imaging permit preoperative planning; the possibility of wide exposure enables reconstruction of both skeletal

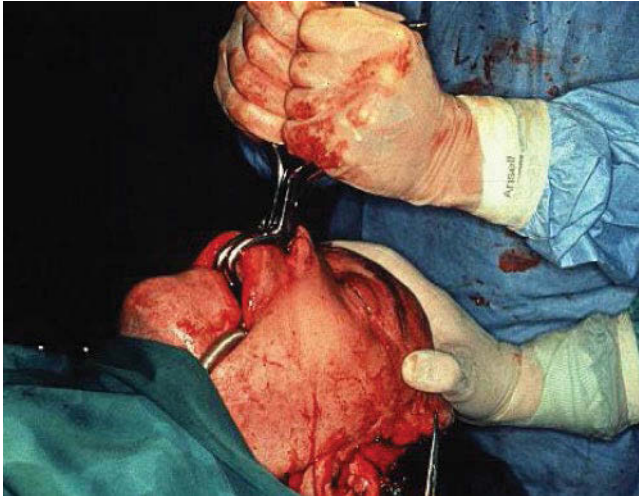


Figure 36.24 Row's dissipation forceps can cause further damage to already compromised tissue.

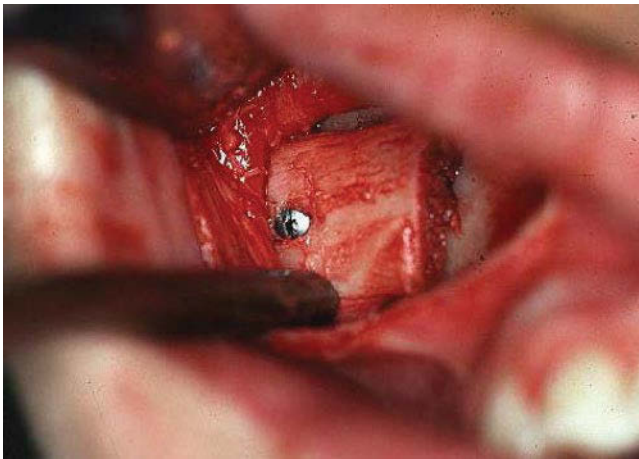


Figure 36.25 A graft across the absent buttress.

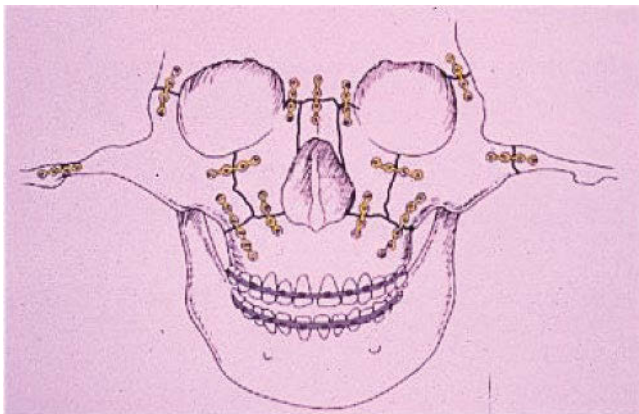


Figure 36.26 An algorithm for fixing the panfacial fracture: i. Stabilize the mandible, ii. Establish the occlusion, iii. fix the outer frame (zygomas), iv. fix the outer frame to the occlusal complex at the buttresses, v. Repair the nasoethmoid complex.

and soft tissues; techniques of grafting and rigid fixation give rise to a high level of predictable skeletal anatomical restoration. The team approach is vital in managing orbital injuries; especially important is the relationship between the craniofacial surgeon and the ophthalmologist.

Anatomy

The orbit is composed of seven bones and shaped like a quadrilateral pyramid; it has a strong anterior rim and four thin walls that converge on a strong bony apex (Figure 36.31). The thick protective outer rim is formed by the frontal, zygomatic and maxillary bones. At the rim the orbit is ovoid in coronal section, becoming more rectangular towards the apex which is almost triangular. The roof, medial, lateral, and inferior (floor) are thin and respond to trauma each in their own way. The portion of the orbit in front of the transverse axis of the globe has little effect on the orbital volume, and hence the projection of the globe of the eye, but more influence on its vertical and transverse position. Alterations to the size and shape of the part of the orbit behind the axis of the globe determine the meaningful skeletal volume and therefore influence the projection of the globe.

Lang, citing Oehmann,²⁹ gives the mean lengths of the adult orbit as: roof: 50.5 mm, floor: 48.4 mm, lateral wall: 47.2 mm, medial wall 40.5 mm.

The eye is protected from blunt trauma by the orbital rim, this being least effective in the inferolateral area. The optic canal enters the orbit at the apex of the pyramid. The canal is on average 9.8 mm long and 4.6 mm wide at its narrowest point. The optic nerve may be injured within the canal, either by forces transmitted through the intact bony canal or the orbital contents, or when a fracture enters the canal. The nerve is further protected from impact by the orbital fat and from stretching by its tortuosity.

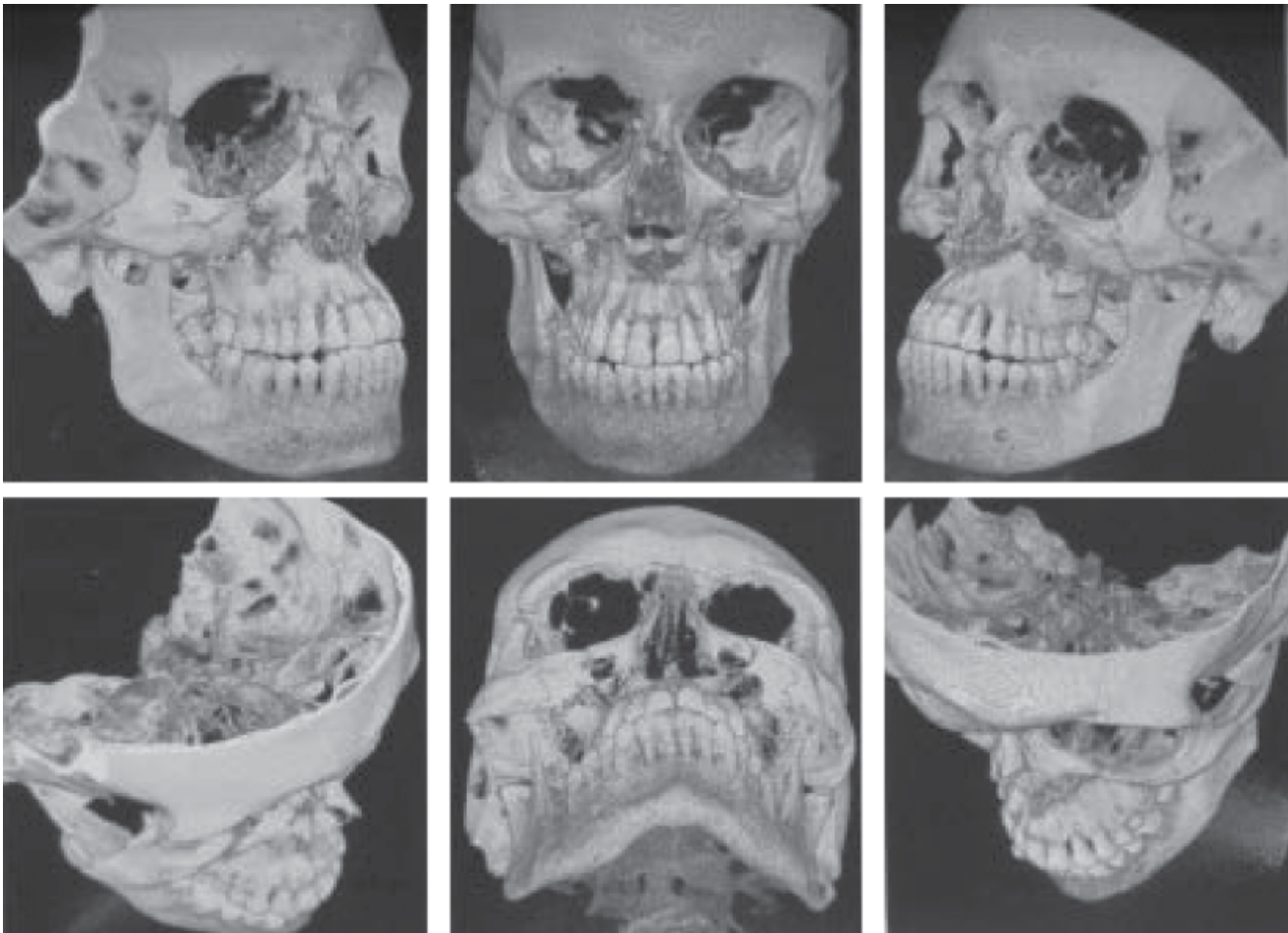
Surgical pathology and pathogenesis

Disruption of the orbit may result from fracture of the orbital rim and walls by extension of extrinsic fractures, that is those arising external to the orbit which are classified according to their major anatomical component (Table 36.1). Intrinsic fractures arise from indirect forces transmitted via the globe and periorbital soft tissues, which produce blow-out fractures of the orbital floor and medial orbital wall (Figure 36.32).

The *orbital roof* is formed by the frontal bone which is thick at the rim and the roof is arched in both the anteroposterior and transverse dimensions. It is relatively resistant to fracturing. Inferior displacement of the roof is the most frequent fracture in this area and in most cases is associated with disruption of the orbital rim. Roof fractures resulting from high-impact injuries to the frontal bone and extensions into the face are more frequently seen in children (Figure 36.33).

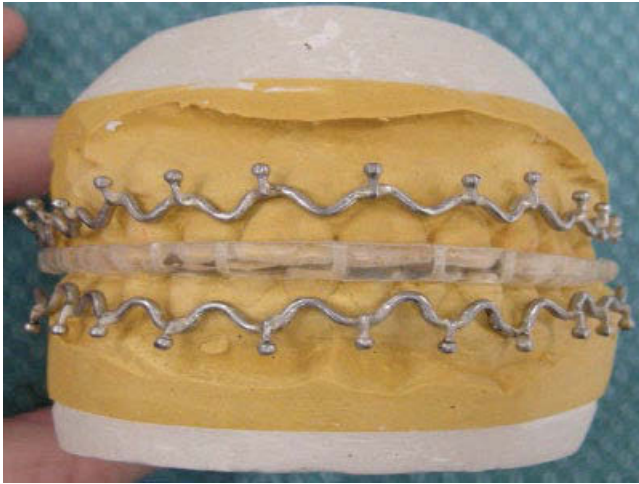


(a)

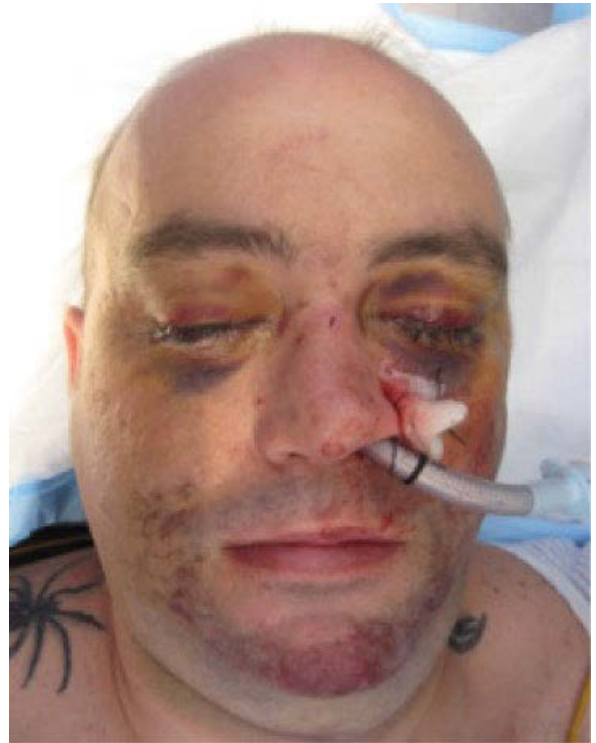


(b)

Figure 36.27 A case study of the sequence of events in repairing this panfacial fracture. (a) Knowledge of the patients premorbid appearance. (b) Detailed study of the fracture pattern. (c) Planned occlusal restoration with a wafer and preformed arch bars. (d) The nasal intubation. (e) The bone graft sites. (f) The coronal scalp flap. (g) Exposure of the zygomatic arch. (h) Plating of the frontozygomatic suture and arch. (i) The transconjunctival incision to expose the orbit. (j) The occlusion restored using the wafer, and intermaxillary fixation. (k) The buttresses plated. (l) The nasoethmoid region reconstructed and the dorsum of the nose grafted with a split costochondral graft. (m) Seven days post surgery. (n) Postoperative 3D CT.



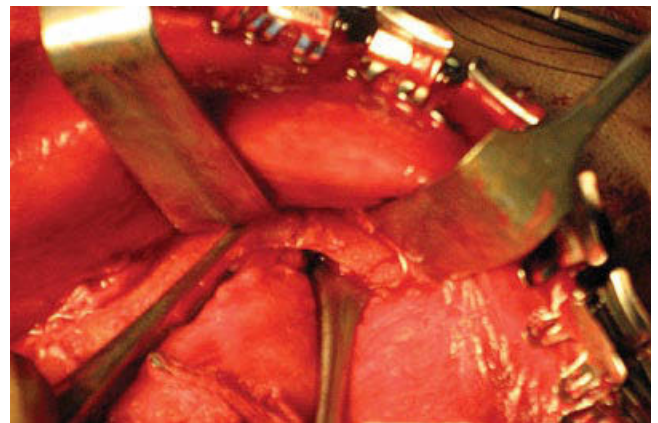
(c)



(d)



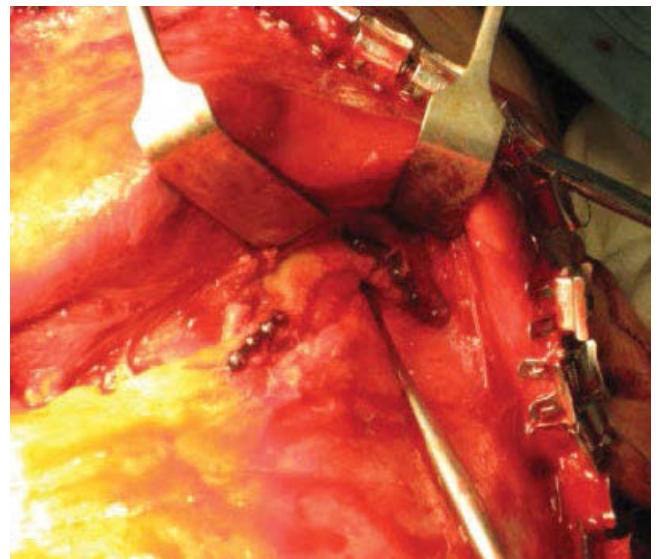
(e)



(g)



(f)



(h)

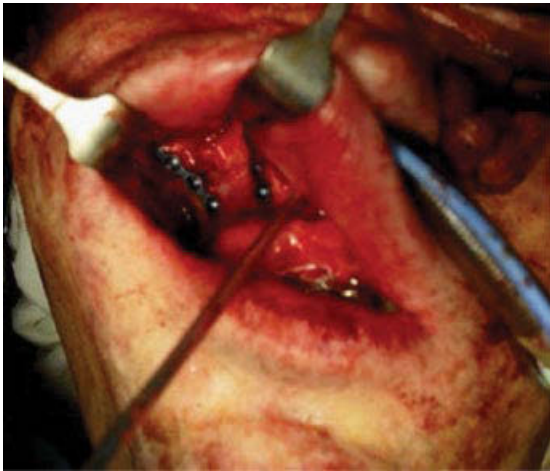
Figure 36.27 (Continued)



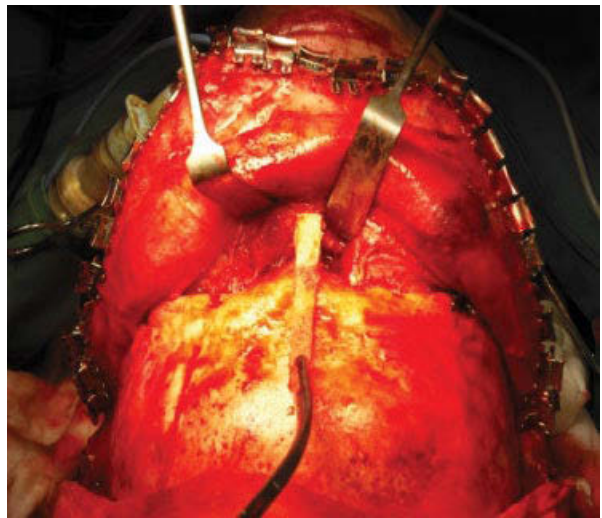
(i)



(j)



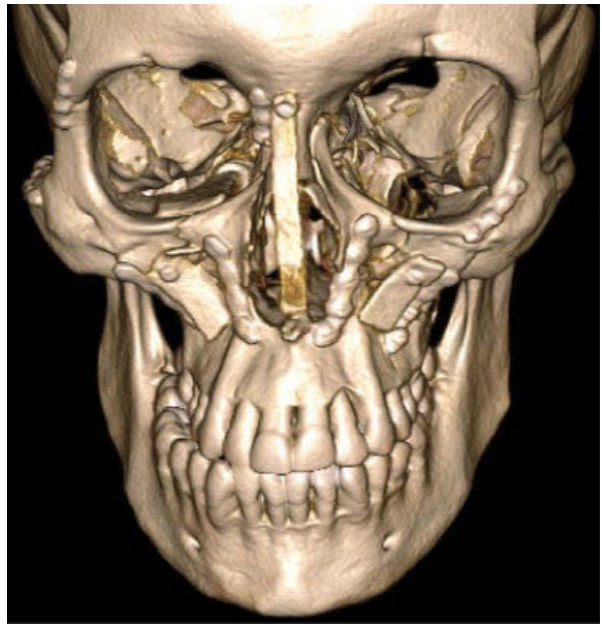
(k)



(l)



(m)



(n)

Figure 36.27 (Continued)

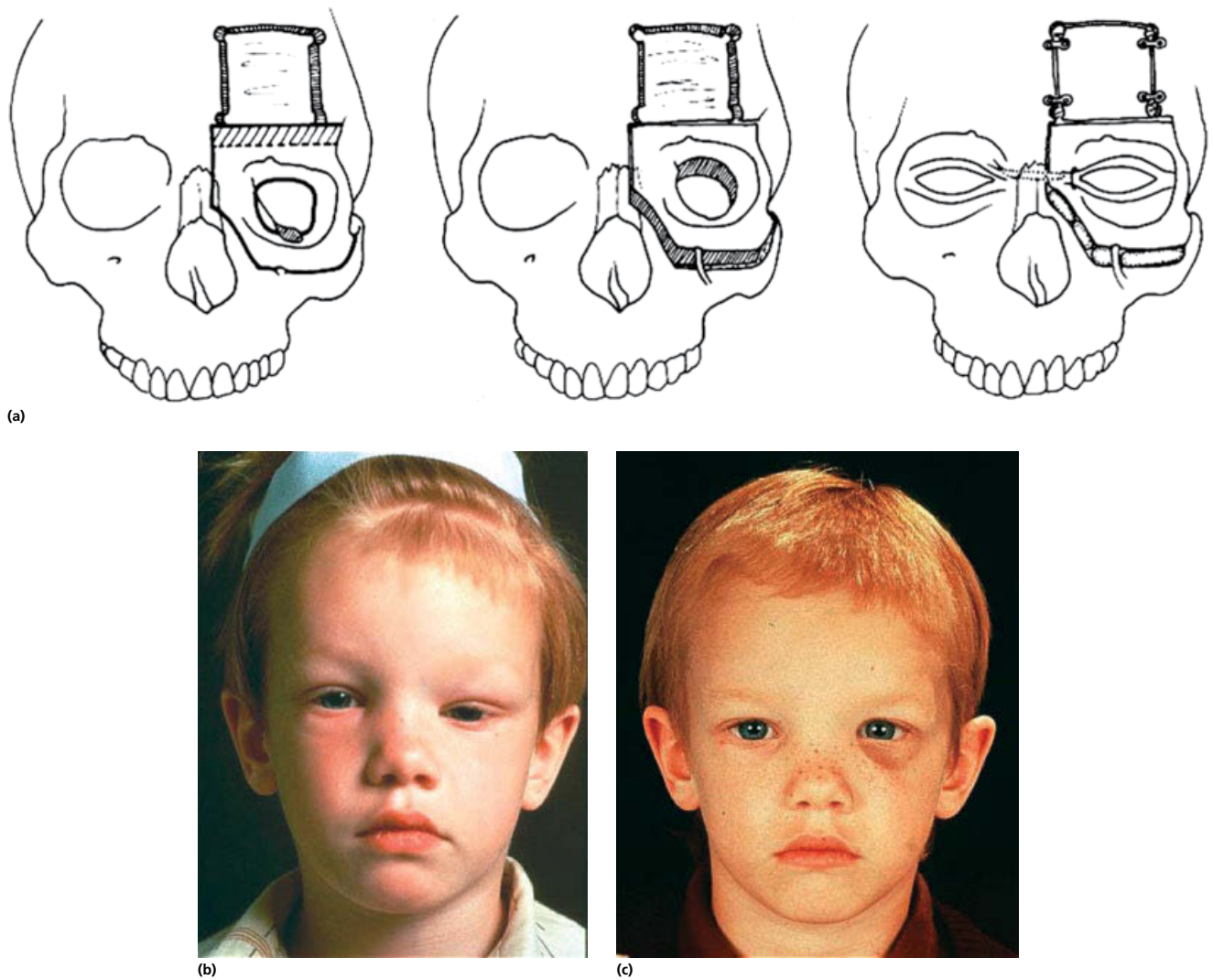


Figure 36.28 Old orbital trauma requiring a box osteotomy to correct the vertical dystopia.

The *orbital floor* is the site of the classic 'blow-out' fracture. The anterior part of the orbital floor medial to the infraorbital nerve canal is concave from front to back, posterior to the axis of the globe it becomes convex upwards because the maxillary antrum bulges superiorly and obliterates the angle between the floor and the medial wall. When reconstructing the floor it is important to restore this posterior convexity in order to prevent enophthalmos (Figure 36.34).

The *medial walls* are almost vertical and have a slight three-dimensional wave formation. The lacrimal bone and the lamina papyracea of the ethmoid bone form the thin, vulnerable portion of the medial wall that is subject to 'blow-out' and 'blow-in' fractures. Isolated medial wall defects in the middle and posterior third of the orbit increase the orbital volume and may cause enophthalmos. Until the advent of CT scanning these injuries were difficult to diagnose and often went undetected. The lacrimal drainage system lies at the anterior aspect of the medial orbital wall and is intimately associated with the medial

canthal ligament. Injuries of these structures are most frequently seen with naso-orbito-ethmoid fractures (Figure 36.35).

The *lateral orbital wall* is formed by the greater wing of sphenoid and the orbital process of the zygoma, it lies entirely posterior to the transverse axis of the globe. Isolated fractures are rare; fracturing usually being part of a zygomatic injury (Figure 36.36).

The *orbital apex* is formed by thicker bone which surrounds the inferior and superior orbital fissures and the optic foramen.

The extraocular muscles are separated from the bony walls of the orbit in its anterior half by fat pads. In the posterior orbit they are close to the bony walls and more vulnerable to trauma from fracture of orbital dissection.

Fractures of the orbit may be classified as being intrinsic to the orbital walls and those fractures of the walls that are associated with fractures of the bones that form the orbital rim. Intrinsic fractures of the orbital walls are characterized as blow-out or blow-in fractures. When there is extension of the fracture



Figure 36.29 An uncorrected Le Fort I fracture with malunion and an anterior open bite corrected by an osteotomy. Source: Winn HR. *Youmans Neurological Surgery*, 2011. Reproduced with permission of Elsevier.

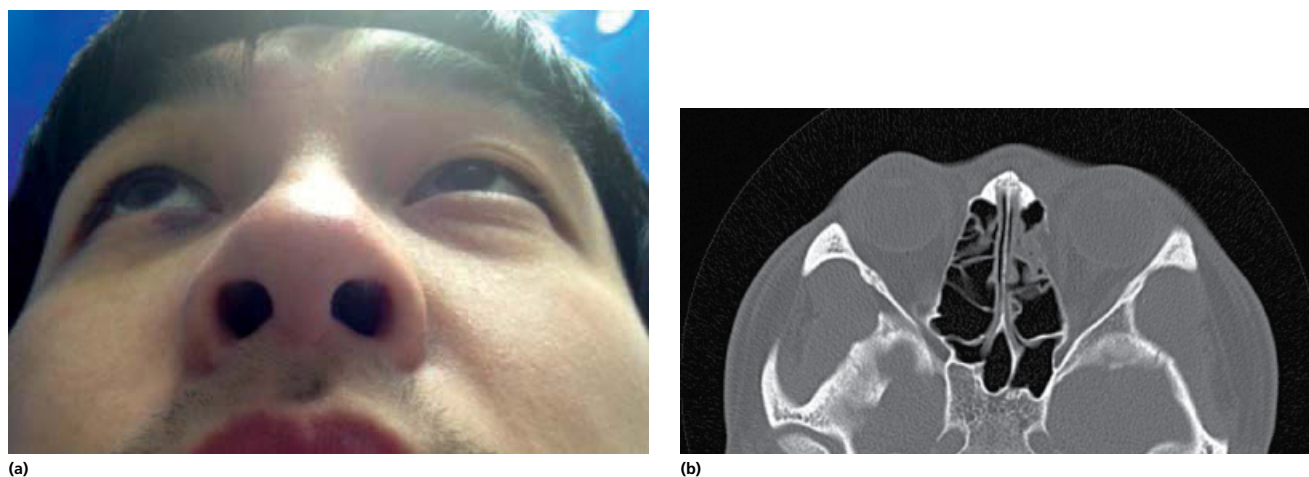


Figure 36.30 A blow-out fracture of the left medial orbital wall produces enophthalmos seen (a) clinically and (b) radiologically.

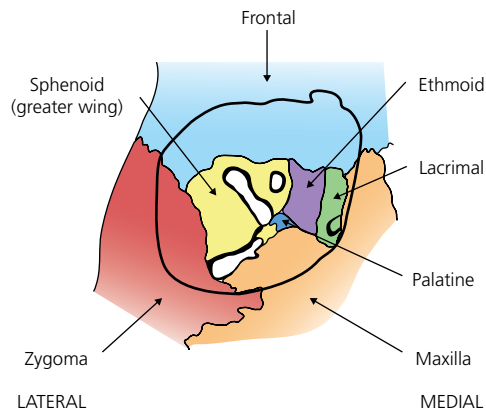


Figure 36.31 Osteology of the orbit showing the contributing bones.

Table 36.1 Classification of orbital fractures

1. Intrinsic – orbital rim intact	(a) Blow-out (b) Blow-in
2. Extrinsic – orbital rim fractured	(a) Blow-out (b) Blow-in

Source: David & Simpson, 1995.² Reproduced with permission of Elsevier.

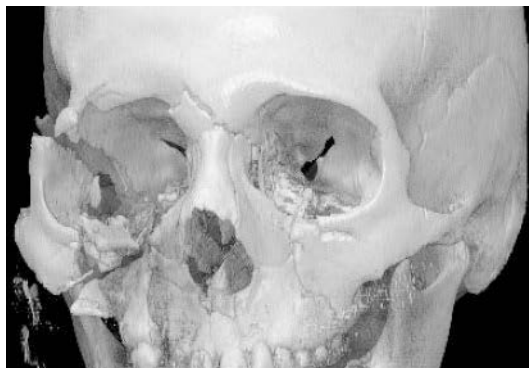


Figure 36.32 Fracture of the zygoma extending into the lateral orbital wall and floor which is so disrupted as to require reconstruction.

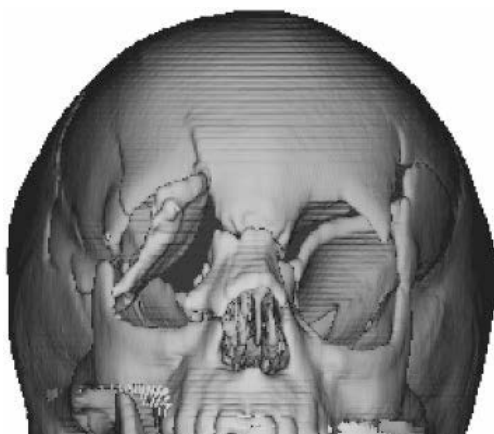
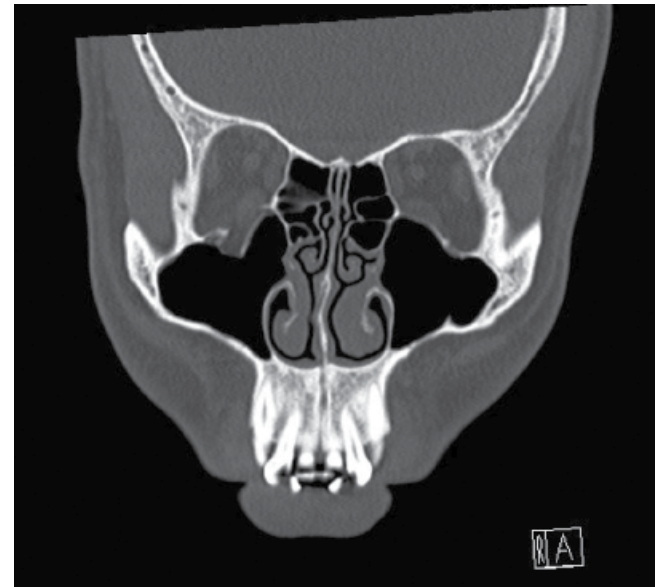


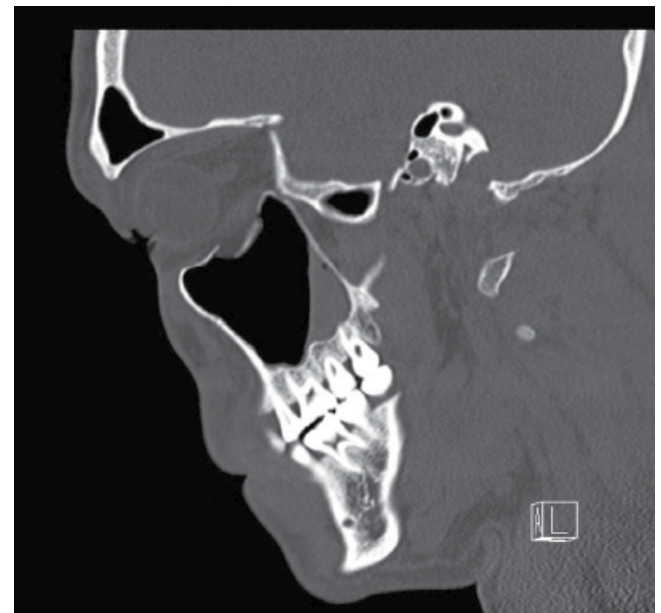
Figure 36.33 High-impact injury in a child producing a blow-in fracture of the orbital roof.

to involve the orbital rim these fractures are often referred to as 'impure'!

The application of sudden blunt trauma to the periorbital soft tissues is transmitted as a hydraulic pressure wave to the orbital walls resulting in the fracture.³⁰ The orbital floor medial to the infraorbital nerve is thin and unsupported and breaks easily, as does the fragile lateral plate of the ethmoid bone. The sequelae of this disturbance include enophthalmos from the expanded volume of the orbital box combined with prolapse of orbital



(a)



(b)

Figure 36.34 A blow-out fracture of the orbital floor. (a) Shows the fragment hinged medially. (b) Shows the same fracture in the sagittal view and also shows the upward bulge of the posterior floor.

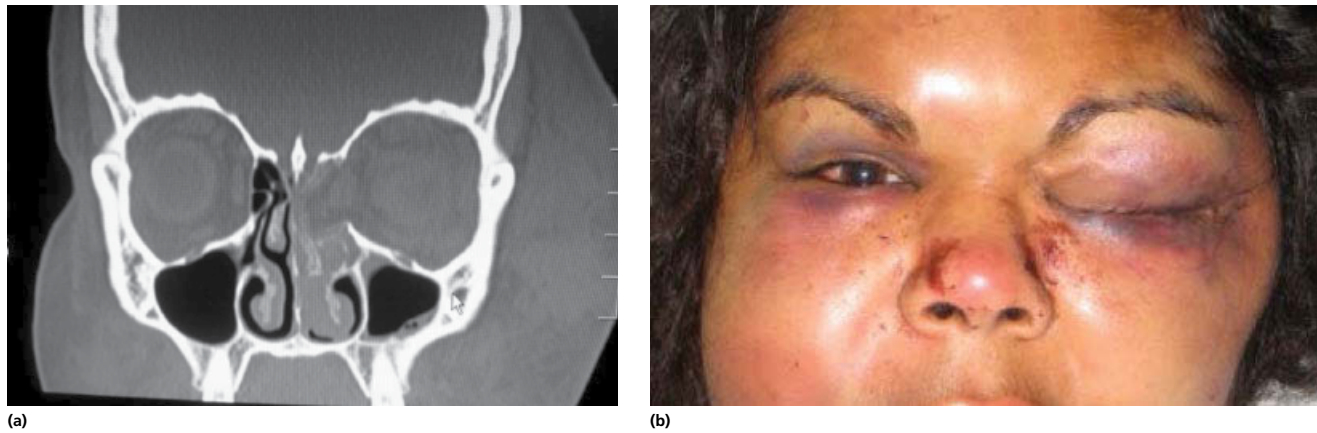


Figure 36.35 A massive medial wall blow-out fracture, the swelling and haematoma hide the inevitable severe enophthalmos.

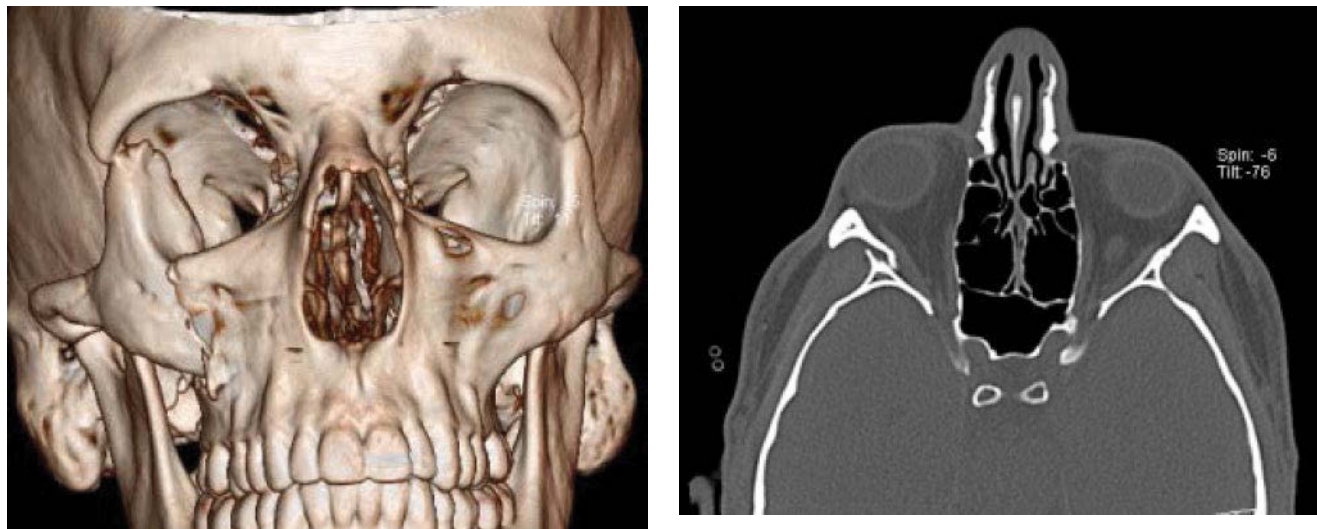


Figure 36.36 The fractured zygoma is displaced posteromedially, overlapping the sphenoid and decreasing the orbital volume.

Figure 36.37 The displaced zygoma decreases the orbital volume and causes proptosis.

contents into the adjacent sinus, possible muscle involvement in the fracture, damage to the infraorbital nerve and diplopia. These fractures usually result from a low-energy impact (e.g. a champagne cork in the eye!). The blow-in fracture³¹ is characterized by inward displacement of bony fragments of the walls and/or margins which results in decreased orbital volume and proptosis (Figure 36.37).

Low-velocity impacts from assaults or falls tend to produce simple linear or blow-out types of fracture involving one or two orbital walls. Medium-velocity injuries have a similar pattern of wall disruption but may be associated with disruption of the orbital rim. The orbital fractures that extend to the orbit from fractures of adjacent bones invariably result from medium- to high-velocity impacts.

The multiple wall fractures resulting from high-velocity impacts that are part of a panfacial fracture exhibit less well-defined patterns and have more comminution and bone loss.

Clinical assessment

Ophthalmological examination is an essential part of the clinical assessment of a patient with orbital trauma, the ophthalmologist being an essential member of the craniofacial team. In addition to the routine examination for fractures such as those of the zygoma, the orbital contents need to be examined by evert the lids with appropriate retractors lest globe destruction be missed. Visual disturbance must be investigated further. The clinician must test for altered sensation in the distribution of the nerves passing through the orbit. Enophthalmos is difficult to determine in the presence of swelling but becomes obvious as the swelling abates. Early marked exophthalmos is indicative of reduction of the orbital volume and requires urgent ophthalmological and radiological examination to determine if there is impingement of bone on the orbital contents or damage at the orbital apex. It is important to test ocular motility. In children, the rare event of a small fracture pinching and entrapping the

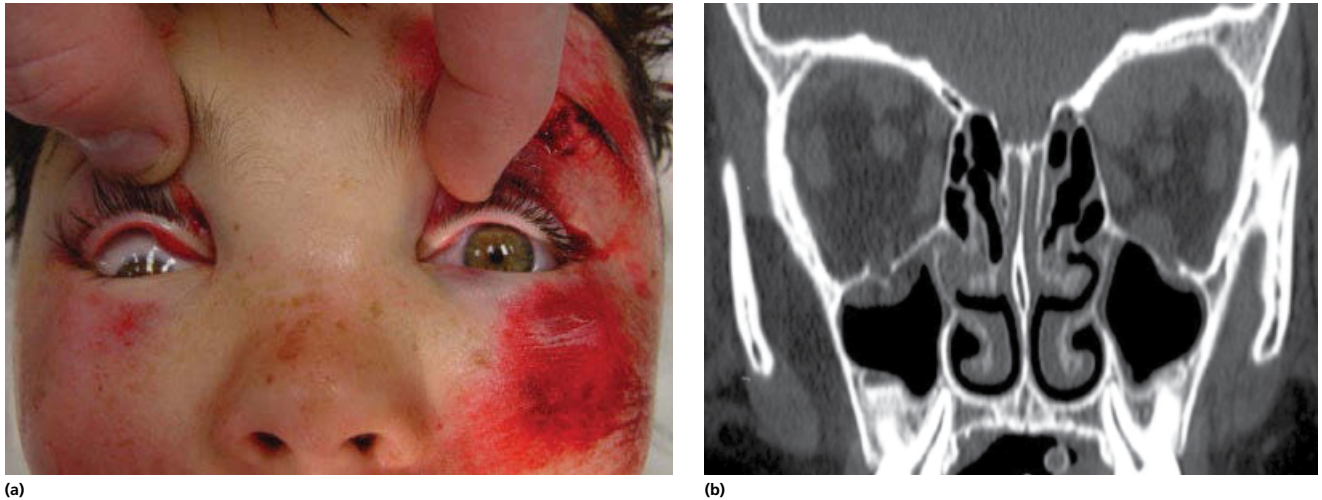


Figure 36.38 The cause of the restriction of upward gaze can be seen in the coronal CT scan: the belly of the inferior rectus muscle is pinched in a small orbital floor fracture. Early release is indicated.

inferior rectus muscle can occur. To avoid damage to the muscle early intervention is recommended. These patients are often very distressed by attempted eye movements (Figure 36.38).

Imaging

CT examination is the diagnostic investigation of choice in orbital trauma, this form of imaging is mandatory for accurate diagnosis and when planning surgical reconstruction.

High-resolution axial CT examination with films of both bone and soft tissue windows show damage to the medial and lateral walls and extensions of fractures beyond the orbit into the adjacent bones. Direct coronal views show the orbital roof, medial orbital wall and orbital floor. When a pure blow-out fracture is suspected, direct coronal scans of the orbit will identify both the extent of the floor and the medial wall involvement and the nature of the soft tissue abnormality. Modern programmes have the tools to define and measure the area of bone destroyed and give a guide to the amount of reconstructive material that may be used at operation. Careful studies will identify the prolapsed fat and the relationship of the orbital muscles to the fracture. Submucosal haematomas or mucosal polyps of the maxillary sinus can be differentiated from herniated soft tissues. Sagittal images viewed in the line of the inferior rectus muscle will show the relationship to the bony damage and also demonstrate the nerves as they pass through the orbital floor.

Comparison with the contralateral orbit provides a qualitative assessment of the degree of enophthalmos. Postoperative CT scanning reveals the results of surgery (Figure 36.39).

Management

As always with craniofacial trauma, the management of orbital fractures is multidisciplinary, the craniofacial surgeon relying heavily on the relationship with the ophthalmologist. Minor

disruptions which do not exhibit clinical signs or cause symptoms are managed conservatively, but followed for some weeks to ensure that late problems such as developing enophthalmos do not occur.

The aim of surgical treatment is accurate restoration of the orbital rim, followed by reconstruction of the orbital walls. The orbital rim is reduced and fixed with miniplates or microplates. The orbital walls are usually reconstructed with bone graft (the author's preference) or alloplastic material. It is important to perform the operative repair as early as possible to minimize the development of soft tissue fibrotic changes. Early swelling and insufficient multidisciplinary work-up usually prevent immediate intervention, however there are exceptional circumstances such as bony fragments impinging on the globe or optic nerve as can occur in some lateral wall fractures and 'blow-in' fractures.

Surgical exposures to the orbit are described elsewhere. The coronal scalp flap giving the widest access to the medial wall in its middle and posterior third. It also gives good access to the lateral orbital wall, roof and, in the experienced craniofacial surgeon's hands, the orbital floor.

The latter can be approached via a subconjunctival route with or without lateral canthotomy, a subciliary approach, or via a lower lid crease incision. More recently there have been advocates of a medial wall repair via a transcaruncular approach to the medial orbital wall (Figures 36.40 and 36.41).^{32,33}

Proper reconstruction of the orbital 'box' returns the globe to its pre-injury position with full maintenance of ocular mobility. Subperiosteal dissection of the orbital walls delineates the fracture and dissection continues so as to expose the surrounding stable bony margins. The herniated soft tissue must be retrieved from the defect in order to restore intraorbital volume and prevent fibrosis.

If the blow-out fracture of the orbital floor is small and isolated, it may be possible to elevate and manipulate the fragment to override the solid margins of the defect. For most defects this

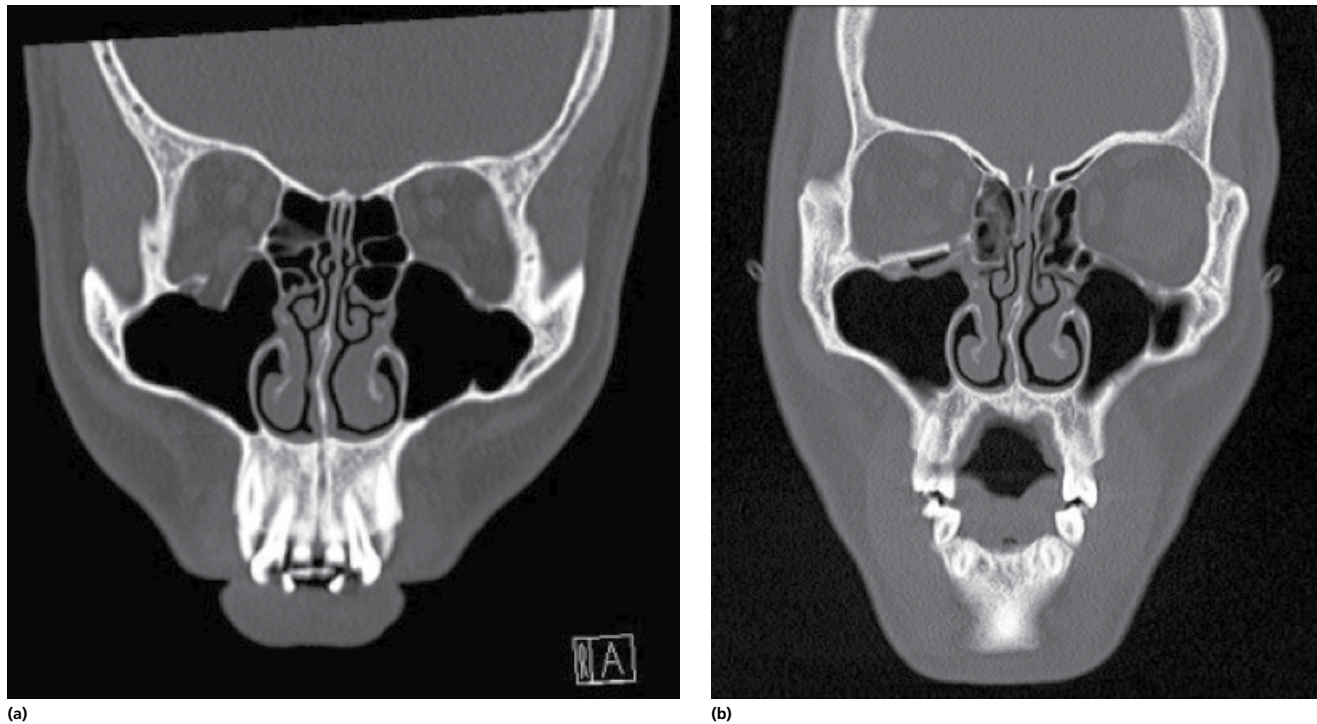


Figure 36.39 Before and after coronal CT showing the repair of a severe blow-out fracture of the orbital floor with a bone graft taken from the blade of the ilium.

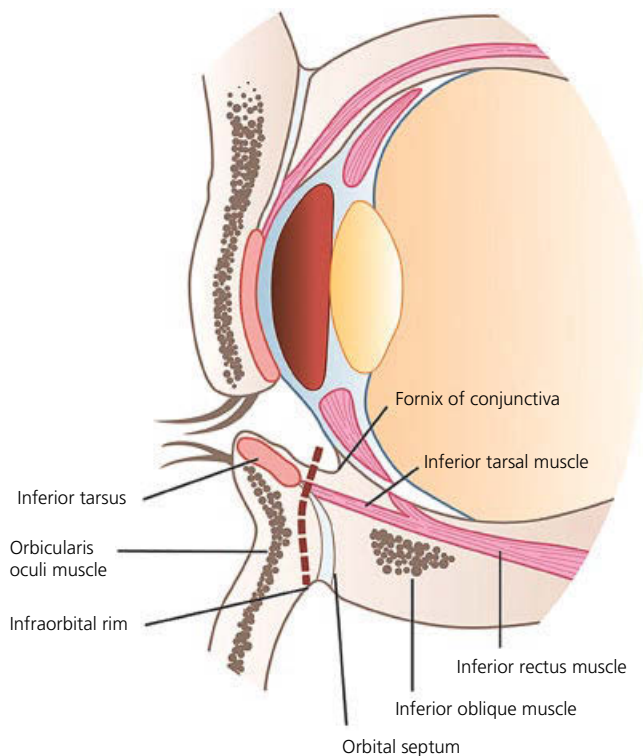


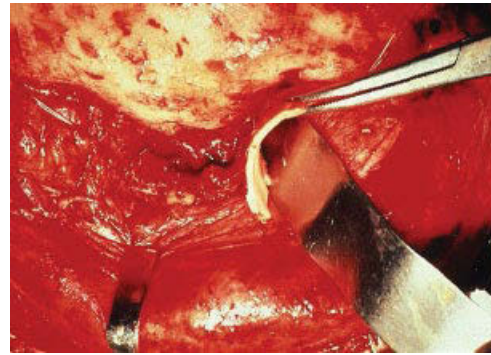
Figure 36.40 A sagittal illustration of the transconjunctival approach to the orbital floor. The approach to the orbital floor begins inferior to the tarsal plate of the lower eyelid. The incision is made in front of the orbital septum down to the maxilla just inferior to the orbital rim. The periosteum is then elevated over the rim to expose the floor.

is inadequate, and the introduction of an orbital wall graft is required.

The choice of materials for orbital wall reconstruction lies between autogenous bone or cartilage and various forms of alloplastic implants. Historically there are many materials used; at one time silastic sheeting was popular, but its tendency to extrude has led to other materials being preferred. Titanium mesh is well tolerated, but with the ingrowth of fibrous tissue it makes reoperation difficult and it is hard to remove. Porous polyethylene (MEDPORE) has become popular for reconstruction. It does not show on postoperative radiology. The author believes that autogenous bone remains the best material for orbital repair. It has minimal tendency to extrude or become infected and it readily remoulds and shapes to the normal orbital contour. The disadvantage is donor site morbidity, which becomes minimal with operator experience.

Calvarial bone enjoyed widespread popularity and is best used when a craniotomy is part of the treatment regimen, and it can be harvested from the inner table of the calvarial bone flap. Splitting off a layer of the outer table risks damage to the brain or at least an unsightly contour defect of the skull. The bone thus harvested is often rigid and difficult to shape.

Split rib graft is available in large quantities; however the author's preferred option is the smooth inner surface of the blade of the ileum approached via an iliac crest incision which can be minimal in length. The quantity and quality of the bone is most suitable for the task of orbital wall reconstruction (Figure 36.42).



(a)



(b)



(c)

Figure 36.41 (a) Bone being inserted into the medial orbital wall via a coronal flap. (b) A medial wall blow-out fracture. (c) Postoperative scan showing the bone graft *in situ*.



(a)



(b)

Figure 36.42 Bone harvested from the inner side of the ileum is smooth, and can be easily contoured for orbital reconstruction.

In uncomplicated blow-out fractures, after anatomical restoration of the orbital bony anatomy with or without a graft, the orbital soft tissues are allowed to redrape.

A forced duction test is performed by grasping the rectus muscle insertions with forceps and moving the globe to demonstrate that muscle entrapment has been relieved. The lid and conjunctival incisions can be closed in a single layer.

In the case of a very extensive defect when the posterior margins may be difficult to reach and define, Tessier suggested removal of the orbital rim, placing microplates on the fragment,

thus obtaining a much better exposure. Once the repair is completed the plated rim is replaced (Figure 36.43).

Complications

Complications of orbital fractures are still seen in spite of the use of modern treatment modalities. Residual problems may stem from the initial injurious process, from incomplete surgical restoration, or from the operative exposure itself.³⁴

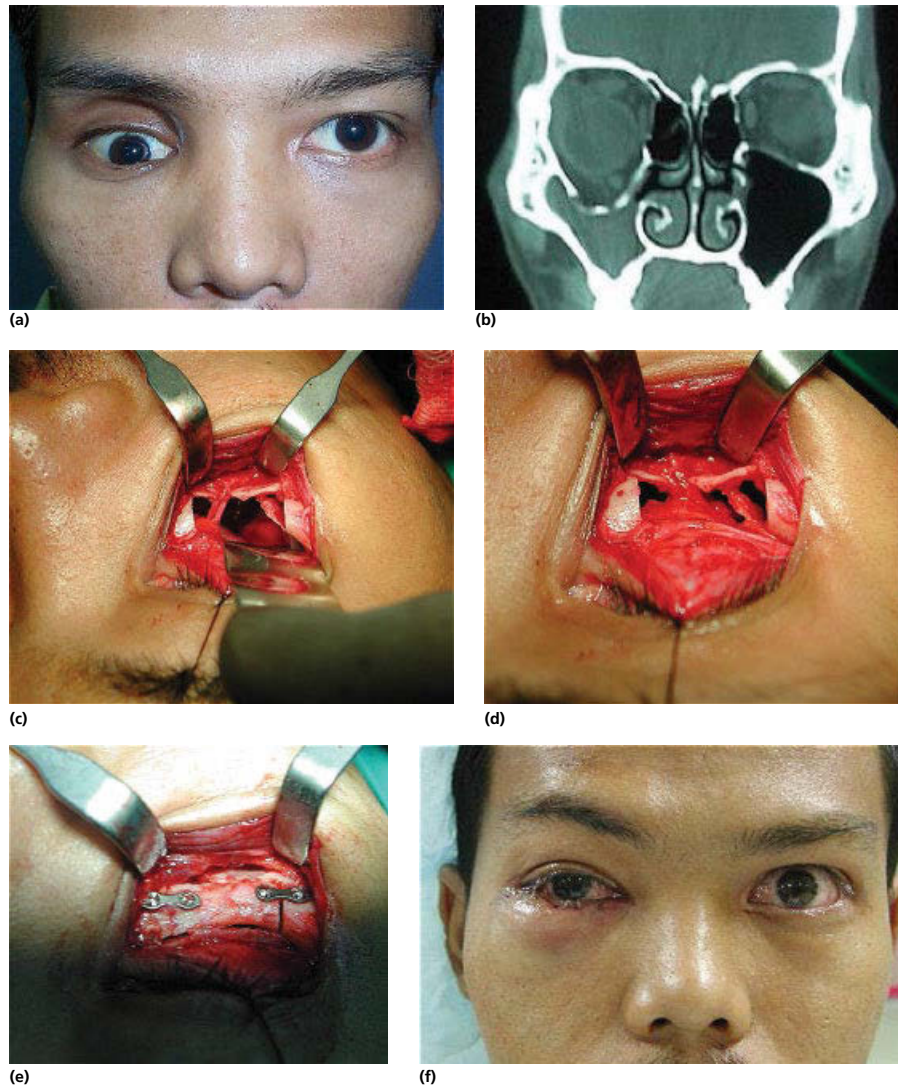


Figure 36.43 (a, b) Patient with a massive orbital floor fracture and global dystopia. (c, d) Removal of a segment of orbital rim exposes the orbital floor which is reconstructed with bone graft. (e) The rim replaced and fixed with microplates (after Tessier). (f) Postoperative correction. Source: Winn HR. *Youmans Neurological Surgery*, 2011. Reproduced with permission of Elsevier.

Ocular problems usually relate to the initial injury and are not affected by the bony reconstruction. Persistent diplopia in the initial postoperative period is not uncommon but is seldom noted in long-term follow-up. Careful ophthalmological assessment will detect any muscle weakness or restriction of gaze and some of these patients may need ocular muscle surgery.

With significant bony displacement at the orbital apex and superior orbital fissure, optic nerve damage and/or ophthalmoplegia can occur.

Enophthalmos is the commonest complication; this has become less since the recognition of and the ability to treat medial wall fractures. It remains hard for the treating surgeon to judge the volume of orbital contents lost or damaged by the trauma and hence if and by how much to overcorrect the orbital wall defect.

Global dystopia may occur with poorly reduced fractures of the bones constituting the orbit, as well as under- or overcorrection of the walls. In a very few cases this may need secondary orbital osteotomy to correct the deformity.

Canthal dystopia occurs medially when the ligament has been disrupted and not correctly reattached, and laterally, most commonly when a lateral canthotomy has been performed and inaccurately repaired.^{35,36}

Mandible fractures

Fractures of the mandible are common, in a recent report from a major Australian trauma centre these fractures were the most common requiring surgery, although orbital fractures, many of

which did not require surgery, were the most common in the series.³⁷ The incidence of mandibular fracture is higher in males than in females with a peak incidence in the third decade of life. Interpersonal violence accounts for the majority of fractures in males, whereas falls account for the majority of fractures in females.³⁸ With the ageing population in Western society, the incidence of mandibular fractures in the elderly resulting from falls is increasing.

The mandible is a very strong bone, designed to withstand forces applied in mastication, however it has areas of weakness that include the condylar processes, the angle and the anterior body in relation to the deep root of the canine tooth. The fact that it has an articulation with the base of the skull at the temporomandibular joint complicates the management of fractures of the mandible. The joint's integrity needs to be restored and its position and function respected in the management of fractures of this bone.

Anatomy

This robust bone occupies a prominent exposed position in the facial skeleton. It articulates with the cranial base through its condyles which lie in the glenoid fossae. The joint is divided into two cavities by a disc of fibrocartilage. The joint acts as a hinge but also allows a sliding movement in the anteroposterior plane.

The mandible comprises a number of components: the condyles, the coronoid processes, the rami, the angles and the two halves of the body joined arterially at the symphysis. Viewed from the lateral aspect, the ramus forms an angle of about 120° with the body of the bone in the adult, slightly more than the very young and elderly. When viewed from the superior aspect there is a slight divergence of the rami. The alveolar portion of the bone supporting the molar teeth does not follow the same line as that formed by the lower border of the mandible.

Functionally, the mandible can be regarded as two conjoined L-shaped cantilevers, acting under the influence of the masticatory muscles. The temporalis muscle inserts predominantly into the medial surface of the coronoid processes, the masseter into the lateral surface of the angle of the ramus. The medial pterygoid muscle inserts on the medial surface of the angle and lower ramus, together with the masseter, it forms a powerful sling acting on the ramus. The two heads of the lateral pterygoid muscles unite to insert into the anterior part of the capsule of the temporomandibular joint and the pterygoid plate. The action of this muscle is important in functional recovery after intracapsule fractures of the condyle and its insertion must be preserved.

In cross-section, the body of the mandible is seen as being composed of a strong inner and outer cortical plates of bone between which are inserted the teeth and their roots which are imbedded in cancellous bone. Between the two plates of cortical bone runs the inferior dental canal which contains the inferior dental nerve, entering the bone at the mandibular foramen and exiting at the mental foramen.

Classifications

Mandibular fractures may be classified simply on the anatomical site of the fracture (Figure 36.44). They may be linear or comminuted, and the fragment size may be displaced or undisplaced. The distinction between compound and non-compound mandibular fractures is important, as any displaced fracture through a tooth root is inevitably compound and fractures of the ramus and condylar region are not compound unless caused by an entering agent, such as a gunshot. The presence or absence of teeth on either side of the fracture line is also important from the viewpoint of treatment by intermaxillary fixation or interfragment fixation, and this justifies the classification (Figure 36.45).³⁹

- Class I – teeth on either side of the fracture
- Class II – teeth on the one side only
- Class III – edentulous.

Clinical assessment

The clinical assessment is based on a thorough history and complete examination, the history being very important because many cases of fractured mandible are due to assault and vehicle accidents which may involve subsequent litigation. The history should establish whether dentures were worn at the time of the accident and whether there had been any previous disease. The fractured mandible may be a part of multiple traumas and resuscitation is the first priority; never the less it is important even in cases with life-threatening injuries to make a thorough preliminary assessment by palpation of the facial skeleton and examination of the oral cavity including the dentition.

Where possible, get the patient to point out the areas of pain or tenderness and ask the patient to feel the teeth with the tongue tip to detect dental irregularities, chipping and loss of dental restoration. The patient is asked to map out any areas of numbness on the face and an accurate record of bruising is made.

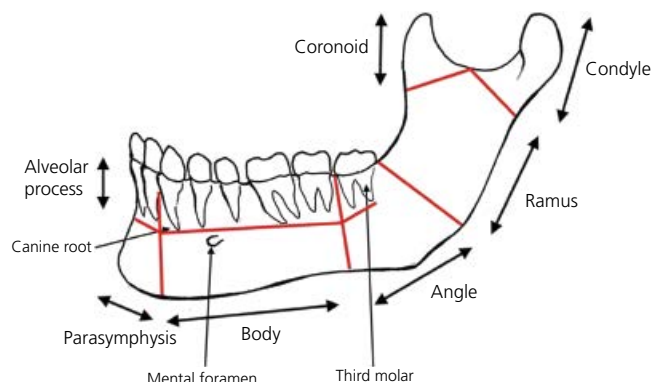
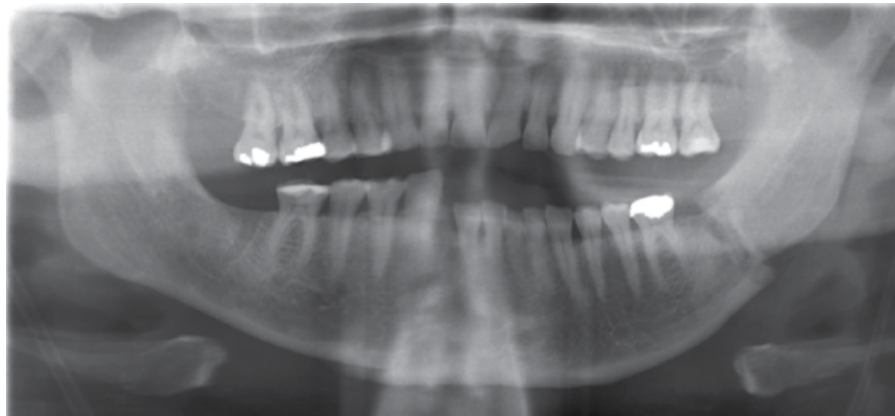
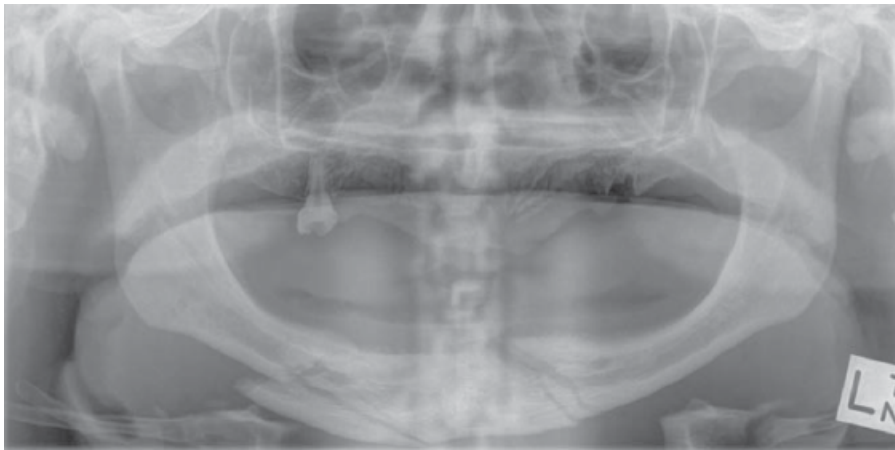


Figure 36.44 Common fracture sites of the mandible. The commonly used descriptive terms for the anatomy of the mandible.



(a)



(b)

Figure 36.45 (a) A parasymphiseal fracture with teeth on both fragments: Class I. A left angle fracture with a tooth on one fragment: Class II. (b) Fractures in an edentulous mandible: Class III.

External and internal lacerations may give a clue to the mechanism of fracture and external chin lacerations may point to a condylar fracture. Steps in the dental arch are noted as they indicate a fracture, as may local swelling and bruising of the gingival tissues or lacerations of the oral mucosa. When the patient is asked to bite the teeth together, they can determine whether the occlusion feels normal and the nature of the occlusion is noted. If the patient is edentulous, the dentures are inspected for signs of injury.

The assessor then proceeds to palpate the mandible through the overlying soft tissues from behind and from in front and notes tenderness and deformity. Compression of the angles will usually cause pain in an otherwise occult fracture. Examination of the temporomandibular joint for pain, swelling, loss of mobility and abnormal movement or crepitus is important.¹⁰

Neurological examination may show sensory loss in the lower lip from damage to the inferior dental nerve.

Imaging

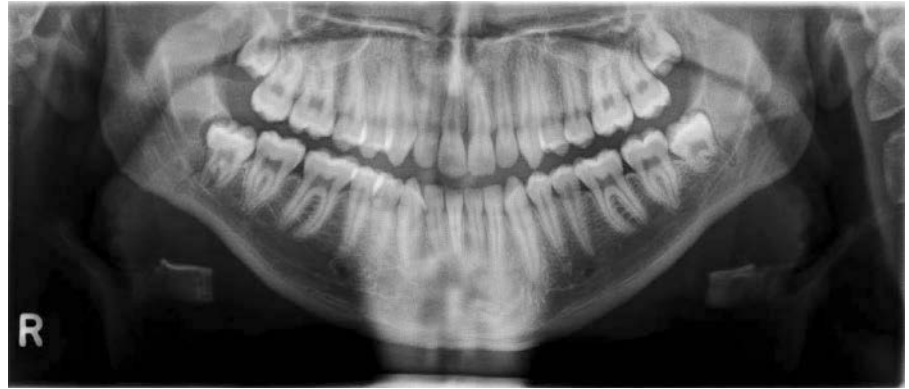
Radiological assessment classically involved the OPG and the Towne's anteroposterior view. The OPG allowed inspection of the entire mandible and mandibular dentition on a single film,

being a composite of the two lateral views. However, the tomographic process results in blurring in the symphyseal region and fractures here may be missed. Moreover, the patient needs to sit and this may be impossible in severely injured or uncooperative patients. This technique remains the mainstay of assessment and follow-up radiology for mandibular fractures. CT images give the most useful information on the integrity of the buccal and lingual plates and modern CT scanning can also give good views of the temporomandibular joints when they are involved in the fracture pathology (Figure 36.46).

Early management

Patients with compound mandibular fractures need to be admitted to hospital, treated with intravenous antibiotics, given appropriate pain management and prescribed an adequate fluid intake and instructed in maintain oral hygiene. In dentate patients with a displaced fracture, dental impressions are taken to provide a plaster cast of the occlusion in the position resulting from injury; these casts are then cut at the fracture site and mounted on an articulator in a predicted pretraumatic position so that they can be used as models in planning how to restore

Figure 36.46 An orthopantomogram showing a fracture of the right angle of mandible involving the partially erupted wisdom tooth. Note the clarity of the image laterally compared with the relative lack of definition at the symphysis.



the pretraumatic occlusion as accurately as possible. From this structure an acrylic wafer can be made for use in the surgical reduction and fixation of the fracture.

Timing of definitive treatment

Often the clinical priorities of other injuries have to be taken into account. Soft tissue lacerations should be debrided and sutured within 12 h of the injury. Some authorities favour early reduction and immobilization of compound fractures of the mandible.⁴⁰ Others⁴¹ believe that the best results occur if operations are done with good instrumentation with experienced staff after thorough assessment and work-up and this may extend the period of time between trauma and definitive treatment if an increased infection rate was not noticed under these circumstances.

Conservative management

Stable fractures without displacement are best treated conservatively by a liquid non-chewed diet and analgesics. Sometimes a soft padded neck collar may be worn to support the chin. This regime can be used for unilateral fractures of the angle and body of the mandible in elderly edentulous patients and in greenstick fractures of children. Some ramus fractures are splinted by the masseter muscle laterally and the medial pterygoid muscle medially and lend themselves to conservative management. Undisplaced hairline fractures of the body or angle which extend to a dental root may cause more of a problem with respect to affecting the viability of the tooth, so long-term dental follow-up is mandatory. In a sensible patient who is likely to comply with good follow-up and dental management, conservative treatment would have a good chance of success.

Principals of operative fixation

Historically, mandibular fractures were fixed by external intermaxillary fixation, but this method has faded into antiquity. More recently, closed reduction and intermaxillary fixation,

where the jaws were fixed together in occlusion for a prolonged period of time, has been used, aided in class II fractures by interfragmentary wiring. However, these methods are rarely practised today, or even thought of since the advent of internal osteosynthesis with miniplates. Although there is still much debate about the number, siting and composition of the plates, this mode of treatment is widely preferred and allows early release of intermaxillary fixation, followed by support of the mandible with light elastics which entails minimal time in hospital, ability to feed and facilitation of oral hygiene. It also avoids the risks associated with prolonged intermaxillary fixation and the presence of the devices that are used to secure it.⁴²

When a fracture extends into a dental root, it is arguable as to whether it is necessary to extract the tooth or not in order to lessen the risk of subsequent infection. The consequence of removing such teeth and converting a potentially stable fracture into an unstable fracture is less of an issue with miniplate fixation used as the primary treatment. The author's rational has been to retain teeth in the fracture line where these teeth have potential function, either in establishing the occlusion or as a fixation post for subsequent prosthodontic work, assuming that the teeth have a chance of survival. When there is severe fracturing of the tooth with comminution of the surrounding alveolar bone, infection is almost certain and the tooth should be extracted. When an impacted third molar is involved in an angle fracture it is customary to extract it as the tooth rarely has a function part in the occlusion, and retention of such a tooth may lead to future infection. Rubin *et al.*⁴³ have shown that completely erupted molars may be left in a fracture line without increasing risk of complications.

Miniplate osteosynthesis

Miniplate fixation in the modern context goes back to 1958 when the Association for Osteosynthesis/Association for the Study of Internal Fixation (AO/ASIF) system was introduced and showed the value of rigid mobilization and coaption of bone ends and securing bone union. Initially the plates were made of stainless steel before Hans Luhr developed a system in Würzburg

using vitallium and producing compression with bicortical screws. More recently, titanium plates have been preferred because they are malleable, radiolucent and have high biocompatibility. The search continues for suitable resorbable implants for mandibular fixation.⁴⁴

Success in plating fractures of the mandible depends on an understanding of the mechanical forces acting through the mandible during mastication. The mandible is normally subjected to tension forces of its upper border and to compression forces at the lower border unless the fracture is inherently self-stabilizing. There will therefore be distraction of the upper or alveoli end of the fracture and the plating system must counteract this distraction. Champy *et al.*⁴⁵ described an ideal line for the siting of the plate as shown in Figure 36.47.

They demonstrated the presence of rotational torsion forces in the anterior mandible, which is a three-dimensional structure upon which bilateral muscular groups act. They also recommended a lower border plate as well as an upper border plate to give better control of these torsional forces. In the tooth-bearing alveolar area the thickness of the bone is variable and unsuitable for screw fixation; the upper boarder of the mandible is therefore the ideal site. Champy *et al.* showed that the average thickness of the outer cortex of the mandible is 5 mm but in some areas the cortex is less than 3 mm thick. There is therefore some danger of injuring the inferior dental nerve. Care should therefore be taken to drill only through the outer cortex and use the smallest possible screws.

In certain situations the usual tension/compression relationships can be reversed. This may be the case when a bite load is placed just anterior to an angle fracture and the contralateral muscle sling is functioning normally but the ipsilateral sling is ineffective from post-traumatic oedema and bruising.⁴⁶ In addition to this theoretical concept of intermittent reversal of the normal tension–compression zones, there is also some clinical evidence of unfavourable results with angle fractures

treated with only a single plate as Champy suggests. Levey *et al.*⁴⁷ had a significantly higher complication rate with using one plate rather than two. It has been the author's experience that the Champy principle with a single upper border plate for angle fractures has been more than satisfactory. It has emerged, however, that in non-compliant patients firmer bracket fixation by applying a second plate may be needed.

The Champy technique of upper border single-plate osteosynthesis is mostly successful for angle fractures providing the patient is compliant and does not chew and the reduction is accurate.

Compression plates

Because the mandible is an asymmetrical structure, bicortical screws and compression plates are required. The compression plating systems have a consistent potential drawback. Because the compression is applied across the buccal plate of bone there is the possibility that the fracture line may be opened on the lingual side and this can lead to a significant cross bite. Efforts have been made to contour the plates extremely accurately so that this does not occur. In practice, it does not appear that the healing claimed by compression systems is any more advanced of that than non-compression systems.

Operative management according to Champy

The patient is prepared in the usual fashion using an acrylic wafer to register the occlusion and the application of arch bars or intermaxillary fixation screws. A naso-endotracheal tube is usually necessary. The incision to explore an angle fracture is placed just lateral to the anterior boarder of the ramus and runs

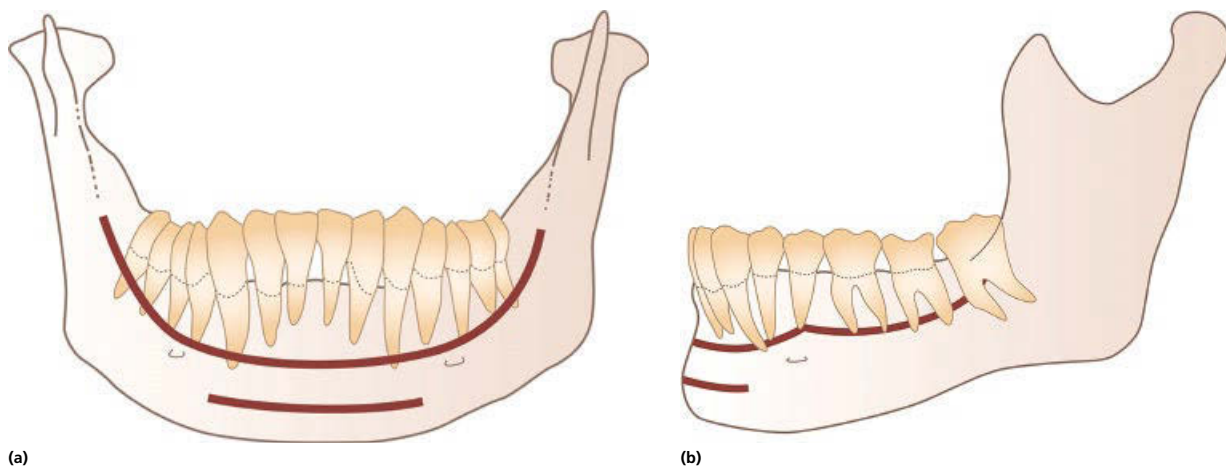


Figure 36.47 (a) The mandibular tension line is at the junction of the dentoalveolar bone and the mandibular base bone (Champy). (b) The anterior body reverse tension line. The anterior lower borderline is the site for lower border plates in conjunction with upper border plates when fractures cross this area (Champy).

down beyond the third molar just lateral to the inferior buccal sulcus. The incision is deep into the bone and the fracture line exposed by subperiosteal dissection down to the inferior border of the mandible. A posterior border retractor combined with a notched anterior border retractor will give good exposure. When the third molar is to be extracted, care is taken not to fracture the external oblique ridge of the bone as this is important for the fixation.

After removal of the tooth, the socket is cleaned and irrigated; care is taken not to injure the inferior dental nerve which is sometimes exposed by the fracture. It is important to spend some time cleaning the edges of the fracture of granulation tissue and irrigating the area thoroughly in order to wash away debris. The fracture is reduced manually into its correct anatomical position. The occlusion is established and intermaxillary fixation is applied. The pretraumatic position of the occlusion is established by the use of the dental wafer. Once the jaw is secured with the intermaxillary wiring and the fracture reduced anatomically, it is relatively easy to apply the miniplate

according to Champy's principles. A four-hole titanium miniplate with two monocortical 5 mm screws on either side of the fracture line is adequate (Figure 36.48).

The incision to explore a fracture of the body is placed to the labial side of the inferior sulcus. In order to preserve the mental nerve, the scalpel is angled back sharply under the sulcus down to the bone, the periosteum is incised and the fracture exposed by subperiosteal dissection in the usual manner. If the fracture is laterally placed, the mental nerve in its foramen must also be exposed and preserved. In both the angle and body fractures, care must be taken to drill only the outer cortex and the plates are bent as accurately as possible to conform to the contours of the mandible. Some plating systems use malleable titanium with no memory and these can be contoured very accurately with the screw pressure.

Sometimes it is warranted to modify the standard method in fractures in the angle area on the external oblique ridge, some operators preferring the six-hole plate and longer screws and where there is some doubt about the reduction of the fracture at the lower



(a)



(b)

Figure 36.48 A left angle and right body fracture in a jaw with carious teeth and teeth involved in both fractures. The monocortical plates have been used according to Champy after removal of the teeth in the fractures.

border of the mandible, some operators prefer to place the second plate in this region, still using an intraoral approach but combined with percutaneous drilling and screwing through a cannula.

With body fractures, it is usual to apply the four-hole plate to the upper border of the body with two monocortical screws on either side of the fracture line and a similar plate applied to the lower border, avoiding the nerve. Intraoral wounds are usually closed with an absorbable suture. The temporary intermaxillary fixation is removed and the excursions of the mandible are checked with special attention to the ability to bite into occlusion. The arch bars or fixation screws are left *in situ* and the jaws are supported by light elastic rubber traction to overcome any imbalance in the masticatory muscles caused by injury and swelling. The elastic traction may be maintained for a number of days, being removed once the patient settles into their normal occlusion.

The postoperative management includes a dose of intravenous antibiotics at the time of surgery, clear fluids, soft diet and mouth toilet. Postoperative radiology is important to check the reduction of the fracture. This regime facilitates early discharge from hospital and encourages self-care. Follow-up at regular intervals including a postoperative dental assessment is important and long-term follow-up is routine for 12 months. If there are clinical reasons, the patient may be seen beyond that in order to check the healing of scars, the recovery of any areas of sensory loss and the return of normal mastication.

Complications of fractures of the body and angle of the mandible

Complications include:

- infection
- non-union
- malocclusion and malunion
- plate exposure
- nerve injury and/or dental injury due to drilling the body of the mandible.

Infection

The instance varies in different reports and the causes are disputed. The argument that early surgery on the mandible prevents infection⁴⁰ has been made; others⁴⁷ reported a high incidence of infection when plates were used. Anderson and Alpert⁴⁸ gave special attention to fractures of the mandibular angle when only one miniplate was used without intermaxillary fixation, giving it an infection rate of 22%. Once infection is diagnosed and a wound culture is taken, intravenous antibiotics are started. If radiology reveals a fracture displacement or bone sequestrum or any complication relating to a plate, the fracture is explored and the foreign and necrotic material removed. Teeth in the fracture line need to be extracted. The occlusion can be re-established and where there is bone contact with the fracture, replating can be undertaken. If there is discontinuity of the bone, a bone graft may be inserted with rigid fixation and adequate drainage.

Non-union

This is clinically indistinguishable from infection and in principle the treatment is the same.

Malocclusion and malunion

Some malocclusion can result from inadequate or inappropriate reduction of the fracture but may also be a consequence of orthodontic movement of teeth which have been wired to arch bars or the result of imbalanced masticatory muscles and the management can be relatively conservative. If the cause of malocclusion is shown by X-ray to be due to inadequate reduction of the fracture, it may have been due to the operative procedure and reoperation is necessary to reduce the fracture again and fix it more strongly in its correct position. If there is true malunion and the bone is united and there is a malocclusion then an osteotomy will be required.

Plate exposure

Miniplate exposure may be due to poor operative technique and suturing mucosa or due to postoperative trauma. It is usually safe to leave the plate exposed until healing is complete in six weeks, in which case the plate can be removed without compromising the result.

Nerve injury

Injury to the inferior dental nerve is usually the result of the trauma but may occur with poor operative technique and radiology will show whether a screw is impinging on the inferior dental nerve canal. Sensory recover or failure of recover to the inferior dental nerve should be watched for over 12–18 months. Some operators would uncover the canal and attempt to anastomose the nerve or indeed graft it.^{49,50}

Fractures and fracture/dislocations of the condyle

Surgical pathology

The condyle is the part of the mandible that passes vertically up from the posterior border above the sigmoid notch to the glenoid fossa of the temporal bone. Fractures may occur at any level in the sigmoid notch, thereby including a variable amount of the ramus in the condylar fragment (Figure 36.49).

Sagittal fractures of the condylar head are rare; however, there may be dislocation of the fractured condyle in a posterior, anterior, medial or lateral position and very rarely vertical dislocation into the cranial fossa.

Attempts at classification

The classification devised by Lund⁵¹ has the merit of being very simple. Type I: enlocated head with or without displacement at the fracture site with no more than 60° angulation. Type II: a dislocated head with 90° or more angulations. Lund also described this fracture as either high, involving the head or neck, or low, at the base of the condylar process (Figure 36.50).

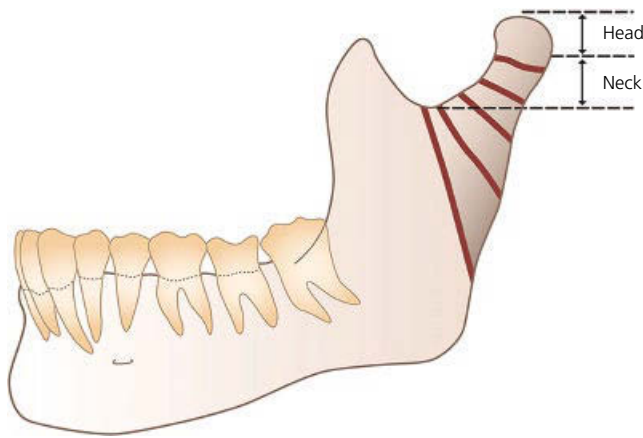


Figure 36.49 Anatomy of condylar fractures. A condylar fracture is one that separates the condylar head from the corpus of the mandible, but excludes the coronoid. These fractures all pass through the sigmoid notch and are deemed high or low.

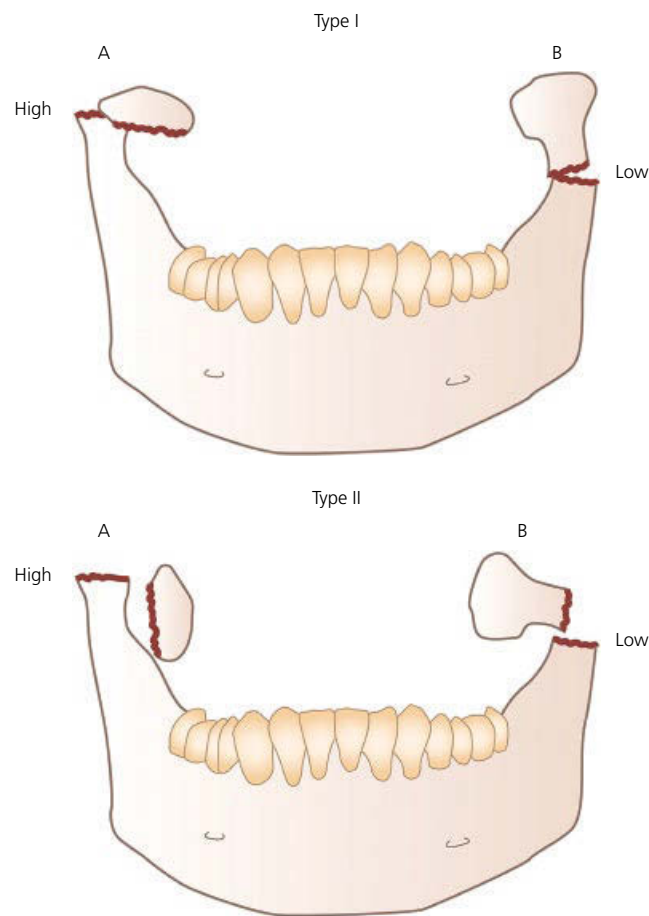


Figure 36.50 Lund's classification of condylar fractures.

Clinical assessment

A proper clinical assessment commences with a thorough history as well as a complete examination, and a history of the causation of the fracture will often point in the direction of the

temporomandibular joint. Pain in the region, together with trismus, is a common combination in these circumstances. Attempting to open the mouth will be painful and the movement restricted and this may be bilateral. When the fracture is unilateral, any attempt at opening of the mouth will cause deviation of the chin point towards the affected side.

Bleeding from the external auditory canal may indicate a compound fracture into the external ear canal and otoscopic examination of the meatus may show a tear in the lining of the canal. Bimanual palpation of the condyles during movement may detect swelling, tenderness and the absence of a normal condylar head. With respect to occlusion, typically bilateral condylar fractures cause an anterior open bite not present before injury, whereas a unilateral fracture causes lateral cross bite. The clinician should always keep in mind the possible presence of multiple facial fractures.

Radiological assessment

The standard OPG and Towne's views have mostly now been replaced by CT scanning, and the modern fine-cut CT scan can be reproduced as a solid image and even converted to nylon models which may, in complicated cases, be of great help in surgical planning (Figure 36.51).

Principles of treatment

The management of these injuries is controversial. In children, there may be restitution of the fractured condylar head and remodelling of the displaced fracture and associated compensatory growth which would support a conservative plan of management in the young. This is not repeated, however, as growth nears completion.⁵² When the fractures are intracapsular and unable to be successfully reduced in an open fashion then the management is conservative, relying on reduction of pain, supporting the occlusion and early mobilization with the application of dental arch bars and elastic traction.

In the mature patient, when there is a fracture that can be fixed by open reduction and internal fixation with plates or other devices, this can be undertaken when the fragment is dislocated or when fixation of other fractures in the face require an intact mandible. Because the surgical approach to the temporomandibular joint is not an easy one and may be associated with complications such as facial nerve damage, there are several criteria that should be fulfilled for operative procedures to be acceptable:⁵³

- The operative procedure must provide full restoration of normal function and occlusion within 6–12 weeks.
- The fracture must be reduced so that the muscles which act on the condyle regain normal tension and position.
- The displaced fragment must be large enough to be reduced and stabilized in three dimensions.
- The stabilization must be able to be maintained until bone union occurs.
- The operation must not damage the attachments of the lateral pterygoid muscle for the condylar head.
- There must be no operative morbidity from injury to the nerves and other important structures or to mandibular growth.

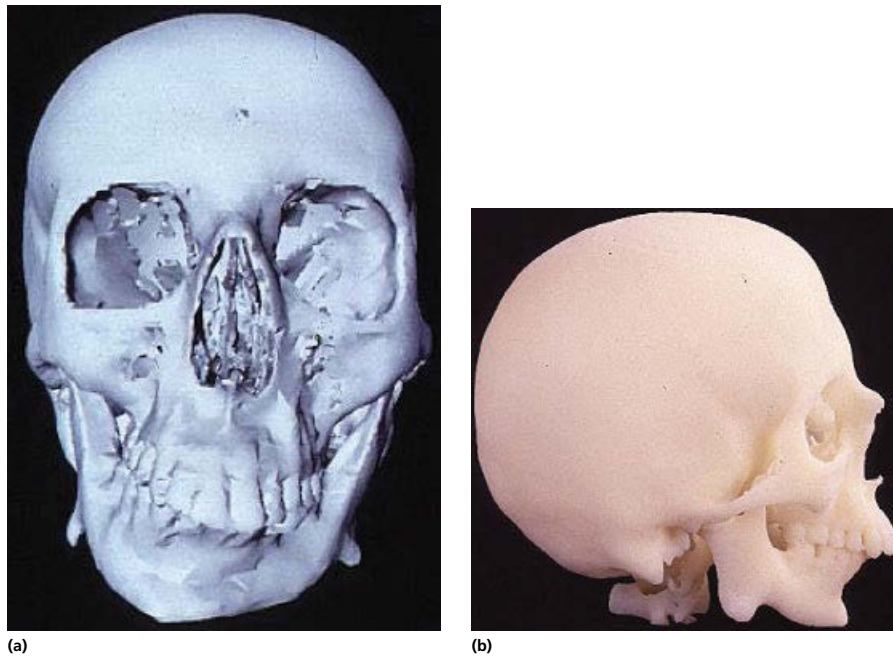


Figure 36.51 (a) A 3D CT image of a patient injured in infancy, there is left temporomandibular joint ankylosis and a typical growth deformity. (b) A nylon model showing the pathology can be useful when planning reconstructive surgery.

Operative management

Zide and Kent⁵³ produced a list of absolute indications for open reduction and internal fixation of fractures of the condyle. Other authors have slight variation on this list but the principals are essentially the same:

- Fracture dislocation of the head into the middle cranial fossa or into the temporal fossa if associated with clinical disability
- Intra-articular foreign body
- Lateral extracapsular dislocation of the condyle
- Inability to open the mouth or to bring the mandible into occlusion after a week with evidence of bony block
- Compound injuries (e.g. gunshot wounds)
- Displaced condylar fracture where the fracture line is near the lower border of the sigmoid notch and the head is dislocated from the glenoid fossa
- Condylar fracture where the vertical displacements associated with displaced fractures of the upper jaw.

The ideal situation is where there is enough bone substance to hold two plates, giving rigid fixation and a capacity for immediate mobilization. The condyle may be approached through an intraoral incision and various skin incisions. Lately, some authors have used endoscopic assistance in this surgery. The author has found the most acceptable approach in his practice is through a coronal scalp flap, but extended in front of the ear to the level of the tragus. It is often unnecessary to make the full coronal incision. Once the zygomatic arch has been exposed, the masseter muscle is exposed and may be transected giving a very good exposure to the condyle, upper part of the mandible and hence the fragments (Figures 36.52 and 36.53).

Brown and Obeid⁵⁴ reviewed the methods of stabilization of the fracture when it has been exposed and reduced. Surgeons have used various types of interosseous wiring, Steinmann pins

with or without wiring and external fixators and miniplates; the latter have the great advantage of giving three-dimensional stabilization while allowing early return to normal function.

Complications of condylar fractures

These include:

- growth disturbance,
- avascular necrosis,
- ankylosis of the temporomandibular junction,
- malunion and other temporomandibular junction dysfunctions.

Growth disturbance

The problem is peculiar to childhood, of course, and may give rise to considerable facial asymmetry. Where the fracture is associated with infection, then severe growth disturbance may occur. This is often seen in developing countries where malnutrition may play a part (Figure 36.54).

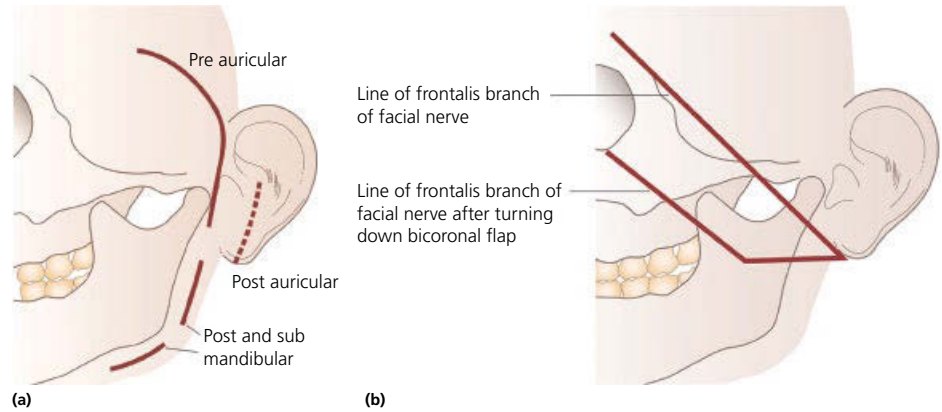
Avascular necrosis

This can be seen when an intracapsular fracture detaches a fragment of the head of the condyle which is denuded of its bloody supply.⁵⁵ It may lead to pain and temporomandibular joint dysfunction. The avoidance of a vascular necrosis is assisted by maintaining all residual capsule attachments of the proximal fragment.

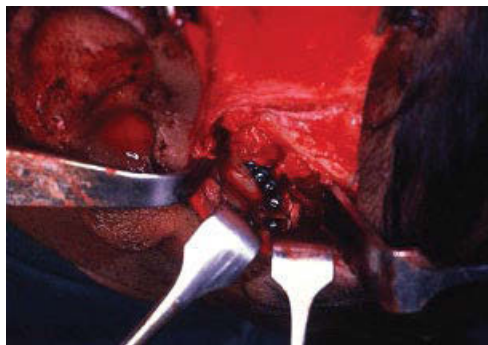
Ankylosis of the temporomandibular joint

This complication may develop when there has been tearing or disruption of the articular disc associated with comminution of the condylar articular surface as an intracapsular fracture. Its occurrence is minimized by early mobilization of the jaw and by careful follow-up. The patients at risk are those who have, for

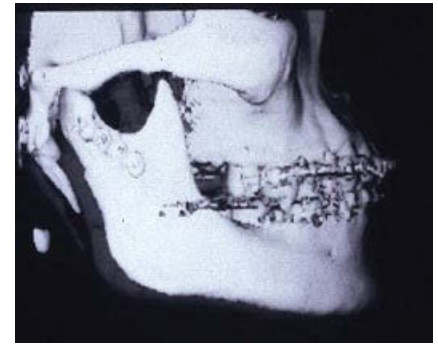
Figure 36.52 External incisions for exposure of the condyle. The coronal flap 'relocates' the frontalis nerve: the act of turning down the flap relocates the nerve to a level below the sigmoid notch.



(a)



(b)



(c)



(d)



(e)

Figure 36.53 (a) A 3D reconstruction of a severe fracture dislocation of the right mandibular condyle. (b) The fracture has been exposed via a coronal scalp flap reduced and plated. (c) The postoperative position is shown, with a single plate. (d) A similar severe fracture/dislocation on the left. (e) Fixed with two plates which is the ideal.

other reasons, long period of intermaxillary fixation or where they have disturbed consciousness from head injuries and the problem has not been one of the first priorities in management. The long-term management of such problems can be extremely difficult.

Malunion and other temporomandibular joint dysfunctions

Standard osteotomies may restore any occlusal discrepancy due to the malunion; however the temporomandibular joint dysfunctions that produce pain and trismus may in some

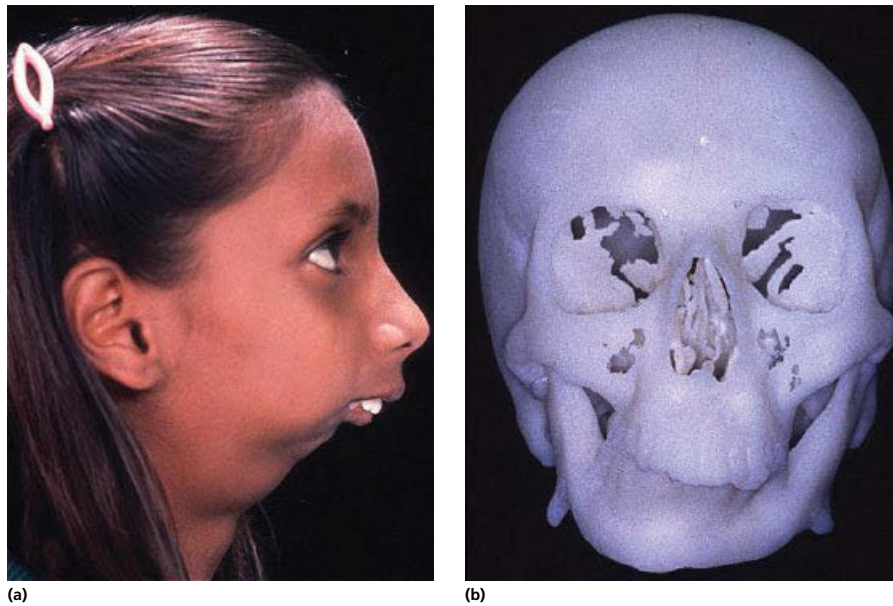


Figure 36.54 (a) The result of bilateral intracapsular fractures of the mandibular condyles in childhood. The severe growth disturbance is accompanied by ankylosis of the joints. (b) A nylon model of a patient at maturity who suffered a high condylar fracture without treatment. Note the facial asymmetry, malocclusion and fused joint.

patients result in crippling and chronic disruptions to their lives in spite of intensive physiotherapy and long-term pain relief.

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PART V

Breast

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CHAPTER 37

Anatomy and physiology of the breast

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The mammary gland is an organ of ectodermal origin, typical of mammals, whose structure reflects its special function: the production of milk for lactation. In human beings the breast has an undoubted aesthetic value, having been the inspiration of many kinds of work of art during the centuries. It also has an erotic relevance, the breast being associated in many cultures with sexuality and sensuality.

The size and shape of women's breasts vary considerably. Some women have a large amount of breast tissue and have larger breasts. Others have a smaller amount of tissue with little breast fat. A woman's breasts are rarely the same size; usually one breast is slightly larger or smaller, higher or lower or of different shape to the other.

The inner structure of the mammary gland is made of epithelial components that consist of lobules, where milk is made, which connect to ducts that lead out to the nipple. These lobules and ducts are located and spread throughout the background fibrous tissue and adipose tissue that form the main mass of the breast.

The structure of the male breast is almost identical to that of the female breast, except that the male breast tissue lacks the specialized lobules because there is no physiologic need for milk production.

Development of the breast (embryology)

Human breast tissue begins to develop at approximately the 5th–6th week of fetal life. It proceeds from a single ectodermal bud and starts developing along the ventral side of the embryo in longitudinal bands of thickened ectoderm called *milk lines*, extending from the axilla to the pelvis. Most mammals develop a series of paired mammary glands along this ridge but in humans only a single pair of glands develop in the pectoral region of the milk line by the 9th week of gestation. It is here that a mass of basal cells proliferates to form the nipple bud. Squamous cells from the surface begin to invade the nipple bud

by the 12th week and while the epithelial cells grow downward as mammary ducts, terminating in lobular buds, the mesenchymal cells differentiate into smooth muscle of the nipple and areola. This specialized epithelium buds into 15–20 branches that first consist of solid epithelial columns and then, by the 20th–24th weeks of gestation, develop inner lumen and eventually canalize to form the lactiferous ducts. These branched epithelial tissues are stimulated to canalize by the placental sex hormones that enter the fetal circulation during the third trimester.

The ends of these branches differentiate into lobulo-alveolar structures that contain *colostrum*, which is a yellow, sticky and serous fluid. This colostrum milk, stimulated by the placental hormones, can be expressed for 4–7 days after birth in both male and female newborns ('witch's milk').^{1,2} During the final weeks of gestation the mass of the mammary gland increases consistently and the nipple–areola complex (NAC) develops and becomes pigmented. With the withdrawal of placental hormones after birth, the colostrum secretion stops and the breast starts a sort of involution.

The supporting structure of the breast, composed of connective tissue and fat, originates from the mesenchyme that invades and surrounds the epithelial system.

Failure of some of these stages in breast development to take place can result in congenital abnormalities of the breast. Ectopic breast tissue may be found in 1–6% of individuals.¹ Accessory nipples (polythelia), for example, are not uncommon along the milk line and are most often found on the chest wall below the breast (Figure 37.1). These form due to a failure of the regression of the milk streak. Inverted nipples are not uncommon either, and they are caused by a failure of the evagination of the small mammary pit at birth. Accessory mammary glands (polymastia), also due to a failure of the milk line to completely atrophy, are usually found in the axilla and cause the patient to complain of a mass or pain in this area. Absence of the breast (amastia) is a rare congenital anomaly; it occurs as a result of an arrested mammary ridge at about the 6th week of gestation.^{1–4}

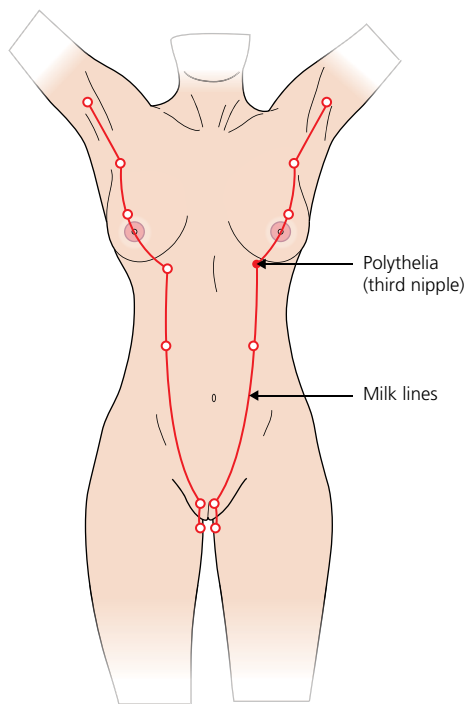


Figure 37.1 The milk line extends from the axilla to the groin. Aberrant breast and/or vestigial nipple and areola development can occur anywhere along this line.

Development of the breast from birth until puberty

During this period the breast consists of lactiferous ducts, with no alveoli. As puberty begins, the circulating oestrogen causes the ductal epithelium and surrounding stroma to grow. These ducts begin to arborize and form collecting ducts and terminal duct lobular units. These ultimately form buds that precede further breast lobules. Surrounding the ducts, vascularity increases and connective tissue increases in volume and elasticity, replacing adipose tissue and providing support for the development of ducts. Breast budding is one of the first sign of adolescence in girls, beginning anywhere between age 8 and 13 years.

Development of the breast during pregnancy

During pregnancy the breast attains its maximum development. Secreting alveoli appear, and there is marked growth of the ducts, lobules and alveoli under the influence of luteal and placental sex steroids and prolactin. During the first weeks, under the influence of oestrogen, there is ductal sprouting and lobular proliferation. By the second month the breasts have enlarged dramatically, with increased nipple and areolar pigmentation.⁵ The alveoli now display a lumen surrounded by the secretory cells. During the second trimester of pregnancy progesterone causes the lobular formation to exceed the ductal sprouting with a notable raise of prolactin levels.^{1,5} At this point the alveoli contain colostrum and the breast continues to enlarge. In the last trimester the stroma surrounding the lobules diminishes to make room for the

hypertrophied lobules and the breast starts secreting colostrum, which is then replaced by true secretion of milk. When lactation ceases, the glandular tissue returns to its resting state.

Development of the breast during menopause

Menopause typically occurs when a woman is in her late 40s and early 50s and it is associated with a variety of symptoms related to the loss of oestrogen and progesterone. The glandular tissue of the breast atrophies, the connective tissue becomes less cellular and the amount of collagen decreases. The loss of strength of the connective tissue usually results in an increase in volume and sag to the breast. However these changes of atrophy are variable and incomplete from woman to woman.

Anatomy of the breast

Breast mound

The breasts are located on the anterior and also partly the lateral aspects of the thorax. Each breast extends superiorly to the second rib, inferiorly to the sixth costal cartilage, medially to the sternum, and laterally to the midaxillary line.^{6,7} The superolateral part of the mammary gland extends towards the axilla, along the lower border of the pectoralis major, forming the axillary tail of Spence. Breast shape and size depend upon genetic, racial and dietary factors, and the age, parity and menopausal status of the individual. Breasts may be hemispherical, conical, variably pendulous, piriform or thin and flattened. Each breast usually assumes a conical form with a base measuring 10–12 cm and a thickness of 5–7 cm.^{3,8} The main bulk of the breast tissue is usually localized to its upper outer quadrant. This is the quadrant more often implicated in breast cancer and in most benign lesions of breast² (Figure 37.2).

The nipple–areola complex

The NAC is located between the fourth and fifth ribs in nulliparous women but its position varies widely when the breasts are pendulous. The complex typically measures 3–4 cm in diameter and is ideally located in the centre of the breast mound. The 15–20 lactiferous ducts open on to the nipple. Both the nipple and the areola consist of keratinizing stratified squamous epithelium with a dense basal melanin deposition. Melanocytes are quite numerous in the skin of the nipple and areola, giving them a darker colour than the remainder of the breast. The areola is a disc of skin that circles the base of the nipple, varying in colour from pink to dark brown depending on parity and race. It contains sebaceous glands, sweat glands and, visibly, accessory glands called *Montgomery's glands*, which open at the periphery of the areola as *Morgagni tubercles* that provide lubrication during lactation.^{3,9,10} Smooth muscle fibres lie underneath the areolar skin; these respond to tactile and autonomic sympathetic stimulation and are responsible for nipple erection and areolar constriction.^{3,11} The NAC is also provided with a rich sensory innervation.

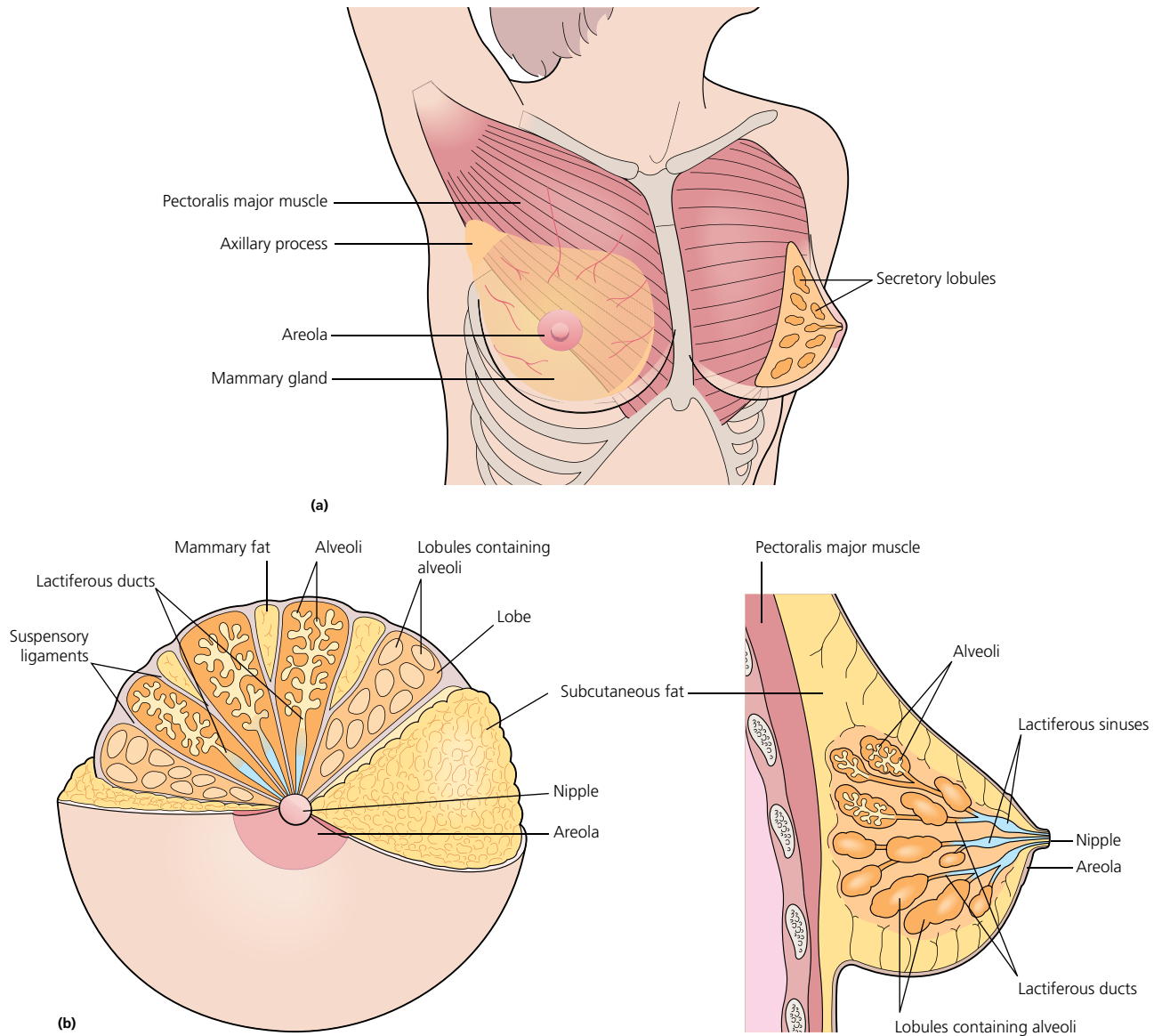


Figure 37.2 (a) Vascular and lymphatic anatomy of the breast region. (b) Section of the breast: (left) inner structure of the mammary gland; (right) section of the breast showing milk flow.

Fascial support and chest wall

Along its deep surface, the breast lies upon the deep pectoral fascia, which in turn overlies pectoralis major and serratus anterior, and inferiorly, external oblique and its aponeurosis as the latter forms the anterior wall of the sheath of rectus abdominis. The fascial relationships of the breast are of practical importance. The superior pectoral fascia, with the anterior layer, envelops the breast ventrally and a posterior layer dorsally. The superficial layer lies immediately beneath the dermis and enables skin flaps to be dissected from the glandular mass of the breast quickly and in a relatively avascular plane.² The deep layer of the superficial pectoral fascia is thicker than the subcutaneous component and lies on the posterior surface of the breast. Between this and the deep pectoral fascia there is a

layer of filmy areolar tissue (*retroamammary bursa*). This is identifiable during mastectomy and allows a bloodless plane of dissection.¹² It also allows the breast to move freely on the underlying fascial covering of the pectoralis major and the serratus anterior. Deep infiltration of a cancer through this space into the underlying pectoralis fascia produces the typical sign of deep tethering of a malignant breast mass.^{2,13}

Connecting these two fascial layers are the *suspensory ligaments of Cooper*, which provide some structural framework to the parenchyma. These ligaments have a degree of laxity and stretch to them that allows breast mobility while maintaining structural integrity. Contraction of this tissue by malignant infiltration results in the characteristic skin dimpling over a carcinoma of the breast (*peau d'orange*).^{2,3,13}

Other additional ligaments that take part in breast suspension have been described. The *ligamentum suspensorium mammae* starts at the clavicle and reaches to the upper border of the breast and the retromammary space.³ It is well defined in some individuals but could be almost indistinct in others. Another ligament structure has been described in recent years: a fibrous bridge exists between the pectoralis muscle and the skin of the inframammary crease. This ligament gives rise to the inframammary fold and acts as a sling for the breast parenchyma.^{14–16} This was found to be continuous with the fifth rib medially and the fascia between the fifth and sixth ribs laterally. Medially, this structure seems to be a condensation of the rectus abdominis fascia and laterally the fascia of the serratus anterior and external oblique muscle. This ligament should be carefully considered during a breast augmentation because preserving the ligament to the bony framework while releasing the pectoralis muscle would minimize the risk of the breast's bottoming out.

A horizontal septum that lies at the level of the fifth rib and traverses the glandular tissue has also been described.^{14–17} This septum becomes thicker along the medial and lateral edges and curves upward into vertically oriented ligaments that attach the breast to the thoracic wall. These ligaments are composed of a superficial and a deep structure and they connect medially to the inframammary crease ligament and laterally to the axillary fascia. The loss of the elastic component in these ligaments or their stretching in time contributes to loss of shape and firmness.

With regard to the rest of the fascia outside the breast area, above the breast, the superior pectoral fascia is continuous with the superficial cervical fascia, whereas at the inferior aspect of the breast, the anterior and posterior layers of the superior pectoral fascia rejoin and are continuous with Camper's and Scarpa's superficial abdominal fascia at the inframammary fold.^{1,13,18} The clavipectoral fascia runs deep to the pectoralis major muscle and surrounds the pectoralis minor muscle. Superiorly, this fascia thickens and attaches to the clavicle. This is an important landmark in surgery because it forms the roof of the axillary space also known as the axillary fascia.^{1,13}

Parenchyma

The breast parenchyma and associated fat make up the bulk of the volume of the breast. The fibroglandular tissue, or parenchyma, of the breast, is divided into 15–20 lactiferous ducts coming from deep within the breast lobules and converging at the nipple in a radial arrangement. These ducts are not always evenly distributed around the breast. The upper half of the breast, particularly the upper outer quadrant, usually contains more glandular tissue than does the rest of the breast. Each duct defines a lobe made of 20–40 lobules, each consisting of 10–100 alveoli. The main duct from each lobe opens separately on the tip of the nipple and possesses a dilated sinus just before its termination in the subareolar tissue. The ductal–lobular unit is the functional unit of the breast. The ducts are lined with stratified squamous epithelium and gradually transition to a double layer, then a single layer of cuboidal cells towards the nipple. The epithelial

lining of the lobule consists of superficial (luminal) A cells that are involved in milk synthesis.^{1,18} The B cells (basal) have stem cell activity.^{1,18} Finally, there are myoepithelial cells located around the alveoli and small excretory mild ducts between the inner aspect of the basement membrane and the tunica propria. These cells are stimulated hormonally.

The lobules are surrounded by connective tissue, but there is no distinct fascia separating the lobules. The connective tissue (stroma) that surrounds the lobules (intralobar) is dense and fibrocollagenous, whereas the intralobular connective tissue has a loose texture, which allows the rapid expansion of secretory tissue during pregnancy. The fibrous tissue surrounds the glandular components and extends to the skin and nipple, assisting in the mechanical coherence of the gland. The interlobar stroma contains variable amounts of adipose tissue, which contributes largely to the increase in breast size at puberty.

Blood supply

The blood supply of the breast is derived from the axillary, internal thoracic artery (internal mammary artery) and the intercostal arteries^{2,19} (Figure 37.3). The largest vessels arise from the internal thoracic artery, the perforating branches of which pierce the chest wall adjacent to the sternal edge in the first to fourth intercostal spaces, giving blood to the inner quadrants. Additionally, the internal thoracic artery gives rise to the posterior intercostal arteries, and branches of the intercostal arteries penetrate the deep surface of the breast. The axillary artery gives off the superior thoracic artery, the pectoral branch of the thoracoacromial artery, the lateral thoracic artery and the subscapular artery. Multiple muscle-perforating branches of the thoracoacromial artery perforate the pectoralis major to enter the breast from the upper quadrant. The lateral thoracic artery traverses along the lower border of the pectoralis minor muscle. During its course along the muscle, it gives off the external mammary artery, which comes ventrally through the muscle to supply the upper outer quadrant of the breast. All of these arteries connect with each other by collateral branches in both the breast and the overlying skin. Overall, 60% of the breast is supplied by the internal thoracic artery, 30% by the lateral thoracic artery, and 10% by minor contributions from the thoracoacromial, intercostals, subscapular and thoracodorsal arteries.¹

The venous drainage heads towards an anastomotic plexus in the subcutaneous tissue beneath and around the areola. This plexus then drains towards the periphery via large subcutaneous veins. These veins then follow their arteries and drain to the intercostals and axillary veins and to the internal thoracic veins. Of note is their communication with the prevertebral venous plexus.

Nerve supply

The innervation of the breast is principally by somatic sensory nerves and autonomic nerves accompanying the blood vessels. The somatic sensory nerve supply is via the supraclavicular nerves (C3, C4) superiorly and laterally from the lateral branches of the thoracic intercostal nerves.⁶ The medial

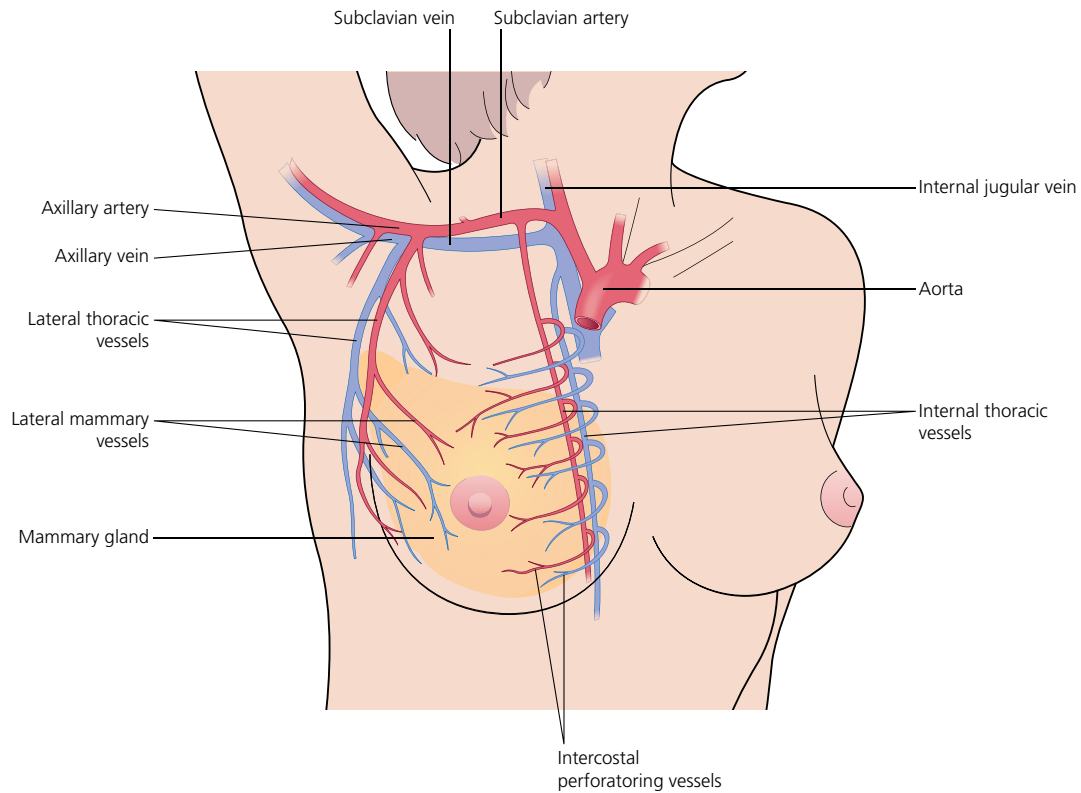


Figure 37.3 Vascular anatomy of the breast area.

aspects of the breast receive supply from the anterior branches of the thoracic intercostal nerves, which penetrate the pectoralis major to reach the breast skin. These lateral and medial cutaneous branches come from the second through to the sixth intercostal nerves. The major supply of the upper outer quadrant of the breast is via the intercostobrachial nerve (C8, T1). It gives a large branch to the breast as it traverses the axilla. The location of the nerves within the breast varies quite often. After passing through the intercostal spaces, the nerves ramify within the breast, sometimes passing along the deep fascia, sometimes passing superficially through the substance of the breast.

With regard to the all-important innervation to the NAC, the anterior and lateral branches of the intercostal nerves and, in particular, the lateral branch of the fourth intercostal nerve tend to ramify predominantly to the subareolar plexus, although lesser and variable contributions from other surrounding intercostal nerves also ramify to the area. Most of the time this lateral branch takes a subglandular course within the pectoral fascia to approach the nipple from the posterior surface, running along the same septum that carries the vascular network.²⁰ On the other hand the medial nipple innervation is most commonly derived from anterior cutaneous branches of the third and fourth intercostal nerves, which take a more superficial approach in the subcutaneous tissue and become more superficial as they reach the medial areola.^{3,6,11}

Lymphatic drainage

The lymphatic drainage of the breast is diffuse and variable. It works through two sets of lymphatic vessels: the superficial (subepithelial or subdermal) and the deep^{1,13} (Figure 37.4). The subepithelial plexus connects to subdermal lymphatic vessels by vertical lymphatics and from them to the deep plexus. In the breast, the subepithelial and subdermal plexus are confluent with the subareolar plexus in which the fine lymphatics of the lactiferous ducts and the lymphatics of the NAC also converge. From the deep lymphatics, the lymph flows towards the axillary and internal thoracic nodes. There are 20–30 lymph nodes in the axilla, divided into five anatomical groups.² The apical nodes unite into the subclavian trunk; on the left side this trunk drains directly into the thoracic duct whereas on the right side it may empty directly into the jugulosubclavian junction or into a common right lymphatic duct.

From a clinic and pathologic point of view the lymph nodes within the axilla are divided into three levels based on the location of the nodes relative to the pectoralis minor muscle.^{1–3,13} Level I nodes are situated inferiorly or laterally to the lower border of the pectoralis minor. This group includes the external mammary, axillary vein and scapular lymph nodes. Level II nodes lie deep or behind the pectoralis minor and include the central lymph node group and the subclavicular nodes. Finally, level III nodes are situated above the upper border of the pectoralis minor muscle and include the subclavicular and apical

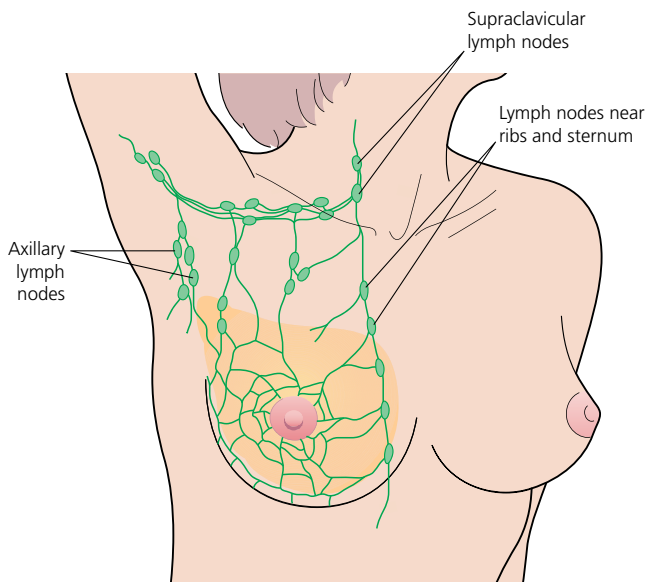


Figure 37.4 Structure anatomy of the inner breast and lymphatic mapping of the breast area.

node groups. The internal thoracic lymph nodes lie along the internal thoracic vessels and are very small. They drain the inner aspect of the mammary gland and also the anterior chest wall, the anterior portion of the diaphragm, the upper portion of the rectus sheath and muscle and the superior portion of the liver. Another group of nodes – the intercostal nodes – take a small part to the lymphatic drainage of the breast. These lie near the rib heads and receive deep lymph vessels from the postero-medial aspect of the chest and some drainage from the lateral extremity of the mammary gland.

The lymphatic drainage from the breast can run different routes before getting to the nodes.³ It may go lateral, medial, transpectoral and retropectoral. The lateral route is the most important, about 75% of all lymphatic drainage passes to the axillary nodes whereas the remainder principally drains medially into the internal thoracic lymph nodes. It is important to underline that any part of the breast could drain into either group although there is a tendency of the lateral group to drain towards the axilla and of the medial to drain towards the internal thoracic lymph nodes.

Transpectoral drainage happens through Rotter's nodes that are situated beneath the pectoralis major muscle. They drain into lymphatics along the thoracoacromial artery to the subclavicular group of level III.

The last route of drainage is the retropectoral, in which the superior and internal aspects of the breast drain into the subclavicular node group in the axilla, passing through the lymphatics located along the inferior portions of the pectoralis muscles. Lymphatics do not normally drain across the contralateral side and this has to be taken into consideration in cancer patients.²

Physiology of the breast

Physiologically, the breast is an organ specialized for milk formation (lactation), including synthesis, secretion and ejection of milk.^{1-4,9,10,18} The secretory units of the breasts are the alveoli, small saccules in continuity with the lactiferous ducts. A complex network of hormones and growth factors controls the production of milk by these secretory units. The fluctuation of these hormones results in important histologic changes in the breast during pregnancy and during the menstrual cycle.^{1,4,5,18}

Oestrogen is the main female hormone responsible for breast development and maintenance. It leads to growth of the ductal system and also maturation and prominence of the nipples, resulting in proliferation of the ductal epithelium, myoepithelial cells and surrounding stroma. Oestrogen is lipid soluble and in a woman's body is made by the ovaries and to a lesser extent by the adrenal glands. It is stimulated to act in the presence of other hormones such as hydrocortisone, insulin-like growth factors and growth hormones.

Progesterone is released by the ovaries and induces development of the terminal ducts and lobulo-alveolar structures. Like oestrogen, it needs the presence of the other hormones, such as growth hormones and insulin, to respond. Both oestrogen and progesterone can increase connective tissue and fat in the breast, thereby leading to the rounded form of the fully developed breast.

Prolactin is produced by the acidophilic cells of the anterior pituitary gland. It cooperates with oestrogen in ductal development and with progesterone in lobulo-alveolar development. Together with cortisol and insulin prolactin helps to differentiate alveolar cells into milk-secreting cells. Generally speaking, it stimulates mammary growth and differentiation and ultimately milk production. Prolactin is primarily stimulated by the thyrotropin-releasing hormone (TRH) and inhibited by dopamine, but it can also be produced by cells in the breast, behaving as a paracrine or autocrine factor.

Oxytocin is a peptide hormone that is synthesized in the hypothalamus and released by the posterior pituitary gland (neurohypophysis). The act of nursing (the sucking reflex) stimulates its release.¹⁻⁴ Oxytocin causes myoepithelial cells to contract, which squeezes milk out from the lobules into the lactiferous ducts.

Human placental lactogen (hPL) is produced by the maternal placenta and serum levels continue to rise throughout pregnancy. It is related to breast growth and differentiation during pregnancy and reaches a peak during the final weeks of gestation, preparing the breast for milk production. Its serum levels decline rapidly after birth.

During the menstrual cycle there is a fluctuating alternation of hormones. In the follicular phase (the days from the first day of menstruation to the ovulation) follicle-stimulating hormone (FSH) levels rise, stimulating the development of follicles and their secretion of oestrogen.^{1,4} Increased oestrogen levels give

rise to cellular mitoses within the epithelial cells of the breast and also increase the blood flow, leading to an increase in breast volume due to intralobular oedema and enhanced cellular proliferation. The luteal phase is initiated by a peak of luteinizing hormone (LH), which leads to an increase in progesterone.^{1,4} As a consequence the mammary ducts begin to dilate and the alveolar epithelial cells to differentiate into secretory cells. Breast epithelial cell proliferation increases tremendously during the luteal phase of the menstrual cycle. With the onset of menstruation the rapid decline in the circulating levels of oestrogen and progesterone in the breast causes a regression of the secretory activity of the epithelium and the breast volume decreases, along with the tissue oedema, reducing the breast volume observed one week after menstruation.

During pregnancy, the breast reaches its maximum development.¹⁻⁵ High levels of oestrogen and progesterone are maintained by the corpus luteum initially and by the placenta during the latter phase. Oestrogen stimulates the ducts and lobules to proliferate and the glandular epithelium replaces the fat tissue. In the second trimester, progesterone activates the secretory epithelium. During the last trimester the alveolar and ductal spaces are filled with colostrum and towards the end of pregnancy there is a rise in prolactin circulation and the production of milk, fat and proteins begins.

After birth, the decrease of oestrogen and progesterone releases the inhibition on prolactin and lactogenesis begins. There are three main lactogenic hormones that act alongside the actions of glucocorticoids, insulin and thyroxine for milk production: prolactin, hPL and oxytocin. hPL prepares the breast for lactogenesis during the final weeks of pregnancy. Soon after delivery hPL disappears and prolactin acts as the sole lactogenic hormone. It causes the synthesis and secretion of milk into the alveolar spaces. Oxytocin causes the myoepithelial cells to contract, increasing milk delivery. The sucking reflex promotes the increase in both prolactin, stimulating more production, and oxytocin, increasing its release.¹⁻⁴

During the first days after birth, the mother's breast does not produce real milk, but it produces colostrum, with a high immunoglobulin concentration that enhances the underdeveloped immune system of the infant. After 5-7 days the milk becomes filled with all the nutrients and antibodies necessary for growth. When breastfeeding stops, the milk is being produced for several months but in lesser amounts. The breast usually returns to its previous size.

The breast undergoes several changes when a woman reaches the age of around 50 years old and enters the menopause. The loss of oestrogen and progesterone as a consequence of diminished ovarian function produces several general symptoms and changes within the mammary gland. The systemic symptoms include night sweats, mood disturbances, vaginal dryness, hot flashes and difficulty sleeping.¹⁻⁴ In the breast there is a decrease of number of lobular units, causing a loss of glandular tissue, which is replaced with adipose tissue. The breast eventually sags, becoming flatter and less voluminous.

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CHAPTER 38

Breast augmentation

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Introduction

Breast augmentation is the most popular aesthetic surgery procedure. Despite or perhaps because of this, it has had a chequered history in comparison to other major aesthetic procedures. The vast amount of space and literature devoted to the subject is not only a testament to its pre-eminence in the field, but also results in arrays of differing opinions and philosophies.^{1–33} This is, perhaps, chiefly because of the use of prosthetic devices and intermittent controversy regarding their safety.^{8,34–36} Czerny reported the first breast augmentation when he transferred a lipoma from a patient's back to the breast.³⁷ Over the years many injectable as well as implantable materials have been used, including shellac, beeswax, epoxy resin, petroleum jelly, Teflon, polyurethane and polyvinyl alcohol to name a few. In 1961 injectable silicone was reported by Uchida.³⁸ The introduction of the silicone breast gel implant by Cronin and Gerow in 1962 revolutionized breast augmentation surgery.³⁹

Silicone, or dimethylpolysiloxane, was first developed during World War II in the field of aircraft engineering. Its softness and inert nature made it the perfect prosthetic substrate for medicine.⁴⁰ Silicone breast implants can be divided into five generations based on the shell thickness, texture and implant fill (Table 38.1). The other major clinically applicable implants are saline implants. Alternative implant materials have been developed over the years, including polyvinylpyrrolide, triglyceride and hydrogel, but none have withstood the test of time.^{9,41} Current implants available on the market are variations on the same theme, divided by gel fill into saline and silicone.^{1,5,8,15,36,42} The silicone fill is subdivided (a) into form-stable and soft, based on level of cross-linkage; (b) by shell into textured and smooth; and finally (c) by shape into round versus anatomical. Following the controversy involving connective tissue disorders in the early 1990s, silicone implants have been once again given US Food and Drug Administration (FDA) approval for use in breast augmentations. They were never subject to bans in the rest of the Western world for the purposes of augmentation.⁴³

Implant evolution

The first-generation implants manufactured by Dow Corning were composed of viscous silicone gel contained in a thick, teardrop-shaped shell. Seams and a Dacron patch present on the periphery and posteriorly, respectively, were thought to help stabilize the implant in position. The high early capsular contracture rates resulted in an evolutionary step towards a second generation of softer implants in the mid to late 1970s, which were smooth, seamless and contained less viscous silicone.^{44,45} Although this reduced the capsular contracture rates somewhat, they were associated with a phenomenon known as 'gel bleed', related to small molecular weight particles of silicone diffusing across the thin silicone elastomer shells. This in turn has been implicated in the pathogenesis of capsular contracture.^{46,47} Recognition of this led manufacturers to develop a third generation of implants with a strengthened silicone shell. Two layers of high-performance elastomer formed the shell, with a fluorosilicone coating barrier between them.

The fourth generation of textured implants was developed partly in response to a desire to emulate the natural pores in polyurethane foam.⁴⁸ Polyurethane foam applied as a thin layer in the 1970s onto the implant shells resulted in very low capsular contracture rates. The process of delamination of the foam from the implant resulted in a non-contractile capsule, which was immobile yet had a soft feel.^{35,49–51} These shells were withdrawn in 1990 after concerns were raised about the carcinogenic nature of the polyurethane by-products,³⁵ but the good clinical outcomes led to development of two forms of pores by American manufacturers: Biocell developed by McGhan Medical and Siltex developed by Mentor. Biocell is composed of an irregular open pore textured surface with an average density of 3.1 pores/mm² and an average pore size of 289 µm. These pores are created by a lost salt technique and develop the capsule through an adhesive effect.⁵² Although this is similar to polyurethane there is no delamination. This shell characteristic has been used for both saline as well as silicone-filled implants, resulting in immobile

Table 38.1 Alloplastic material

Implant generation	Production period	Shell characteristics	Silicone gel characteristics	Shape
First	1962–1970	Thick shell (average 0.25 mm), Dacron patch	Thick viscous	Anatomical
Second	1970–1982	Thin shells (average 0.13 mm), no patch, smooth surface	Less viscous softer gel	Round
Third	1982–1992	Thick low bleed shells, smooth surface ¹	More viscous than second generation	Round
Fourth	1993–present	Thick, strong, low bleed shell, smooth and textured surfaces	More viscous cohesive gel	Round and anatomical
Fifth	1993–present	Thick, strong, low bleed shell, smooth and textured surfaces	Cohesive and form-stable gel	Round and large array of anatomical

yet soft implant feel and lower contracture rates.⁵³ Siltex is a textured surface characterized by a pattern of nodules ranging in height from 65 to 150 μm and in width from 60 to 275 μm .⁵² Implants made of this material do not adhere to the surrounding tissues, so they are mobile in the pocket. They also have lower contracture rates, whether saline or silicone filled.⁵⁴

The fifth generation of silicone implants are textured implants with form-stable silicone. This is achieved by increasing the cross-linkage of the silicone gel, allowing the implants to maintain their shape in all positions.^{36,55,56} The only dual-chamber implant that remains on the market is the Becker inflatable implant.

Saline implants were first reported by the French, as part of a drive for a less invasive and smaller scar surgery in 1965.⁵⁷ This remained the initial motivation for their commercial development in the United States rather than the nature of the filler or capsular contracture rates. The main disadvantage of these devices is their deflation rate and feel. The deflation rates in the initial offerings were as high as 75% at 3 years. The shells were thickened with a room-temperature vulcanized process. This reduced their deflation rates. In relation to their feel there are two main issues – surface wrinkles and the round ball appearance when overfilled.^{57,58} The current accepted rate of deflation is at 1–4% for the first 10 years.⁵⁹

Capsular contracture pathophysiology and study outcomes

Capsular contracture remains the most significant drawback of breast implants.^{12,60,61} The risk of capsular contractures warranting revisional surgery is estimated to be anywhere from 5 to as high as 59%.^{62,63} Most capsular contractures occur in the first 18 months post surgery.^{4,10,13,60,62–66} Baker devised the most commonly described classification of capsular contractures, but one drawback is that it suffers from a relative subjectivity of assessment.⁶⁷ The pathogenesis of capsular contractures remains unclear and is most likely multifactorial. Two theories have emerged as major contenders – the scar formation^{68,69} and infective theories.^{1,70–73}

Histologically, these capsules are made of an inner layer of cellular elements, synovial type metaplasia, fibrocytes and multinucleated giant cells, a middle layer of vascular networks and an outer layer of collagen bundles.^{36,44,47,52,74,75} A plethora of studies have been produced to implicate any and all factors involved in surgery, the device fill and shell; surgical access incision and pocket; use of antiseptics and antibiotics; use of drains, as well as surgical technique.^{18,31,70–72,76–78} Subpectoral pockets were developed in response to the contention that protection, coverage and regular contraction of the muscle would hinder and disrupt the formation of periprosthetic capsule. The fact that no one definitive study exists controlling for all variables indicates strongly that capsular contractures are most likely a result of multiple factors.^{28,31,66,79}

A large number of studies have been produced over the years, especially at the inception of each generation of implants to proclaim their superiority over the previous generation. Many if not most of these studies have lacked a rigorous scientific methodology in double blinding, randomizing, assessing and follow-up criteria, which weakens their assertions.^{64,80} More recent studies have shown a trend to reduced capsular contracture rates during follow-up: 15–20% for the Inamed,⁶⁰ 2.4% for the Mentor 6-year follow-up series and 1.7% for the Danish registry series.⁸¹ It is interesting to note that in all these series secondary operations ranged between 4.8% for the Danish series and 18–28% in the Mentor and Inamed series respectively. Another interesting fact is the almost universal (91%), use of the inframammary approach in the Mentor series (reporting lower capsular contracture rates) and the almost even distribution of the periareolar and inframammary approaches in the Inamed series.

In general, however, the choices are related to: access incision, implant type, type of surgical pocket, periprosthetic drains, and the use of perioperative antibiotics and irrigation of the pocket with antiseptics and/or antibiotics solution.

Access incision

The most commonly used access incisions available for primary breast augmentation are transaxillary, inframammary and periareolar. Umbilical incision and access remains in the realms of surgical possibilities, with little reasonable surgical philosophical

justification in the opinion of the authors. The debate regarding the relative merit of each access site has been ferociously debated over the years by the great and the good. There is compelling evidence that in addition to reduced access, periareolar incision exposes the implant to potentially higher risks of bacterial contamination and as such is associated with increased capsular contracture rates.^{82,83} An interesting finding was increased rates of capsular contracture in transaxillary incisions in comparison to both inframammary and periareolar incisions.⁸⁴

Implant type

One of the major advantages of saline implants was originally thought to be their lower rate of capsular contracture. Early intracellular evidence of silicone in macrophages from silicone implants was held to be an indication of their superiority and reason for reduced capsular contracture rates. Similar evidence, however, has since been demonstrated in saline implants.^{85–87}

Saline versus silicone

Supporting evidence demonstrating lower contracture rates for saline-filled implants came relatively early on.^{65,88–90} Despite the early enthusiasm and convictions of surgeons that saline implants resulted in lower capsular contracture rates, there have been many contradictory results. Most recently, a systematic analysis of randomized controlled trials failed to demonstrate a clear reduction of capsular contractures with the use of saline implants.⁹¹

Surgical pockets

Subglandular

A systemic review published in 2006 reviewed the results of various trials comparing textured and smooth implants in subglandular breast augmentation.⁶⁴ They identified six randomized controlled trials.^{53,54,63,74,76,92–94} Four trials used saline and two used silicone gel. In total, these trials included a total of 235 patients. One trial did not show any difference between capsular contracture rates,⁹² whereas the remainder confirmed a lower capsular contracture rate for patients with textured implants.

Subpectoral

A study of 55 breasts, which is one of the only major, randomized trials of this kind, compared textured and smooth silicone implants placed in the submuscular pocket. The final outcome trended towards lower capsular contracture rates in the textured group (from 15% to 8.6%) but the results were not statistically significant.⁹⁵

Finally, a more recent meta-analysis using most of the above studies demonstrated a five-fold increased incidence of capsular contractures with smooth implants.⁶²

Periprosthetic drains

There is some evidence that periprosthetic drains are associated with a higher risk of infection and also capsular contractures. Many surgeons advocate not using drains in primary breast augmentation.^{96,97}

Perioperative antibiotics and irrigation of pocket with antiseptics and/or antibiotics solution

Periprosthetic and subclinical infection theory has a significant body of evidence behind it.^{71,73,98,99} What is less clear is whether it is in fact the cause of capsular contracture. It has been shown that up to 80% of female breast samples contain bacteria.¹⁰⁰ Bacterial presence in up to 83% of nipple secretions is considered a cautionary tale for the use of periareolar incisions.¹⁰¹ The most commonly implicated organism is a coagulase-negative *Staphylococcus epidermidis*, although many other organisms have been isolated. Basic science evidence of an accelerated capsular contracture following *Staphylococcus aureus* contamination has also been demonstrated.¹⁰²

The results of pocket irrigation with antiseptics and use of antibiotics have been contradictory.^{1,54,70,71} Adams and colleagues used a triple antibiotic solution, the Adam's solution (1 g cefazolin, 80 mg gentamicin and 50 000 bacitracin, replaced by 50 mL povidone–iodine from 2000 onwards mixed with 500 mL normal saline) to show a reduction of capsular contracture (Baker III/IV) rates to 1.8 for primary augmentation and 0% for augmentation mastopexy during a 6-year prospective clinical study.¹⁰³ This is supported by other similar studies showing a reduction in capsular contracture rates.¹⁰⁴

Use of Betadine irrigation of the implant pocket first championed by Burkhardt and colleagues^{54,76} as a means of reducing capsular contractures has been confirmed to reduce capsular contracture rates and to have no deleterious effects on the silicone shell of the implants, as reported by one anecdotal case.¹⁰⁵ On the other hand, Gylbert and colleagues were unable to show any statistically significant difference in capsular contracture rates, using preoperative antibiotics in primary breast augmentation.⁶⁵

Indications

Glandular hypomastia may occur as a developmental or involutional process and affects a significant number of women. Developmental hypomastia is often seen as primary mammary hypoplasia, or as a sequela of thoracic hypoplasia (Poland's syndrome) or other chest wall deformities. Involutional hypomastia may develop in the postpartum setting and may be exacerbated by breastfeeding or significant weight loss. When compared to the norm, inadequate breast volume may lead to a negative body image, feelings of inadequacy and low self-esteem. These disturbances may adversely affect a patient's interpersonal relationships, sexual fulfillment and quality of life.

The initial consultation for augmentation should begin with open-ended questions about the patient's goals and expectations for the procedure. Patients today have often spent some time researching the procedure either through friends or through the internet. The surgeon should be able to form an impression of the patient as a well-informed, psychiatrically stable person with appropriately realistic expectations for the procedure. Any

concerns about the patient's level of understanding, unrealistic expectations or self-esteem issues should be fully explored prior to proceeding with surgery. A careful medical history and physical examination is essential for the assessment of risk factors and candidacy for breast augmentation.

The ideal size and shape of the female breast is inherently subjective and relates to both personal preference and to cultural norms. However, most surgeons will agree that there are certain shared characteristics which represent the aesthetic ideal of the female breast form. These characteristics include a profile with a sloping or full upper pole and a gently curved lower pole with the nipple–areolar complex at the point of maximal projection. The breast structure itself may be thought of as the breast parenchyma resting on the anterior chest wall surrounded by a soft tissue envelope made up of skin and subcutaneous fat. Clearly, the resulting form of the breast after augmentation mammoplasty will be determined by the dynamic interaction of the breast implant, the parenchyma and the soft tissue envelope.

Assessment and implant selection

Patients seeking breast augmentation need to undergo a thorough consultation process. At the initial consultation the breast should be assessed against the patient's expectation. In order to facilitate this crucial process a number of methods are available.

A thorough physical examination begins with observation and careful documentation of any signs of chest wall deformity or spinal curvature. It is imperative to document and draw attention to any asymmetry of breast size, nipple position or inframammary fold (IMF) position. Careful palpation of all quadrants of the breast and axilla is required to rule out any dominant masses or suspicious lymph nodes. While palpating the breast, the surgeon should carefully assess the quantity and compliance of the parenchyma and soft tissue envelope. The soft tissue pinch test is a useful method of assessment, in which the superior pole of the breast is gathered between the examiner's thumb and index finger, measuring the thickness of the intervening tissue. In general, a pinch test result of <2 cm will often indicate a need for subpectoral placement of the implant. It is also important to characterize the amount, quality and distribution of the breast parenchyma as it may be necessary to reshape or redistribute the parenchyma to achieve the desired shape of the breast mound. The elasticity of the skin should also be characterized by observing its resistance to deflection and noting any signs of skin redundancy or stretch marks.

Manufacturers have developed a preoperative planning system to facilitate patient assessment and implant selection. In addition, the authors utilize 4-D technology to assess both patient's chest wall and soft tissue abnormalities. Chest wall, soft tissue asymmetries and combinations of both are important to identify preoperatively. This tool is also utilized to understand the patient's perception of the desired look by

showing the outcomes of using different sizes and styles of implant. This is also then projected in the operating room for surgical planning.

Operative techniques

Preoperative markings

The general principles of surgery need to be observed even more stringently when use of prosthetic material is involved. Once the patient has been appropriately informed and consented by the surgeon, the most basic operative technique decisions involve choices in access incision and pocket creation. Use of sterile technique, no-touch approach and perioperative antibiotics are naturally essential.

Access incision

Inframammary

This is by far the most common and versatile access incision. It allows clear dissection, subsequent visualization of the implant pocket and placement of the implant in any pocket. The evidence also suggests that it is associated with least capsular contractions.^{82–84} This may be due to clear visualization and 'dry' technique, allowing good haemostasis and reduced risk of bacterial contamination through the incision and operative field. Its direct access to the glandular–muscular interface and overlap allows manipulation of either or both for the best symmetrical and aesthetically pleasing outcome.

The drawbacks of this access are the relative visibility of the scar, its site and placement. The incision should be placed in the predicted location of the new IMF, which has been determined and marked preoperatively. An incision length of up to 5.0 cm is required. The incision should be designed with the majority of the incision lateral to the breast midline, as this will place the resulting scar in the deepest portion of the new IMF.

Periareolar approach

The periareolar approach for augmentation mammoplasty was described by Jenny in 1972. The principal advantage of this incision is that the resulting scar is usually well-camouflaged and quite inconspicuous. The periareolar approach allows easy adjustment of the IMF and direct access to the parenchyma for scoring and release when the lower pole of the breast is constricted. Whether this is a transparenchymal approach or periparenchymal via dissection to the inferior pole, this incision allows for access for creation of subglandular, submuscular as well as subfascial pockets. In addition to the higher risk of contamination and apparent association with higher capsular contracture rates, it is associated with reduced sensitivity of the nipple–areolar complex (NAC) as well as increased rates of periareolar pain.^{82,83,106} The dissenting view has been offered by a much smaller series of 20 patients not showing any difference between the two incision types.¹⁰⁷

In pigmented skin the risk of keloid and depigmentation remains high. It is also exceedingly difficult to place many cohesive or anatomically shaped implants through this incision. Despite the evidence, this access continues to enjoy popularity with many surgeons who argue for its use given the 'direct' access, 'remote' control of the IMF and 'invisible' incision.¹⁰⁸ In the opinion of the authors in light of the basic science and clinical series evidence there is no justification in using the peri-areolar incision.

Transaxillary incision

This incision technique was first developed in the 1970s as an open technique, before being adapted to a minimally invasive approach.¹⁰⁹ Although this technique theoretically offers access to creation of all three implant pockets, the traditional open technique involved blunt dissection of the inferior origins of the pectoralis major muscle for creation of the submuscular pocket. The open blunt technique led to a series of suboptimal aesthetic outcomes. This was in part due to uneven coverage of the implant and its malposition owing to, in particular, a lack of controlled dissection of the vascular inferomedial aspect of pectoralis major.³² This is also where major intercostal vessels traverse the space, increasing the risk of haematomas. As saline implants became popular, especially in the United States, proponents of this technique increased. Introduction of saline implants through a 2–3 cm incision, enhanced by precision view allowed by endoscopic assistance, became a major standard. Currently, the technique is performed with a slightly longer incision of 4–5 cm for use of silicone implants. Although textured implants can be placed via this incision, anatomical-shaped implants cannot routinely be placed.³² The IMF is lowered 1–2 cm depending on size and asymmetry through this technique.

The proponents of this technique point to its track record in safety, its minimal incision hidden in the axilla and remote access and better control of the IMF.³² With the exception of the evidence implicating this incision in increased capsular contractures,⁸⁴ the authors believe that this technique is more anatomically and surgically sound than the periareolar technique but less versatile than the inframammary incision. However, the purported control over the IMF is offset, in the opinion of the authors, by the fact that there is little or no possibility for a nuanced manipulation of the gland–muscle interface or position at the IMF. This is imperative in view of the authors, as it gives a more direct control on the NAC-to-IMF distance as well as the shape of the lower pole. Despite this, Tebbetts reports excellent equivalent outcomes with both dual-plane and transaxillary techniques with long-term follow-up.³⁰

Transumbilical

This technique was developed in the 1990s.¹¹⁰ The first large series reported by Dowden argued for its use based on the following: versatility (access to both subpectoral and subglandular pockets), minimal scarring and no implant tension on

wound, reduced loss of nipple sensation, elimination of deadspace, and, finally, good control of the implant pocket.¹¹¹ This technique is only suited to inflatable saline implants. It involves blunt dissection over the rectus sheath towards the breast. Creation of the subglandular or subpectoral pockets is achieved by means of avulsion of the tissues, including the inferior pectoralis major (for the subpectoral pocket). Despite purported good results by Dowden and other surgeons, the authors feel this is not a surgically sound technique. It has poor to no visualization of the operative field, which defines surgery and its planned accuracy. It only has application for inflatable saline implants. Its safety based on basic surgical principles cannot be recommended in comparison to any of the other incisions.

Pocket creation

Subglandular

The original implant pockets were dissected in a subglandular plane. Where soft tissue envelope is considered thick enough, many surgeons continue to use a subglandular pocket for implant placement.

Submuscular

This approach is developed in either a complete or partial submuscular pocket. The lower edge of the pectoralis major is dissected off the chest wall and the lower sternum. For a total submuscular pocket it may be necessary to also dissect serratus anterior and the external oblique off the chest wall. The issues of animation of the implants with contraction, the implants riding too high and the wide cleavage are well-known drawbacks of the submuscular plane. By definition with this technique it can sometimes be difficult to place the NAC on the most projected part of the breast tissue.

Tebbetts described the dual-plane technique as a means of addressing the various shortcomings with each technique and allowing a nuanced approach to implant placement and pocket dissection.^{28,31} He described dissection of tissue in both submuscular as well as subglandular planes. The lower edge of the pectoralis major is dissected in all cases followed by dissection of the breast tissue from the underlying pectoralis major. He described three different levels of dissection of the breast tissue–muscle interface and redraping of the pectoralis major muscle on the implant as projected NAC level (Figure 38.1):

- Type I: No dissection, the inferior border of the muscle covers the implant and entire NAC level.
- Type II: The inferior border of the muscle drapes over the implant to the level of the NAC.
- Type III: The inferior border of the muscle retracts to the superior margin of the NAC.

The authors believe that appropriate utilization of the dual-plane technique provides the most flexible and nuanced approach for implant positioning and placement. It goes a long way to alleviating the implant animation problem due to muscle

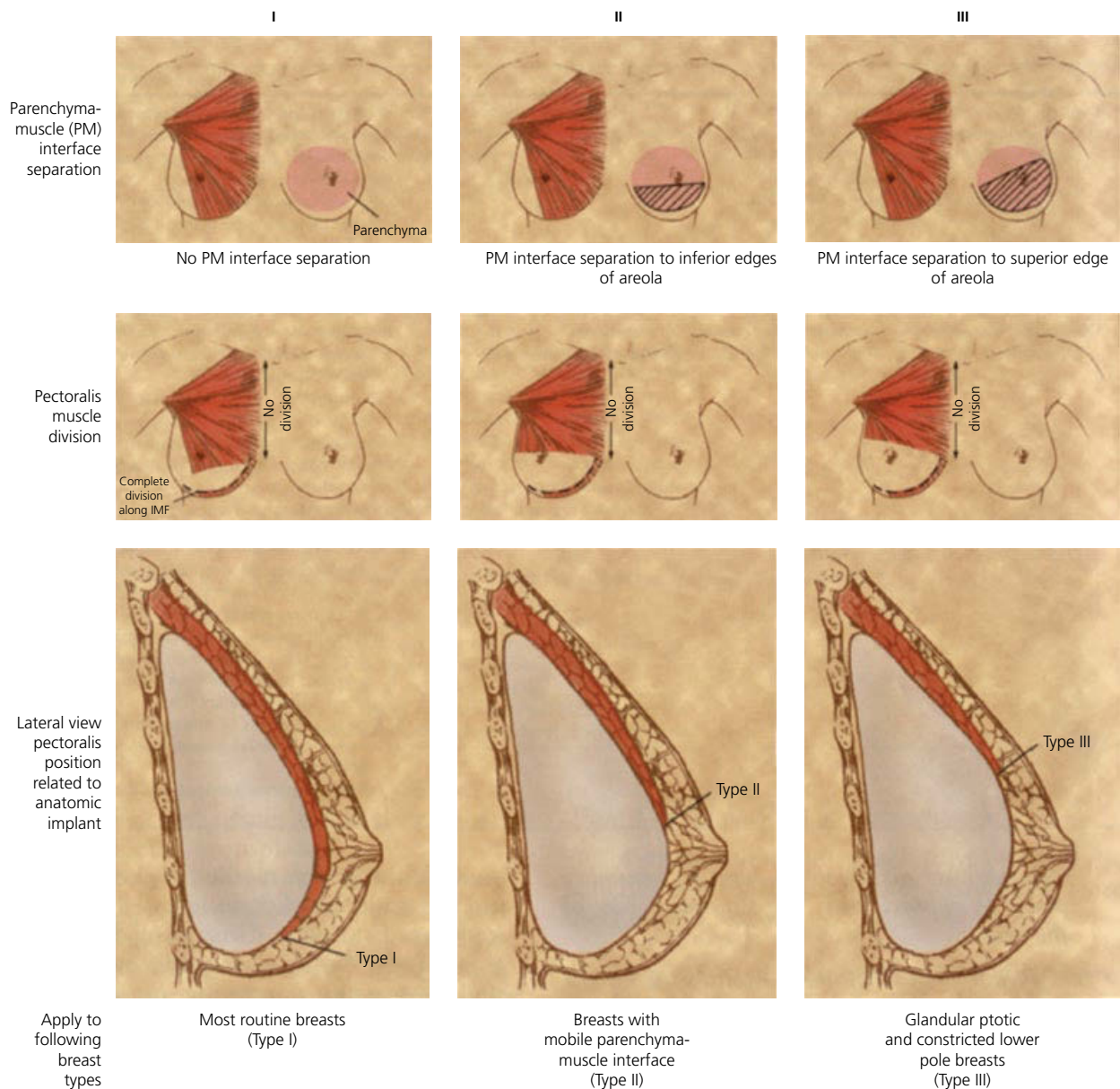


Figure 38.1 Schematic diagram representing the extent of dissection of breast parenchyma off the pectoralis major and subsequent pectoral release in dual-plane breast augmentation. Source: Tebbetts, 2006.³⁰ Reproduced with permission of Lippincott Williams & Wilkins.

contraction. It allows a good soft tissue coverage of the upper pole, and facilitates not only the expansion of the lower pole but eases adjustment of the IMF should that be necessary. Additionally, this technique has been shown to reduce capsular contracture rates.⁶⁶

Subfascial

This is a more recent technique. Here, the implant is placed under the pectoralis major fascia. Very low or no capsular contractures in patients undergoing this technique have been reported.¹¹² The authors have little experience with this technique, but observe that the pectoralis major fascia is rather thin

and indeed may not alleviate the issues of implant animation or optimal NAC position on the breast mound.¹¹³

Personal preference

Preoperative markings are made with the patient in the upright position and are useful as a reference point during the actual procedure. The surgeon should mark the chest midline in the frontal view from the suprasternal notch to the xiphoid process, the existing IMFs and the likely position of the new IMFs as the proposed limits of the dissection. The patient is then

asked to place her arms behind her head to visualize the displacement of the nipple and the true IMF. The senior author uses the Akademikliniken method for preoperative markings. The two vital parameters to determine are the vertical position of the implant on the chest wall and the position of the new IMF. This latter is determined by the width and projection of the implant, the amount of breast glandular tissue and the characteristics of the skin envelope. The natural shape of breasts is not circular nor spherical; ideal implants in the opinion of the authors are form-stable anatomical implants, simulating natural breast shape. The ideal natural appearance of the NAC is at mid-height of an anatomical form-stable implant. The NAC is elevated with augmentation; the easiest way to simulate this is by asking the patient to clasp her hands over her head and marking this against the sternum at midline. Central points are relatively constant as reference positions. Based on these markings the mid-height of the implant is determined on the chest wall.

The next step is to determine the position of the new IMF. There are multiple ways of estimating this. In general, for small breasts the IMF is lowered by about 1.0 cm, for moderately sized breasts it is lowered by 1.0–1.5 cm and for larger breasts it is lowered by 1.5–2.0 cm. Another way to determine this is by pinching the lower pole breast tissue with a caliper and dividing the value by 2. These would have to be slightly modified for ptotic breasts, where there is excessive skin at the lower pole.

The patient is then placed in the supine position, centred on the operating table with the pelvis directly over the flexion point of the bed. The arms must be well secured to the armboards, which are placed at 90° angles to the torso. These preparations are required so that the patient may be placed in the upright seated position as often as needed during the procedure. The sterile preparation and draping of the anterior chest must provide visualization of the patient's shoulders as an important anatomical reference point. Triple antibiotic solution irrigation is used for all implant cases regardless of the incision type.

The authors use the inframammary incision approach in all procedures. The location of the incision is based on the implant choice with the added subglandular tissue. The senior author in almost all cases creates a dual-plane II–III pocket with anatomical implants. The pocket is precisely dissected to snugly accommodate the implant. After creation of the subglandular plane, the lateral border of the pectoralis major muscle is identified. The muscle edge can be lifted by forceps to allow easy entry into the submusculofascial plane. This plane is readily identified by the wispy areolar connective tissue and ease of dissection. An extended electrocautery instrument is used to complete the dissection. The inferior origin of the pectoralis major is released from lateral to medial at the level of the IMF. Division of the pectoralis continues medially to the sternal border. Partial deep division may selectively be carried out 1–3 cm above the xiphoid, depending on which implant is used. Lateral dissection is avoided in order to let the implant push the pocket out itself and to reduce the risk of injury to the lateral neurovascular bundles.

The nerves can be stretched to accommodate the implant but should be preserved to minimize postoperative sensory changes.

When the pectoralis major muscle is elevated, care must be taken to leave the pectoralis minor down on the chest wall. This will minimize bleeding and allow correct placement of the implant. Once adequate haemostasis is obtained and pocket dimensions are finalized, the pocket is irrigated with an antibiotic-containing solution, and the implants are carefully placed by a minimal-touch technique and change of glove. Implant 'sizers' are avoided to reduce unnecessary pocket manipulations and possible contamination of it.

After the implants are in place, the patient is placed in a 90° upright position and evaluated from various perspectives. Any asymmetry or underdissected areas are marked, and the patient is placed back in the supine position. A multilayer closure is performed with absorbable suture. It is important to close off the implant pocket with a separate layer of suture before closing the skin. No drains are placed. Once closure is complete, Steri-Strips are applied along the direction of the incisions and a post-surgical bra is fitted (Figure 38.2).

Complications and treatment

There are inherent risks involved with all surgical procedures, and when they involve a prosthetic device these are considered more significant and have to be considered over longer follow-up time periods. Some may be considered trivial, others are devastating to both patient and surgeon. Regardless of estimated likelihood, these all should be discussed in detail with patients undergoing aesthetic surgery. They generally pertain to aesthetic outcome and surgical complications.

Aesthetic outcomes that are salient in the discussion with the patient include capsular contractures, malposition of implant, implant visibility/palpability/rippling, sensory and asymmetrical position changes to NAC, breast size asymmetry, contour deformities, galactorrhoea, skin stretch deformities, glandular atrophy over time and need for device change with passage of time.^{16,36,60,81,106,107} Surgical complications that need to be addressed include postoperative haematoma, device failure/rupture/rotation (anatomical implants)/infection, hypertrophic/keloid scarring as well as implant-related T-cell lymphoma.^{12,60,81,114,115}

Aesthetic and sensory outcomes

Capsular contractures

These are by far the most common reason for reoperation. Premarketing studies from Inamed,⁶⁰ Mentor⁵ and the Danish registry reports⁸¹ are among comprehensive studies addressing complications following breast augmentation. The topic of capsular contractures, the study outcomes and preventative strategies have been discussed at length in the previous section. During follow-up of 5373 women, the Danish registry study indicated that over a mean follow-up period of 3.8 years 4% of women underwent secondary surgery. Once the patient presents

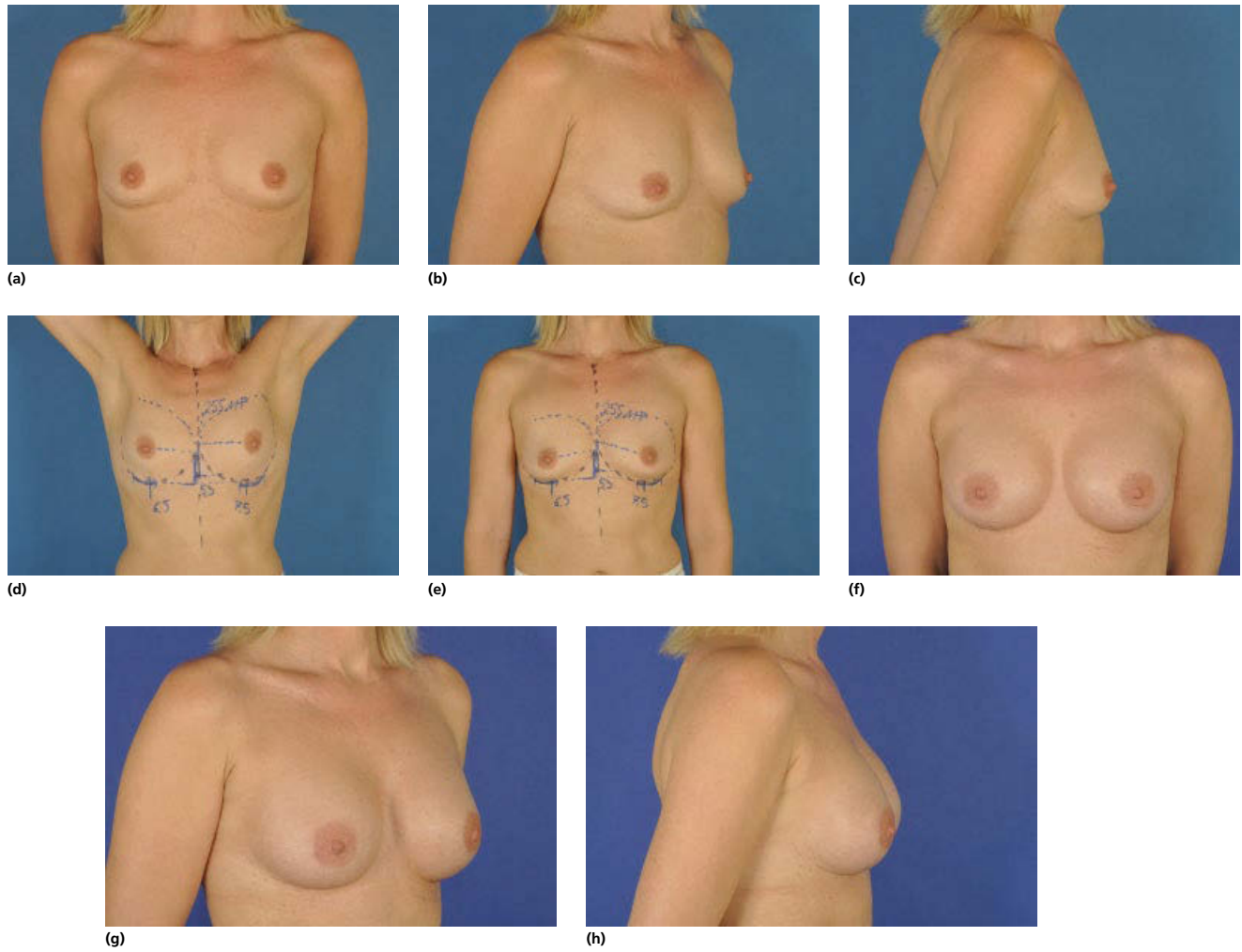


Figure 38.2 (a, b, c) Preoperative appearance antero-posterior, three quarters and lateral views. (d) Projected nipple-areolar complex after augmentation. (e) Relative markings at rest. (f, g, h) Postoperative views.

with a significant capsular contracture of Baker III/IV grades, the only option is surgery. This surgery can take the form of capsulotomy, scoring and device exchange alone, capsulectomy and device exchange or neo-pocket creation and device exchange. Considering the discussion in the previous sections about the cause of capsular contractures and the roles of bio-films, bacterial inoculation and contamination of the capsule, the authors feel that it is good surgical principle to excise the capsule where and when possible to create a new implant pocket and exchange the device.

More recently the use of acellular dermal matrix (ADM) has found a place in the debate on capsular contractures. Much like the concept of a 'fire break' skin graft in Dupuytren's disease, ADM has been found to prevent incorporation into the forming capsule. Histologic, basic science and clinical evidence indicate that placement of ADM yields an abrupt disruption of the forming capsule, thereby alleviating capsular contractures. The authors favour the use of ADM in revisional augmentation surgery.^{116–120}

Malposition is the second most common problem associated with breast augmentation and one of the most common reasons for revisional surgery. This occurs in about 1.2–1.6% of cases.^{81,83} The implant can be displaced in any direction – medially, laterally, superiorly or inferiorly – resulting in symmastia (when medial displacement is bilateral), fall into the axilla or a double-bubble deformity. Double-bubble deformity is defined in type I, the so-called 'waterfall deformity'; the implant sits superiorly to the breast and skin envelope that have 'slipped' over the implant with ptosis. This type is most commonly seen when complete submuscular pockets are used or when the breast is suffering from significant ptosis (grade III) prior to surgery and augmentation rather than augmentation mastopexy is performed. Type II deformity is related to incorrect lowering of the IMF during surgery, when the implant sits below the well-formed IMF and breast tissue.¹²¹

Malpositioned implants can be difficult to treat. In the lateral implant displacement, which is most commonly due to overdissection of the implant pocket or contraction of the pectoralis major muscle, the lateral part of the pocket can be excised and

closed or simply closed to tighten the pocket more medially. Use of ADM in all malposition revisional surgery has been described and must be considered in the planning stage.^{122,123}

Synmastia is notoriously difficult to treat. It is the result of over-dissection of the medial pocket and the sternal origins of the pectoralis major muscle in the submuscular planes.¹²⁴ Here the use of ADM can be most useful, the capsule can be used to augment the medial border of the pocket in addition to a layer of ADM placed medially. A reduction in implant size where possible is helpful.

Wrinkling/rippling

This is most commonly reported with the saline implants, particularly in textured subtypes. In a recent study this was reported to be observed in 37.5% of cases and was not related to underfilled, recommended filled or overfilled devices.¹²⁵ It is also more common when implant-to-parenchymal ratio is greater than 50%.⁶⁸

Asymmetries

All breasts are asymmetrical, so preoperative observation and documentation is good practice. The patient is made aware of these, which hopefully makes possible asymmetries as a result of the augmentation procedure more acceptable.

NAC sensory changes

As discussed previously, these are more common in periareolar approaches.¹⁰⁶ In the normal course of an inframammary or transaxillary approach, transient altered sensation may occur. Generally it is considered that permanent changes are more common when implants larger than 350 mL are used. Although Mofid and colleagues,¹⁰⁷ in assessing a small cohort of patients and controls using accurate measurements, argued that changes occur in all patients undergoing augmentation operations, in general the extent of this change is linear in relationship to the volume of the implants.

Surgical complications

Haematomas

These are not common – usually in the order of 1% risk⁸¹ – but they must be evacuated. Many authors have noted that persistent haematomas or significant seromas lead to capsular contractures.^{68,69}

Device failure

More recent generations of implants have proved to be more reliable than earlier versions. As noted previously saline implants now enjoy an approximately 4% deflation over the first 10 years.⁵⁹ The Inamed 6-year follow-up study of silicone implants reports a 3.5% device failure.⁶⁰

Infection and exposure

This is a dreaded complication for both patient and surgeon. The reported rates of infection are between 0.9% and 1.9%.^{5,81} The clinical picture presents along a spectrum. Traditionally, the

implants were removed and the patient was prescribed antibiotics. However recently, salvage procedures have become more common. These range from closed-system antibiotics irrigation to conservative wound care and passive drainage in addition to systemic antibiotics. The more aggressive end of the spectrum involves exchange of the implant, provision of coverage with muscle or using posterior capsule. Spear reports a very good salvage rate of infected or exposed implants with seemingly good medium-term follow-ups.¹¹⁵ However, given the discussion above pertaining to capsular contracture and presence of biofilms, the authors are of the opinion that in the long term these patients will inevitably develop capsular contractures. Except in the mildest cases the authors advocate capsulectomy and exchange of implant after the initial infection has been treated and the tissues have regained some of their suppleness.

Late seromas

Appearance of a late seroma in an augmented breast is a source of concern for both patient and surgeon alike. Its management despite flow charts and various reports^{126–128} becomes more a matter of surgical principles and judgement in the authors' view. It is ultimately a decision related to the cause of seroma. There are three main possibilities – idiopathic, infective or neoplastic – and each broadly requires a different approach. The 20 mL estimated volume on ultrasound remains the threshold for seroma formation that the authors consider.

Many authors treat seromas with antibiotics empirically and observe the clinical progression of the patient. We believe that if a patient presents with a new-onset late seroma, an ultrasound-guided diagnostic aspiration is mandatory. The fluids are all analysed for microscopy, culture and cytology with Wright Giesma-stained smears, for positive immunohistochemistry for CD30-positive cells, anaplastic lymphoma kinase protein, translocation studies and others indicated in consultation with the haematology/oncology team. Where the fluid is sterile, the idiopathic seroma may be an indication of a subclinical infection of the biofilm, which may not be picked up on microscopy or culture. In these cases if persistent and unresolved with a course of antibiotics, the authors advocate a capsulectomy and device exchange. Our assumption is that there is a subclinical biofilm infection, which will not be resolved with antibiotics treatment.

Where clinically mild, the infective seroma can often be treated with antibiotics. Although this may not lead to capsular contractures in the short term, this has to be weighed against long-term anticipation of capsular contracture as a consequence of subclinical biofilm infection. The decision as to when and if the device should be exchanged, along with a capsulectomy and where possible change of implant pocket, must be discussed and weighed against secondary surgery and its inherent risks as a revisional augmentation procedure. Where the seroma is of a malignant nature (anaplastic large T-cell lymphoma (ALCL)), the treatment option is referral to the breast and haematology/oncology teams. Removal of the implant and capsulectomy is part of the treatment regime.

Rotation

Rotation is not a problem with traditional implants. The anatomical implants have the advantage of filling the lower pole and are especially useful with patients with mild ptosis (grade II good skin envelope) who are not willing to undergo or not suitable for augmentation mastopexy. However, up to 1% of anatomical implants undergo rotation in their pockets which requires secondary corrective surgery.^{22,26,97,123}

T-Cell lymphoma

T-Cell lymphoma is a lymphoproliferative disorder that has been diagnosed in association with breast implants. Malignant cells have been isolated infiltrating the capsule as well as in the periprosthetic seroma fluid. Its immunohistochemical markers are uniform expression of CD30 protein, ALK, clonal T-cell rearrangement as well as the presence of t(2;5) translocation.¹²⁹ Treatment has varied from removal of implant with or without capsulectomy, followed by either standard chemotherapy or local radiotherapy.³⁴

This is considered an association as the statistical analysis along with anatomical site suggest an absolute increase in the risk of developing ALCL with breast augmentation. However, clear pathogenesis theories have not been put forward.

Scar formation

Keloid and hypertrophic scar formation need to be discussed with patients preoperatively. Their treatment is part of the standard algorithm discussed elsewhere in this book.

Autologous fat injection

Fat grafts have become a popular means of soft tissue augmentation in plastic surgery in recent years. Traditionally, concerns regarding survival and calcification had led to a ban of the procedure by the American Society of Plastic Surgery. However, this has changed in recent years with better understanding of fat and stem cells as well as better techniques for harvesting and engrafting.¹³⁰ With regard to meaningful breast augmentation, however, fat-based autologous augmentation, despite its obvious attraction, remains a goal rather than an attainable outcome.

One of the more novel techniques developed so far involves the BRAVA-based technique. This device, which has been on the market for 10 years, is an external tissue expansion device acting through suction. The resulting short-term tissue expansion, according to its developer and proponent, results in an expanded fibrovascular scaffold, which is fertile ground for fat engraftment.¹³¹

The most significant study of breast augmentation involving the BRAVA system was a multicentre study of 81 patients, which showed promising results both visually as well as in reported data. A mean augmentation of 233 mL is reported at 12 months based on 3D calculations. The patients had a baseline MRI

preoperatively, a three-month and a second follow-up MRI at least six months postoperatively. The key findings were an approximately 90% estimated graft survival and a stable volume between the first and second postoperative MRI, indicating permanency.¹³¹

The authors have no direct experience of the BRAVA technique, but they routinely use fat for contour deformity correction, especially in reconstructive cases. Larger controlled augmentation volumes remain, for now, in the realm of promising trials.

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CHAPTER 39

Breast reconstruction

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Introduction

The presence of breasts is a defining feature of adult mammalian gender. It must therefore be considered an essential element of human femininity, and the role it plays in female psychology cannot be understated.^{1,2} The requirement for reconstruction of the female breast is almost exclusively consequent on breast surgery for breast cancer.

Providers are becoming legally bound to educate and prepare patients who require mastectomy for reconstruction, such that informed choices can be made. They are obliged to offer a multidisciplinary setting as part of a care pathway, in particular allowing for cross-specialty engagement to allow for immediate reconstruction. The surgery of reconstruction has become a recognized specialist area, which by virtue of its appreciation of three-dimensional form, and its possible call on microsurgical skills, naturally falls within the domain and skill set of the plastic surgeon.

In this chapter the techniques of breast reconstruction and the principles behind them are reviewed, and it is hoped this will offer the practitioner a ready reference for what we trust will be a meticulously if not passionately executed endeavour.

Reconstructive aims

Any attempt at reconstruction should appreciate the nuances of breast form, volume and its complex three dimensionality. Its position on the chest wall is determined by its footprint,^{3–5} a fixed entity that does not change with age, and that is set by the inframammary fold (IMF) at the 6–7th rib inferiorly, the 2nd rib superiorly, 1–2 cm behind the anterior axillary line laterally, and the lateral border of the sternum medially. The footprint sets the basic breast position on the chest, and by complementing the opposite side creates the necessary foundation for symmetry.

The breast conus represents the tissue that projects from the chest wall, a complex of skin envelope with its glandular and fatty content that falls inferiorly to a variable extent over the

IMF in a manner termed ptosis. Unlike the footprint, the conus is variable with age. Creating, therefore, a symmetrical ptotic conus precisely positioned on the breast footprint embodies the art of breast reconstructive sculpture. The skin envelope aids in determining shape and any damage or removal of skin will influence its final shape. The ultimate goal of breast reconstruction is to create symmetrical, natural-looking breasts and not necessarily a specific shape or volume.

Aesthetic and functional considerations

Breast ideals have changed over time, as depicted in art and reflecting past and existing cultural differences. There is also a potential variation between what the surgeon and the patient perceives as aesthetic. For this reason it is critical to be attentive to the patient's wishes, appreciating that simple breast symmetry rather than an idealized form may well be their preference.

However, insofar as it is possible to specify what an ideal breast should be, the following can be stated:⁶

- Breast size relates to the size and shape of the torso.
- Nipple placement is critical – ideally 19–24 cm from the suprasternal notch. The nipple is centralized over the breast mound with a ratio of volume above the nipple to below the nipple of 45:55. The nipple should ideally be oriented vertically at a 20° angle.
- The majority of breast volume should be in the lower outer quadrant – following the natural gravitational vector, with the minority in the upper inner quadrant. The line from the nipple to the clavicle, on lateral view, should be straight or slightly concave. The angle at the IMF should preferably not be too sharply defined but become sharper with age, as volume increases and therefore gravitational pull increases. The angle between the breast mound and thorax at the medial and superior border of the footprint is very obtuse. Sharp angles here create an artificial 'implant' look. The angle at the lateral border is slightly sharper but never exceeds about 60° in standing position.

- A critical measurement of the aesthetic breast is the line from the inferior margin of the areola to the IMF. This should be somewhere between 6 and 8 cm, depending on the breast volume. Any such excess of tissue below the nipple creates a 'bottoming out' appearance.
- It is not possible to create a functionally lactating breast, nor one that is necessarily erogenously responsive. However, the reconstruction should not be functionally disadvantageous. It should be pain free, without any disabling donor-site complications, protectively sensate, and have a tissue consistency and movement similar to that of normal breast tissue.

Oncology

Breast cancer is now considered to be the most common invasive cancer, and is clearly a global phenomenon of almost epidemic proportions. In the United States, approximately 232 340 new cases of female invasive breast cancer are predicted to occur in 2013.⁷ It accounts for 29% of all cancers in women and is second only to lung cancer as a cause of cancer deaths. In terms of incidence, one in nine women who reach the age of 85 in the US will develop breast cancer.⁸ Rates have steadily increased by around 1–2% per annum from the 1980s, although that has been explained by the widespread use of mammography screening. The impact on healthcare systems remains immense for treatment as well as restoration.

Risk factors

Of all risk factors, the primary one is that of gender. The additional X chromosome exposes breast tissue to the cyclical fluctuating influence of powerful hormones, with constantly changing reproductive stimuli. Any decrease of the number of cycles theoretically reduces the proliferation of breast cells and thus the risk of breast cancer. Age >30 years old at first full-term pregnancy, nulliparity, early onset of menses <12 years old, and late age at menopause beyond 55 years old are related to increased risk of breast cancer.⁹

Of equal importance to female gender is the risk factor of age. Half of all cases occur in women older than 65. In 2004–2008 95% of newly diagnosed breast cancers occurred in women 40 years old and older.¹⁰ In fact the curve is bimodal, with the first peak at 50 years and the second at 70 years.¹¹

Familial factors are associated in 20% of cases with a relative diagnosed with the disease. The risk is 4 times higher if a mother or sister is affected, and 5 times higher if two or more first-degree relatives have been diagnosed with breast cancer.

Hereditary breast cancer accounts for 5–10% of the cases.¹² In 1990 the genetic susceptibility to cancer was attributed to two specific loci, those being tumour suppressor genes or in this case *BRCA* genes. They characteristically repair damaged DNA, and reduce the likelihood of cells proliferating excessively. Mutations in the *BRCA* complex (1 and 2) were able to be identified through blood tests and genetic profiling.

Women with *BRCA1* mutations are estimated to have a 44–78% risk for developing breast cancer by the age of 70; the corresponding risk for *BRCA2* mutations is 31–56%.¹³ These women are also at increased risk of developing ovarian cancer. It is now possible to test patients with a strong family history in order to counsel them on their own susceptibility. Their assessment, follow-up and treatment should take place in the context of a multidisciplinary team composed of oncologists, radiologists, general surgeons, gynaecologists, pathologists and plastic surgeons. Once the diagnosis has been established, some of these women will opt for prophylactic bilateral mastectomies rather than chemoprevention and aggressive follow-up. Bilateral prophylactic mastectomy reduces the risk of breast cancer in these patients by 90%.^{14,15} It is of great importance to be aided into an informed decision with regard to the appropriate type of reconstruction for them.

The greater prevalence of breast cancer in developed nations suggests lifestyle factors play a significant part. Rates in the developing world such as the Far East, in Eastern and Central Europe are rising rapidly. Within the developed nations, prevalence is lower in areas of social deprivation as many of the risk factors prevail among more affluent populations.^{16,17} Within the same context, obesity increases the risk for postmenopausal breast cancer¹⁸ and alcohol consumption has a dose-related effect.¹⁹

Extensive research has taken place to establish the relationship between increased risk of breast cancer and hormone replacement treatment (HRT), which involves combination of oestrogen and progesterone for women with postmenopausal symptoms. Women on HRT have an increased risk of developing breast cancer and this risk is higher the sooner they start or the longer they continue the treatment.^{20,21} Last but not least, significant exposure to radiation at different ages also appears to increase the risk of breast cancer.

Pathophysiology

Most of the breast cancer tumours arise from ductal breast elements. Repetitive hormonal stimuli on the normal breast tissue, theoretically promote the malignant transformation of the ductal cells. Ductal carcinoma *in situ* (DCIS) is characterized by malignant cells confined into the borders of the basement membrane. Approximately 15–30% of newly diagnosed cancers are in the form of DCIS in women under a screening mammography programme. It is unknown which of those will progress into invasive carcinomas but there are histological parameters such as comedo necrosis, nuclear grading and solid growth that manifest a more malignant cytology and thus greater possibility of progression to an invasive tumour.²²

The most common histological types of breast cancer are infiltrating ductal carcinoma (75%), followed by lobular carcinoma *in situ* and infiltrating lobular carcinoma, which arise from the breast lobules. Other histological types are medullary (5%), which carries a favourable prognosis, mucinous or colloid (5%), tubular (1–2%), papillary (1–2%), metaplastic (<1%) and

mammary Paget's disease (1–4%). These have their own behavioural characteristics, which align with the presence or absence of oestrogen or progesterone receptors, or the overproduction of human epidermal growth factor receptor 2 (HER2).

Hormone and targeted treatment

Women who test positive on immunohistochemistry staining for oestrogen hormone receptors are amenable to hormone therapy such as tamoxifen, which is an antagonist of oestrogen receptors and prevents oestrogen from binding and stimulating cancer cells. When administered as adjuvant treatment it reduces the risk of recurrence and death.^{23,24} In addition, tamoxifen is proven to reduce the risk for breast cancer significantly when administered prophylactically to women with known high risk.²⁵

However, patients on tamoxifen treatment are at risk of venous thromboembolic events²⁶ and after communication with the oncology team they are advised to discontinue it approximately two weeks before a free flap reconstruction.

Another type of medication, used specifically for postmenopausal patients, with a cancer positive for hormone receptors, are the aromatase inhibitors (AIs). The AIs have the ability to inhibit the enzyme responsible for the production of oestrogens in the periphery from adrenal gland precursors. They have been found to be of benefit in the treatment of early as well as advanced breast cancers.^{27,28}

Approximately 15–30% of the breast cancers overproduce human epidermal growth factor receptor 2 (HER2). Trastuzumab (Herceptin) is a monoclonal antibody that targets the HER2 protein of breast tumours and it offers a survival benefit in metastatic cancer.²⁹ In addition, Herceptin, when administered in conjunction with chemotherapy for early-stage breast cancer, reduces the risk of recurrence by 52% and death by 33%, compared to chemotherapy alone.³⁰

Trastuzumab has been related to cardiotoxicity manifested as decreased left ventricular function and cardiac monitoring is advised for patients treated with Herceptin.³¹

Chemotherapy

The surgical and medical treatment of breast cancer is individualized. Factors that are taken into consideration in the administration of adjuvant chemotherapy are the size of the invasive component of the primary tumour, the nodal status, hormone receptor expression and HER2/neu expression. Histologic parameters that are taken into consideration as well are the lymphovascular invasion and histologic grading.

Adjuvant chemotherapy is administered for 3–6 months and it includes a combination of different medication. It has been successful in demonstrating reduction in recurrence rates and improvement of the survival outcome.³² An overview of the randomized trials on administration of adjuvant chemotherapy in early-stage breast cancer showed a substantial reduction of 5-year recurrence rate and 15-year mortality rate regardless of the oestrogen receptor status of the primary cancer.²⁴

The implications of chemotherapy on reconstruction have been comprehensively reviewed in a meta-analysis by the American Society of Plastic Surgeons. There is no consistent finding that preoperative chemotherapy is a significant risk factor for complications in expander/implant reconstructions. It is advised that adjuvant chemotherapy should be completed 4–6 weeks prior to reconstructive surgery, and that adequate white cell level should be confirmed preoperatively. There is no definite data on effects of chemotherapy on autologous reconstruction.^{33,34}

Prognosis

Prognostic factors include the status of axillary lymph nodes (number of nodes involved), tumour size, lymphatic or vascular invasion, age, histological grade, histological subtype, response to adjuvant treatment, oestrogen/progesterone receptor status and *HER2* gene amplification.

The key objective in breast cancer treatment remains that of complete local cancer tissue removal such as to prevent recurrence or wider spread, and to treat more widespread disease. Complete surgical excision with negative margins remains the principal method of treatment and can be curative in patients with early disease, with adjuvant therapy (chemotherapy and radiotherapy) used to treat micrometastases.

Oncological-surgical treatment

Mastectomy

Breast cancer surgery has its origins in an aggressive technique developed by Halsted in 1882, without reconstruction in mind, and with extensive tissue clearance and disfiguring chest wall scarring. This *en bloc* removal of all breast tissue and pectoralis major muscle, including the axillary nodes (and/or internal mammary lymph nodes), was modified by Patey to a procedure where the pectoralis major muscle was spared. A 'simple' or 'total' mastectomy is one where all breast tissue including the skin and nipple areolar complex (NAC) is removed, possibly accompanied by the ipsilateral axillary lymphatics.

In modern times, the appreciation of the wider and complex ramifications of breast cancer treatment has brought with it the now universal acceptance that the disease should be managed by an oncological team. The disfiguring consequences of the surgery, and the development of surgical techniques to reduce this, has prompted cancer surgery to evolve to facilitate reconstruction. Increasingly, the standard preservation of the IMF and superolateral fold, a useful residue of skin envelope, has been supplemented with preservation of even the areola or NAC (SSM or skin-sparing mastectomy) which confers a greater likelihood of achieving symmetry. Importantly, reviews have shown that a skin-sparing periareolar subcutaneous mastectomy does not increase the likelihood of local recurrence.^{35,36} The advantages of the SSM are the judicious use of native skin

with its preferential colour and texture match, the retention of the breast footprint and critical elements of the skin envelope. There may be less need for surgery to the contralateral breast, and a smaller reconstructive skin paddle may be required. Surgical judgement is required to ensure that the length and thickness of the undermined flaps are viable, as necrosis can compromise wound healing and result at worst in a failed reconstruction. If necessary, a Wise pattern is employed to tailor the skin flaps to an appropriately sized envelope. The reconstruction essentially involves the filling of a skin envelope with synthetic or autologous tissue, or both, and any NAC defect is replaced by a skin island from the flap.

The NAC has been shown to be free of cancer cells when there is no multifocal disease, the tumour is less than 2 cm diameter (T1), and there is greater than a 4 cm distance between the tumour and the NAC.³⁷ This therefore applies particularly to risk-reducing prophylactic mastectomy. The mastectomy may then be areola sparing, or nipple and areola sparing. The nipple itself can be removed together with the mammary gland in one single specimen. The skin of a flap that reconstructs the breast mound can later be used for the nipple reconstruction together with re-approximation of the areola.³⁸ Results of a retrospective study also show that a nipple-sparing procedure may be used in relevant clinical circumstances.³⁹

Studies have now shown that 'lumpectomy' or partial mastectomy (breast conserving surgery) with adjuvant radiotherapy is comparable to modified radical surgery not only in survival terms, but also in local recurrence rates.⁴⁰ In early cases, therefore, therapeutic mammoplasty with radiotherapy is used, where after a local excision varying local reconstructive patterns are employed to reform the breast to an aesthetic shape, with or without contralateral surgery.

The axilla, axillary dissection and sentinel lymph node biopsy

Approximately 80% of the breast's lymphatic drainage is through the axilla and the axilla is the commonest site of metastasis in breast carcinoma. Axillary node status is one of the critical indicators of prognosis. Until recently, identification of axillary lymph node spread was through clinical examination and axillary node sampling at levels 1 and 2 (axillary nodes below subclavicular vessels). More recently, at clinical assessment, the routine use of axillary ultrasound and fine needle aspiration has become commonplace. With nodal positivity, wider axillary clearance was then usually considered necessary. However the inevitable consequence of this was chronic brachial lymphoedema, possibly with chronic pain, paraesthesia, recurrent infections and seroma.

The identification preoperatively or indeed intraoperatively of sentinel nodes with contrast injections (vital dyes, radioactive tracers or combinations of the two) into the peritumour area has been introduced into the clinical pathway. These nodes are the first nodes or groups of nodes that drain from the breast to the axilla.⁴¹ If the biopsied sentinel node is disease free, no axillary

dissection is performed.^{42–45} Hereby, through a minimally invasive procedure the cancer can be staged in patients who have clinically negative nodes and who have T1 to T2 tumours.

As definitive therapy, studies have shown cancer outcomes for both approaches – axillary dissection and sentinel node biopsy with axillary sparing, similarly matched for disease free survival and local recurrence.^{46–49} Any adjunctive therapy may have been at the oncologist's discretion, and the use of axillary radiotherapy as an axillary-sparing modality has been shown also to be clinically justified.⁵¹

Radiotherapy

The breast and radiation therapy

Radiation therapy has an established role in the therapeutic management of locally advanced breast cancer in reducing the local recurrence and providing a clear overall survival advantage. This has been well established by multiple trials.^{52–54} The American Society for Therapeutic Radiology and Oncology recommend postmastectomy radiation therapy for breast cancer patients with advanced disease (T3 or T4 tumours) or at least four positive axillary nodes. The role of radiation therapy in patients with T1 or T2 tumours and one to three positive axillary nodes⁵² remains questionable. Additional to this is the controversy about which would be the correct reconstructive method for patients who received radiation.

Radiation pathophysiology and effects on tissues

There is a range of effects that can occur in irradiated skin over time. In general, radiation can damage cells by directly disrupting DNA or through free radical production. This damage results in disorganized fibroblast activity, microvascular thrombosis and inadequate tissue oxygenation. During the 3rd to 6th week of therapy, populations of basal layer stem cells become depleted in the treated area. The gross skin changes occurring with standard schedules and doses of radiotherapy to the breast are categorized as early or late effects, as determined by the time at which they present. This is covered in more detail elsewhere, but a brief account of salient features in breast treatment are discussed below.

Early effects are defined as those that occur within 90 days of the first radiation therapy. The skin reactions include dryness, epilation, pigmentation changes and erythema. Dryness and epilation are results from destruction of the dermal layer. Hyperpigmentation is a result of epidermal melanocyte stimulation. Some patients may develop hypopigmentation due to complete eradication of all melanocytes. Acute erythema is caused by cytokine-mediated inflammatory reactions.

Late effects are considered the effects that present after 90 days from the first radiotherapy. These effects are related to dermal injury. Until the moment that late effects appear the skin can appear 'normal'. Late effects can appear after months or even

up to 15 years later. These late effects are directly related to the dermal fibroblast response to radiotherapy and the resorption of collagen fibres. Telangiectasia is another effect of radiation that can manifest months or multiple years after the therapy. If necrosis appears this is associated with doses of radiation higher than those used in breast cancer treatment.^{55,56}

Patients who underwent lumpectomy and irradiation are also predisposed to an increased incidence of complications, although the extent of radiation damage may be more limited. Recent research has shown that even when a three-beam radiation has been used (a modern radiation technique), the complication rate remains high in comparison with the non-irradiated breast.⁵⁷

Radiotherapy and breast reconstruction

Evidence indicates that radiation therapy, regardless of when it is administered (before reconstruction or on a reconstructed breast), is associated with an increased risk of complications and/or reconstructive failure in patients undergoing postmastectomy expander/implant breast reconstruction. A prospective cohort study, which controlled for confounding factors in multivariate logistic regression analysis, demonstrated that postoperative radiation therapy to an implant was associated with a six-fold increase in risk of complications compared with no radiation therapy. Patients who received postoperative radiation therapy to an implant were also 5.1 times more likely than patients who received no radiation therapy to experience reconstructive failure.⁵⁸ Additionally, a 5-year follow-up retrospective cohort study found that implant patients with radiation had a 61% total complication rate compared to only 21% among patients without radiation.⁵⁹

Patients should be counselled regarding these associated complications. Complications may include early and severe capsular contracture, infections, implant displacement, skin erosion and perforation followed by implant exposure, rippling, seroma, haematoma and wound-healing problems.

Autologous breast reconstruction in an irradiated area can be performed without major risks or complications. Recipient vessels within the irradiated field are surrounded by an increased amount of fibrosis but can still be used. If there is doubt, recipient vessels from outside the irradiated field can be used. Soft tissues that have become too fibrotic or damaged by irradiation, are removed and replaced by soft, pliable and healthy tissues of the flap.

If irradiation is to be performed after ablation and reconstruction, we now recommend against performing a primary autologous reconstruction. Even though the side effects of radiotherapy on pedicled or free flaps are minimal and severe complications are rarely seen, we feel it is safer to temporarily introduce an implant or expander (depending on the amount of skin deficiency) into the mastectomy pocket to keep the skin expanded during adjuvant radiotherapy. Expanders can be deflated or inflated during the radiotherapy sessions according to the available devices and to the guidance of the radiotherapist. Once the adjuvant treatment is completed, a

delayed-primary reconstruction can be performed by autologous means by filling up the existing pocket. This way the flap is never directly irradiated, avoiding fibrosis and long-term shape and volume changes.

Implants/tissue expansion/acellular dermal matrix

Alloplastics – synthetics (implants) with acellular dermal matrix/tissue expansion

Introduction

The placement of a suitable implant under the remaining tissues on the chest wall after mastectomy would seem logically to be the simplest and indeed cost-effective way to create a breast mound. In the United States, this is the commonest type (of the order of 67%) of breast reconstruction.^{60,61} The reconstruction may involve a primary implantation, as a staged procedure with the use of a tissue expander, and with possibly further bulking or implant protection using a flap (such as the latissimus dorsi muscle with or without skin). It should be performed with an appreciation of its limitations; it is difficult if not impossible to create a ptotic breast or to match a large breast, and the best results are usually obtained with bilateral procedures. Implants carry with them specific complications, the most important being capsular contracture, which becomes more likely when used with radiotherapy. Implant failure and silicone leakage with silicone gel-filled implants are also relevant.

Technically, the implant comes to rest on the chest wall and pectoralis minor muscle, to a great extent being covered by the pectoralis major muscle (thereby providing partial robust soft tissue cover). However, the lower pole of the implant is covered only by skin and subcutaneous tissue, which renders it at risk for extrusion and failure. Solutions such as the serratus anterior coverage may blunt the IMF and result in a suboptimal shape. The use of a buried dermal flap of the breast inferior pole has been described.⁶² There has been recent promise in the use of an additional biologically engineered tissue layer over the relatively poorly covered lower pole of the implant, and for this acellular dermal matrix (ADM) has found a place in potentially improving outcomes.

Current implant materials

The first attempts to create or augment a breast mound with synthetic material began in the 1960s with silicone implants. This is covered in more detail in the breast augmentation chapter. Briefly, the implant that Cronin and Gerow developed consisted of a teardrop-shape silicone rubber envelope filled with liquid silicone.⁶³ On the back of the implant a Dacron patch was placed to prevent rotation. About 50 years were needed to develop the fifth-generation implants used today. Contemporary models are based on the characteristics of the filler, the shell, the shape and the surface configuration.

The two main types of breast implant in use today are silicone and saline filled. Regardless of the content of the implant, the

shell is made up of silicone elastomer. The majority of implants used today are silicone filled and this is due to their superior feel and durability. In the Unites States, saline-filled implants have been used because of concerns voiced in the early 1990s and the moratorium on the use of silicone implants between 1992 and 2006.^{64–66} The main reason for this was the suspected complication of systemic autoimmune disease. Worldwide studies consisting of over 25 high-level scientific studies and over 10 meta-analyses have failed to prove any such link.

Medical-grade silicone has the advantage of being one of the least bioreactive materials available for medical devices. It consists of mixtures of various chain lengths. The current fifth-generation cohesive gel, developed since the mid-1990s, is a result of the extensive cross-linking of the silicone polymer, conferring properties of greater memory and shape retention. These implants are described as form-stable, offering a consistent anatomical breast shape. They also reduce the likelihood of rippling, and folding or collapse in the upper pole. In the unlikely instance of rupture, they will not leak and give rise to silicone migration to elsewhere in the body.^{67,68} These implants have now been developed with a variety of dimensional options in relation to base-width, projection and height, all of which can be assessed and applied at the surgeon's discretion in order to optimize symmetry and the aesthetic outcome.

Attempts have been made to replace the contents of silicone implants, particularly when the health risks of silicone gel remained under question. Soya-filled (trilucent) implants appeared on the market for a short time, as did various hydrogels (Poly Implant Prostheses (PIP)) and a pure form of triacylglycerides. These two formulations were withdrawn from the market because of the inability to demonstrate safety data.^{69,70} Public confidence in silicone implants was once again undermined when poorly engineered implants with an unacceptably high failure rate and inferiorly graded silicone (PIP) were allowed medical certification.⁷¹ Under scrutiny is also an association between silicone implants and anaplastic large cell lymphoma, although to date the number of cases remain very few and any link has by no means been formally established.^{72,73}

Implant shell

The first implant shell, developed by Cronin and Gerow, was smooth surfaced and relatively fragile, and was related to higher rates of failure and capsule formation. A second technological development arose a few years later, where a polyurethane foam coating created a rough porous surface with an open cell structure. This allegedly prevented circumferential collagen deposition, thereby reducing capsular contracture and lowering rupture rates. In addition, they disguise or at least reduce rippling, and their tendency to adhere to tissues to reduce 'bottoming out' also made them potentially appealing alternatives. Polyurethane devices were briefly discontinued because of the effect of a polyurethane by-product known as 2,4-toluenediamine (TDA). It was identified as a carcinogen in rodents with unknown potential risks in humans. The US Food and Drug

Administration (FDA) has concluded that TDA-induced breast cancer was an infinitesimal health risk to women with such breast implants.^{74,75} Polyurethane-coated implants have now been reintroduced, and are favoured as a replacement implant where capsular contracture exists, or indeed even as a primary option.

Risk factors for implant and expander-based reconstruction

The American Society of Plastic Surgeons has published a comprehensive set of evidence-based guidelines for the use of implants and expanders in breast reconstruction.⁷⁶ Risk factors have been identified and weighted according to rigorous meta-analysis. These include infection, haematoma, seroma, wound dehiscence, skin flap necrosis, expander/implant deflation, capsular contracture, hypertrophic or keloid scarring, and venous thromboembolism disease. The strongest weighting, and therefore the risk factors that deserve the most caution, are smoking and obesity. Less weighted but nevertheless of significance is postoperative radiotherapy to both implants and to expanders. Diabetes and preoperative radiation are less significant, as are breast size, and hormonal therapy or chemotherapy.

Capsular contracture

The spontaneous elaboration of a deforming layer of scar tissue around an implant has been a confounding complication ever since silicone implants were introduced. Shell texturing, and lately the reintroduction of a polyurethane coating, has had a significant impact on reducing its incidence. The Benediktsson and Perbeck study⁷⁷ evaluated rates of capsular contraction (Baker classification, Table 39.1) around saline-filled textured implants, finding that the rate of capsular contraction was significantly higher for irradiated than for non-irradiated breasts. The difference in contracture rates was not evident during the first six months but was highly significant thereafter, even 5 years later. This has resulted in reconstructive strategies such as staged definitive implantation when possible adjuvant radiotherapy is anticipated (the 'delayed-immediate' approach).

Tissue expansion

The introduction of tissue expansion in 1982 by Radovan paved the way for breast skin enlargement in circumstances where skin cover might be in short supply.⁷⁸ The relative rigidity of the ribcage provides a suitably firm base for the expansion. With serial episodes of saline inflation of the silicone balloon to a point of overexpansion advised at 30–35%,⁷⁹ extra skin may be created to allow for a larger implant, if not to help to create

Table 39.1 Baker capsular contracture classification

Grade I	The breast is normally soft and appears natural in size and shape
Grade II	The breast is a little firm, but appears normal
Grade III	The breast is firm and appears abnormal
Grade IV	The breast is hard, painful to the touch and appears abnormal

ptosis. In general, it would seem that anatomically shaped, textured expanders with an integrated port are preferred. The shaped or bi-dimensional expander allows for preferential expansion of the lower pole.^{80,81} However, this necessitates at least a two-staged procedure, which the Becker double-lumen implant (a combination of saline expander and silicone implant) has endeavoured to reduce to one stage.⁸² Although the expanded skin might allow for a larger implant, however, a key disadvantage has been the inability to create significant ptosis. This seemed only feasible with the importation of additional tissue, such as with a latissimus dorsi flap.

The apparent suitability of ADM in these procedures, however, has added further support for implant placement without the need for flap reconstruction. Tissue expansion under the collagen scaffold of ADM has now found a definite place in the attempts to create an implanted ptotic breast. However, an irradiated chest is considered a relative but not absolute contraindication to tissue expansion,⁸³ with the tissue being less pliable, rendering expansion difficult and unpredictable. This results in less favourable outcomes, with more asymmetry and less projection in comparison to non-irradiated controls.

Acellular dermal matrix

Subpectoral implant placement following subcutaneous mastectomy leaves the lower pole covered by a relatively thin layer of skin and subcutaneous tissue. This can prove to be a weak support for an implant, prone to poor radiation tolerance, and with a potential for breakdown and implant exposure. It can attenuate the IMF, and allow superior migration of the inferior edge of the pectoralis muscle.⁸⁴ The raising of local muscle tissue such as rectus abdominis/serratus anterior serves only to potentially increase morbidity and deform breast shape by restricting lower pole expansion.⁸⁵

This problem has originally been addressed by the placement of an allogenic dermal sublayer attached to the inferior pectoral muscle edge superiorly, and the IMF inferiorly, and the serratus anterior muscle laterally.^{86–88} ADM is a cell-depleted biological tissue layer, usually of human or porcine dermal origin, which biologically integrates through revascularization into the tissues into which it is placed. In doing so it adds a permanent collagen scaffold or hammock to the inferior pole of the reconstructed breast. As such it allows for a greater volumetric increase and limited degree of breast ptosis, thereby potentially removing the need for a contralateral procedure. There have been improved results in terms of appearance, stability of implant position, ptosis, reduced rippling and radiotherapy tolerance, with decreased capsular contracture rates.^{84–86} The attractiveness is strengthened by there being no donor site morbidity and scar, with a single scar limited to the breast, a reconstruction with local compatible tissue, and with no preclusion of a further surgical procedure (such as autologous reconstruction) if required. Contrarily, and besides being expensive, there have been reports of greater incidences of infection, seroma and skin necrosis,

although not sufficient as to contraindicate its use.⁸⁹ Particular caution has to be exercised in attempting to reconstruct a large breast (i.e. greater than 600 g).⁹⁰ The American Society of Plastic Surgeons guidelines on the use of ADM recognize the increased reported complications in many studies, but also appreciate its potential benefits in selected cases. They advise therefore that it is subject to the clinician's discretion.⁹¹

Immediate versus delayed reconstruction

There would logically appear to be a benefit in patients having breast reconstruction as a delayed second procedure some time after their cancer surgery, in order to be more certain of the cancer clearance and to allow for adjuvant therapy. Surgeons may therefore be inclined to prefer this.⁹² It would shorten their operative time, and allow for patients to adjust to life without a breast, and give time for consideration of the reconstructive options and their risks. Immediate reconstruction, however, allows for a one-stage procedure and facilitates reconstruction when much of the breast skin envelope, and the IMF, can be preserved.

Studies have shown there to be no increased risk from an oncological standpoint, however.⁹³ An increase in complications was identified, in particular wound infections,⁹⁴ but there was a definite patient preference for, and demonstrated psychological benefit from, immediate reconstruction.^{95–97}

Nevertheless, immediate reconstruction carries with it service implications, including greater interspecialty collaborative working. Current guidelines from the American Board of Plastic Surgeons are that, in the light of incomplete evidence, the timing of reconstruction should be on an individual basis.⁷⁶

Where nodal status and indeed adequacy of resection proved uncertain, and where adjuvant radiotherapy might be expected, the M.D. Anderson Centre in Houston pioneered the concept of 'delayed-immediate' reconstruction, whereby the immediate procedure simply involved the submuscular placement of an underfilled expander. This is in view of the greater incidence of complications encountered in radiation to reconstructed breasts (whether implant-based or autologous). Here it was felt logical to perform at least a preparatory immediate procedure that might optimize later reconstruction. This would allow for later expansion once the adjuvant radiotherapy was completed, or facilitate autologous reconstruction by maintaining a suitably placed subcutaneous cavity that might accommodate a definitive soft tissue transfer at a later stage.⁹⁸

Latissimus dorsi

Historically, the latissimus dorsi musculocutaneous flap can be considered the forerunner of breast flap reconstruction, having been described originally in 1906 by Tansini,⁹⁹ with resurgence in the 1970s when Bostwick¹⁰⁰ introduced a skin island to the flap.

The proximity of its robust vascular pedicle (the thoracodorsal artery and venae comitantes) and the ready availability of suitable soft tissue placed the latissimus dorsi musculocutaneous flap in a pre-eminent position for the creation of a breast mound during the last three decades of the twentieth century. However, the volume achieved with the flap may be limited due to insufficient tissue bulk and an implant or fat grafting is sometimes required. When used with an implant it can reduce capsular contracture and rippling.

The donor site morbidity with regard to loss of function and scarring is a matter of debate in plastic surgery literature. Some studies have shown that the loss of a latissimus dorsi muscle does little to limit function¹⁰¹ and the scar can be placed transversely, to be concealed within a bra strap, or can be a short oblique scar in the relaxed skin tension lines. When extended latissimus dorsi flaps are harvested, the contour deformity of the back can be more conspicuous.

Relative technical ease and reduced operation times have made it stand the test of time. This flap is considered an alternative option when the abdomen, lumbar and gluteal areas are scarred or otherwise unsuitable as a donor choice.¹⁰²⁻¹⁰⁴

Technique

The patient is positioned in a lateral decubitus position. The preoperatively designed skin paddle is incised until the muscle fascia. The skin around the skin paddle is undermined over the muscle so that the muscle can be mobilized. Flap elevation is easier by identifying the anterior margin of the muscle. The vascular pedicle runs underneath the latissimus dorsi muscle. The thoracodorsal artery enters the latissimus dorsi muscle after giving a branch to the serratus anterior muscle. This branch should be preserved. The vessels then split into a descending and transverse branch, allowing the surgeon to harvest only the antero-lateral strip of muscle if a limited reconstruction is required.

A subcutaneous tunnel is then created and the flap is transferred without tension in its recipient site. To achieve maximal anterior transposition, the tendinous insertion can be released. It is a consideration to release only 90% so that inadvertent traction of the pedicle will be prevented.^{105,106}

Decisions regarding division of the thoracodorsal nerve remain controversial and tend to be at the surgeons discretion. Transection of the motor nerve can be performed immediately. This will lead to progressive loss of volume of the breast but will avoid inappropriate contraction of the muscle and unwanted displacement of the (breast and) implant. If left intact, it maintains the greatest muscle volume by reducing atrophy.

The flap has undergone modifications. To add further bulk, additional fat can be harvested deep to Scarpa's fascia over the muscle in the lumbar or the scapular area, leaving only 1-cm-thick overlying skin flaps. A fleur-de-lys skin

island can also be taken to maximize the skin flap area. In addition, a muscle-sparing technique of the perforator-based thoracodorsal artery perforator (TDAP) flap is used for more limited mastectomy defects, mainly in the lateral quadrants of the breast.

Complications

The major disadvantage of combining a latissimus dorsi flap with an implant is that one combines the long-term morbidity of both techniques, mainly the creation of a significant scar on the back, potential loss of function and capsular contracture around an implant.

Flap necrosis is a possible complication. Problems with flap inset and wound healing are rare. Total flap loss is exceptional and mostly due to technical problems or coagulopathies. Partial flap loss is more frequent, especially in the most distant part of the flap (infero-medial at donor site) that ends up at the supero-medial part of the breast, especially in extended flaps. These issues are more frequent in patients who have undergone irradiation.

Recent studies from Saint-Cyr *et al.*¹⁰⁶ have shown that use of the pedicle descending branch muscle-sparing latissimus dorsi flap produces minimal donor site morbidity. The tendency to seroma formation in the undermined cavity has been drastically reduced by quilting sutures.¹⁰⁷

As with any wound that is closed under tension, the donor site may tend to form hypertrophic scars. Breast-shaping problems are often seen, mainly in the long run. Fullness of the sub-axillary region, where the proximal part of the muscle passes, is predominantly seen when the tendon has not been transected completely. For the same reason it is very difficult to create a well-defined lateral breast crease in a single procedure. Prolonged contraction of the latissimus dorsi muscle (especially when the latissimus dorsi motor nerve is left intact) will pull the breast and the underlying implant laterally resulting in asymmetry and insufficient definition and fullness of the medial breast mound.

The skin texture and thickness is very different from the breast skin and if incisions are not well positioned over the breast, a pincushion effect can disfigure the shape of the breast.

Pedicled transverse rectus abdominis myocutaneous flap

One of the most popular and most frequently performed autologous tissue flaps in breast reconstruction is the transverse rectus abdominis myocutaneous flap (TRAM flap). The flap was first described by Holmström,¹⁰⁸ was popularized in 1982 by Hartrampf *et al.*,¹⁰² and has been modified to minimize abdominal wall morbidity by harvesting only the muscle surrounding the perforators (the muscle-sparing TRAM flap).¹⁰⁹

The rectus abdominis muscle has a dual blood supply – the superior epigastric artery and the deep inferior epigastric artery. In the pedicled TRAM flap operation, the superior epigastric arterial system is utilized, with a horizontal lower abdominal skin paddle. Skin and subcutaneous tissue perfusion is via para-umbilical perforating vessels that originate from an intramuscular network nourished by the superior as well as the deep inferior epigastric vessels. The size and location of these vessels are variable. Venous drainage can be either from the comitant veins of the deep perforators or through the superficial system. The perforating arteries and veins supply a random cutaneous vascular network or plexus.

Historically, four perfusion zones have been recognized, with zone IV being the most distant part from the perforator vessels. Blood flow and, thereby, tissue survival will depend on antero-grade flow of arterial blood to the most distant parts and sufficient retrograde venous pressure to pump the blood back to the perforators or the superficial venous plexus. Blood pressure and cardiac output are therefore just as important to distant blood flow as vascular anatomy. The old concept of delineated zones is therefore considered historical and obsolete and should be abandoned.

As the pedicled TRAM flap relies on a very long pedicle that is subject to anatomical variations on top of unfavourable flow physiology, along with the possibility of a dominant superficial venous plexus, the incidence of partial necrosis is high. Attempts to augment vascularization by harvesting a second rectus muscle lead to an unacceptable high rate of donor site morbidity and should be avoided.

Technique

Technical details

A midline mark is made from above the umbilicus, through the TRAM skin island to the pubis. This line can be used as a guide in placing the midline of the upper abdominal flap to the midline of the pubic area when closing the abdominal donor site. The transverse skin island of the TRAM flap is marked over the lower abdomen and approximates the same tissue that is removed in a cosmetic abdominoplasty.

The flap can be designed on one or both rectus abdominis muscles, with most surgeons electing to perform a unilateral flap to minimize donor site morbidity. In cases of a unilateral reconstruction the contralateral muscle is used due to better flap rotation and manipulation. When a bilateral reconstruction is needed, both rectus abdominis muscles can be used with each muscle being used for the ipsilateral side. Donor site morbidity is once again relatively high.

A first incision is made along the upper edge of the skin island until the fascia of the rectus abdominis muscle is reached. The chosen muscle is identified to the level of the costal margin. The anterior rectus fascia is then incised medially and laterally over the muscle.

The inferior part of the flap is then incised to the level of the rectus muscle. After identifying the medial and the lateral

border of the muscle, the inferior margin of the rectus is divided with electrocautery and the entire posterior surface of rectus muscle is raised from the posterior sheet up to its costal margin attachments. At that point a costal vessel is ligated to give a more tension-free transposition.

Once the dissection is completed a subcutaneous interconnection (tunnel) must be created between the costal attachments of the rectus abdominis muscle and the mastectomy defect. Once the flap has been passed through the tunnel to the defect, the skin island must be checked for venous congestion and arterial perfusion. The most distal part of the skin paddle needs to be systematically removed to avoid high rates of partial flap necrosis.

The donor site defect is usually repaired with large non-absorbable sutures. Some surgeons recommend reinforcement of the fascial repair with a mesh overlay. This can, however, lead to additional complications of chronic discomfort or pain. The abdominal wall skin defect and replacement of the umbilicus is performed as in an aesthetic abdominoplasty.¹¹⁰

Pedicled or Free TRAM flap

When patient and surgeon have decided that the breast should be reconstructed with abdominal donor tissue, the surgeon then has to decide whether a pedicled or a free TRAM flap is appropriate for the clinical circumstances that exist.

The advantage of the free TRAM flap technique compared to its pedicled sibling is improved blood supply to the skin island by virtue of its vascular pedicle being the larger deep inferior epigastric artery. The free flap, therefore, is used in patients at higher risk for partial flap loss with the pedicled technique. Such high-risk patients include smokers, the obese and patients with prior abdominal surgery. Patients without these risk factors can be expected to achieve good results with either the pedicled or free flap technique.¹¹⁰

Complications

The most common complications are fat necrosis followed by infection, seroma, abdominal hernia, partial flap loss, and deep vein thrombosis.

Removal of the entire or a full-width part of the rectus abdominis muscle will lead to irreversible loss of abdominal wall competence and force, resulting in abdominal wall hernia or bulging, a decreased ability to perform daily activities in work and sports, decreased mobility, chronic abdominal wall pain and imbalanced muscle forces around the lumbar spine, resulting in chronic back pain. Complications are more frequent in patients who have undergone a bilateral TRAM flap.¹¹¹ The denervation of the rectus muscle will lead to loss of breast volume several months after surgery. Additionally, epigastric bulging is often seen as a result of subcutaneous tunnelling of the muscle, leading to poor definition of the medial IMF.

Free TRAM

The free TRAM flap involves the use of abdominal excessive tissue composed of skin, subcutaneous fat and variable amount of rectus abdominis muscle as a free tissue transfer mound. It is with no doubt the flap that has undergone the most extensive evolution throughout the last 30 years.

Since 1982, when it was popularized by Carl Hartrampf¹⁰² for breast reconstruction in the pedicled form, several other versions have emerged in the process of improvement. Although aesthetically pleasing results have been demonstrated with the pedicled technique, the need to offer patients a reconstructive option with the least donor site morbidity possible has focused interest in muscle-sparing techniques and more specifically muscle-sparing free flaps. Holmstrom and Robbins^{108,112} described the free rectus abdominis myocutaneous flap for breast reconstruction even before it was described in a pedicled form.

Indications and contraindications

Patient-, surgeon- and unit-related factors are the salient points in an individualized reconstructive approach. This type of autologous reconstruction demands adequate, redundant abdominal tissue that can replace in volume the original breast. Preoperative physical examination will also reveal previous scarring which will be critical in deciding the appropriate type of reconstruction. Before the implementation of the CT angiogram, patient selection was dependent on the medical history and physical examination (presence of adequate panniculus, scars, smoking, obesity). The assessment of the abdominal perforators in terms of their calibre, numbers and flow was done intraoperatively.

Recent implementation of the CT angiogram of the abdominal perforators has become routine practice because it assists in patient selection and reduces operative time.^{113,114}

High-risk patients who are obese, heavy smokers (>10 cigarettes/day) and those with significant comorbidities such as diabetes mellitus will benefit from the free muscle-sparing TRAM flap.^{115,116} In addition to the excellent vascularity, studies have shown that it involves less immediate postoperative pain, faster abdominal recovery and shorter inpatient hospital stay^{117,118} when compared to the pedicled TRAM flap.

Contraindications to a free TRAM reconstruction related to patient factors are highlighted in the initial patient consultation when there is inadequacy of abdominal tissue, abdominal scarring and general patient comorbidities that forbid a long general anaesthetic. A CT angiogram for the assessment of the abdominal wall perforators is essential in patient selection. In addition to the above, the surgeon's experience and microsurgical expertise and the availability of a qualified team that will provide postoperative monitoring and support are necessary prerequisites for this type of reconstruction.

Advantages

One of the fundamental differences between the free TRAM flap and the pedicled one is the dominant blood supply. The free TRAM is based on the deep inferior epigastric artery which has been shown to have higher pressure and total blood flow compared to the superior epigastric artery, which is the dominant blood supply to the pedicled TRAM flap.¹¹⁹

Another fundamental difference is that the free flap, although initially introduced as a non-muscle-sparing technique, has evolved into a muscle-sparing technique which nevertheless involves harvesting of a small segment of the rectus abdominis muscle and anterior rectus sheath. This muscle segment safeguards the musculocutaneous perforators in the effort to maintain a robust blood supply in addition to reducing the functional muscle sacrifice. The muscle-sparing technique does not, however, spare the motor nerves of the rectus abdominis muscle as they pass from lateral to medial and from proximal to distal. In that way, large parts of the remaining muscle are denervated and left non-functional. The impact of this denervation is unclear, however, as muscle continuity seems to be more important.

Free tissue transfer combines large volumes of donor tissue, good vascularity, easier inset and elimination of the contour epigastric deformities associated with the subcutaneous tunnelling and the medial IMF breach seen in the pedicled TRAM flap.

The free TRAM flap can be used in immediate or delayed reconstruction and can be anastomosed to either the internal mammary vessels or the thoracodorsal vessels according to the surgeon's preference or special circumstances, such as patients with sentinel biopsy and previous axillary dissection.

The internal mammary vessels are favoured for the well-documented advantages of more medial positioning of the flap, better venous drainage¹²⁰ good arterial flow and unscarred tissue in comparison to the axilla after axillary surgery. Irradiation to the internal mammary lymphatic chain has not shown significant changes to the internal mammary vessel parameters.¹²¹ Last but not least, anastomosis to the internal mammary vessels allows for easier assistance positioning and training opportunity.

In unilateral cases, direct closure of the anterior rectus sheath is always possible in muscle-sparing-free TRAMs, but in bilateral reconstructions closure is not always possible without the use of a mesh.

Complications

Complications can be classified into flap and donor site complications.

Fat necrosis has the potential to occur with every kind of free abdominal flap reconstruction. In theory this is related to the size and position of the perforators, the size of the panniculus, and patient risk factors such as smoking and obesity. The free muscle-sparing TRAM has shown a reduced rate of fat necrosis compared to its precursor, the pedicled form.¹²² Partial flap loss

and complete flap loss are possible complications of both pedicled and free TRAM.

Kroll *et al.*¹²³ and Mizgala *et al.*¹²⁴ have reported that the degree of abdominal wall weakening is proportional to the amount of sacrificed rectus abdominis muscle. This means that the muscle-sparing technique, if performed correctly, by maintaining the nerve supply and vascularity to the remaining rectus abdominis muscle carries reduced risk of abdominal bulging, strength and contour deformity complications.^{115,126} There is no difference in abdominal morbidity between pedicled TRAM flaps and full-width free TRAM flaps.

Flap complications as well as donor site complications have been shown to be significantly higher in obese patients, whereas donor site abdominal flap necrosis and hernias are significantly higher in smokers.^{127,128}

Deep inferior epigastric artery perforator flap

The deep inferior epigastric artery perforator (DIEAP) flap represents the most evolved version of abdominal flap technique for breast reconstruction. Its unique difference from the TRAM flap is the complete preservation of the rectus abdominis muscle with the usage of skin and subcutaneous fat only, based on musculocutaneous perforators of the deep inferior epigastric artery.

Koshima and Soeda¹²⁹ pioneered the deep inferior epigastric artery skin flaps for pedicled groin reconstruction and later Koshima *et al.*¹³⁰ described 13 free thin paraumbilical perforator-based flaps for intraoral defects. The use of the DIEAP free flap for breast reconstruction was reported for the first time by Allen and Treece.¹³¹

Indications

Selection of patients utilizes the same criteria as a TRAM flap. Initial consultation of a patient seeking autologous breast reconstruction should focus on the present comorbidities and previous operations, physical examination to assess the size of the abdominal skin excess and abdominal scarring and the pre- or postoperative cancer treatment, such as chemotherapy and radiotherapy. The ideal patient is free of comorbidities, not overweight or obese, non-smoker and has not had a previous abdominoplasty or abdominal liposuction. Abdominal suction-assisted lipectomy is considered a relative contraindication but preoperative radiological imaging can safely depict the intact abdominal perforators and allow for a successful DIEAP reconstruction.¹³²

For patients who have a diagnosed prothrombotic condition in addition to the breast cancer, an individualized antithrombotic regime should be decided in conjunction with the haematology team.

Preoperative CT angiogram (CTA) of the abdominal wall perforators is now routinely used in microsurgical centres as it benefits both patient and surgeon.^{113,114} This is performed in the

process of the patient selection as it assists in the assessment of the perforator location, their size and intramuscular course which, on some rare occasions, can render a patient a poor candidate for this type of reconstruction (Figure 39.1). The CTA is reported by a specialist radiologist who displays the perforators on a reproducible X to Y axis centred to the umbilicus. These measurements are reproduced in theatre before the operation and the perforators are marked on the abdomen and checked with a handheld Doppler.

The flap is centred over the location of the perforators that are estimated to be the best by CTA, however, the design should be symmetrical over the midline to allow an aesthetic donor site closure.¹³³

Technique

A few technical points on raising a DIEAP flap should be highlighted.

When the distal incision of the flap is performed in the suprapubic crease, the superficial inferior epigastric vein should be preserved and taken with the flap in the maximum length possible. In the event of inadequate venous flow, these vessels will take the role of a 'lifeboat'.

Raising the flap usually starts from lateral to medial until the lateral perforators are visualized. If a CTA has been performed then dissection is faster until this point. All the perforators must be visualized and preserved before the best one is chosen and the rest are ligated. Often the deep inferior epigastric artery (DIEA) separates into a medial and a lateral branch in the middle third of the rectus abdominis muscle. The lateral branch is the dominant one in 54% of the cases.¹³⁴ In general, lateral

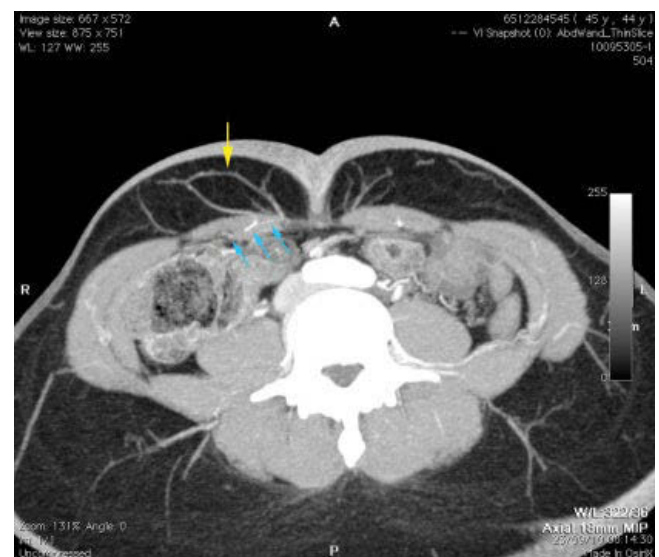


Figure 39.1 Preoperative CT angio image at the level of the umbilicus showing two important perforators on the right side connecting through a subdermal vascular plexus that drains immediately into the superficial epigastric vein (yellow arrow). The intramuscular course of the branches of the deep inferior epigastric artery can also be seen (blue arrows).

perforators are used for bilateral cases (hemi-abdomen flaps) or in obese patients, whereas medial perforators are chosen when a maximum of tissue over the midline needs to be vascularized. The most distant part of the flap is always sacrificed to limit the risk of partial necrosis.

When more than one perforator is located on the same longitudinal row then they can be preserved. On these occasions muscle fibres in between these perforators might have to be divided but the success and the uniqueness of the DIEAP technique relies on strict muscle and fascia preservation and maintenance of the motor nerve supply to rectus abdominis muscle. Division of muscle fibres or motor nerves is to be avoided in all circumstances unless absolutely necessary. If a motor nerve is divided it should be repaired.

The anterior rectus sheath fascia is divided longitudinally distally and 3–4 cm proximally once the perforators have been selected. The main pedicle is identified and dissected off its muscle branches safely by staying close to the vessel and under complete muscle relaxation (Figure 39.2). The pedicle is dissected almost until its origin, providing a length of 10.3 cm and average calibre 3.6 mm.¹³⁵ The thoracodorsal or internal mammary vessels can be used for the anastomosis. The flap skin paddle, which can measure 12–15 cm in height and 30–45 cm in width, can be fashioned in an oblique or horizontal orientation, but it is of great importance to avoid torsion or kinking of the pedicle in this process.

For very large DIEAP flaps one must anticipate inadequate arterial supply and use the ipsi- or contralateral superficial epigastric artery or the contralateral DIEA to supercharge the flap.

Donor site closure is fairly straightforward. The anterior rectus sheath is closed primarily with a continuous mattress

absorbable suture. The abdominal closure is performed in the standard abdominoplasty technique (Figure 39.3).

Complications

The complications of a DIEAP technique are similar in nature to the TRAM flap and are related to fat necrosis, which ranges from 6 to 13%, partial flap loss 7%, and complete flap loss 0–2%.^{136–139} Incidence of abdominal bulging is much lower than TRAM flaps and abdominal hernias are avoided with DIEAP flaps. In addition, the trunk flexion capacity and abdominal strength was found to be reduced in patients who underwent TRAM reconstruction compared to DIEAP.¹⁴⁰ It has been well documented that obese patients and smokers have increased risks of flap and donor site complications.^{127,128}

Superior gluteal artery perforator flap

The gluteus maximus muscle flap is a type III flap according to Mathes and Nahai classification. The main vascular pedicles supplying this muscle are the superior and inferior gluteal arteries. Perforators of either of these vessels are used to raise skin and subcutaneous tissue flaps for autologous breast reconstruction.

Taking in to account that the free DIEAP flap is now the gold standard technique, the superior gluteal artery perforator (SGAP) flap is an alternative method of breast reconstruction. In 1983 Shaw¹⁴¹ popularized the myocutaneous free flap for breast reconstruction but in the process of evolution into the new era of perforator flaps Allen and Tucker¹⁴² reported the muscle-sparing SGAP flap breast reconstruction. Similar to the TRAM flap, the muscle-sparing technique has reduced donor site morbidity.¹⁴³

Indications and contraindications

The natural tendency for fat deposition in the gluteal area and the concealed scars under normal underwear render this area a good candidate for donor site in patients who have extensive abdominal scarring or minimal abdominal redundancy. Patients with previously failed free TRAM or DIEAP flaps, preference on donor site scarring, and adequate gluteal soft tissues for small breast replacements are the main reasons why patient and surgeon would choose this technique over the abdominal type of reconstruction.

Contraindications to this technique are related to poor patient general health, smoking and previous surgery in the region, such as suction-assisted lipectomy.

Technique

This technique involves harvesting a fusiform shape of tissue composed of skin and subcutaneous fat based on the perforators of the superior gluteal artery (SGA). Markings are performed in a standing and prone position and it is very important to orient the skin paddle in the best possible cosmetic position, which is in a horizontal and slightly oblique orientation.¹⁴⁴

The surface anatomy landmarks used to localize the supra-piriform exit point of the SGA are the posterior superior iliac

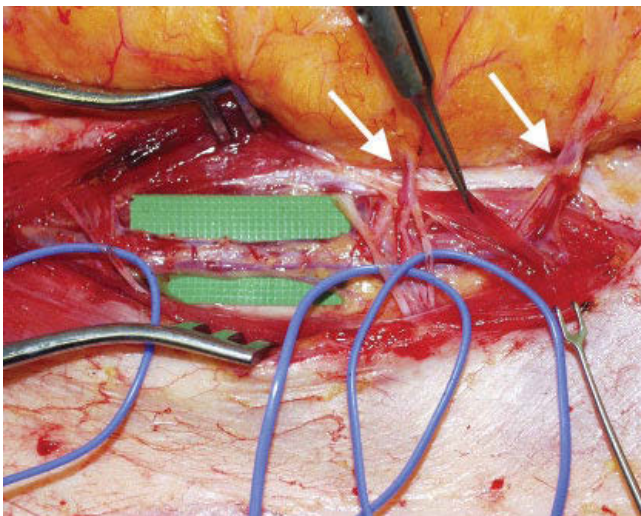


Figure 39.2 Typical intraoperative view of a deep inferior epigastric artery perforator (white arrows) dissection on the left side. Motor and sensory (right vessel loop) are freed and retracted by blue vessel loops. The main deep inferior epigastric vessels are highlighted by the green background. A tiny strip of muscle can be transected to allow harvesting of both perforators.

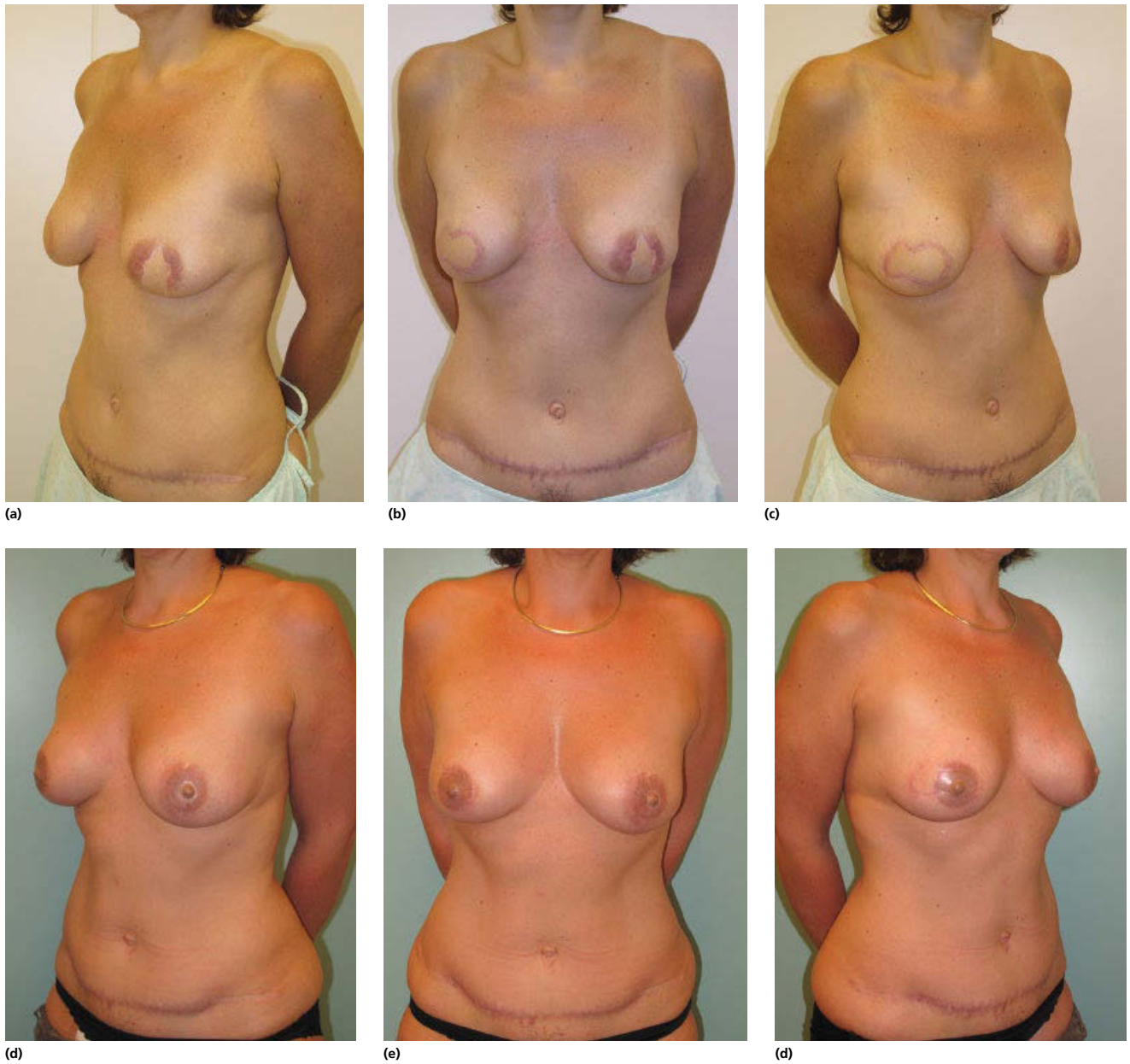


Figure 39.3 A 42-year-old woman after invasive ductal carcinoma at the right breast and confirmed *BRCA1* mutation. Status nine months after right skin-sparing and left areola-sparing mastectomy and bilateral immediate DIEAP flap reconstruction (a, b, c). The skin islands are used to reconstruct the nipples. The entire nipple–areolar complex is then tattooed in a third step to create a uniform colour over the scars (d, e, f).

spine and the lateral point of the greater trochanter. A line is drawn connecting these landmarks and the junction point of the middle and proximal third of this line marks the SGA. The perforators are located lateral and distal to this point along this line, by a handheld Doppler preoperatively.

The average skin paddle measures 10 cm in width and 20–26 cm in length. Elevation of the flap starts laterally to medially, making sure that the fascia of the gluteus maximus is identified and raised with the flap. One perforator is enough to supply the flap. Dissection must proceed with good exposure by separating the muscle fibres along their orientation. When the sacral fascia is

encountered and divided, dissection continues until the SGA and vein are reached, at which point the length of the pedicle is 7–12 cm and the diameter appropriate for anastomosis.¹⁴⁴

The recipient vessels can be either the internal mammary (IM) vessels or the thoracodorsal vessels, but due to the short pedicle compared to a DIEAP flap, the IM vessels are favoured. A two-team approach is possible in this type of technique, with the patient in a lateral decubitus position until the teams are ready to perform the anastomosis. At this point the donor site is closed primarily and the patient is turned into a supine position.

The SGAP flap can be performed as a sensate flap by harvesting one of the nervi clunii superiores along the superior incision of the flap. These are dorsal branches of the thoracic and lumbar segmental nerves, which can be coapted to the anterior ramus of the lateral branch of the 4th intercostal nerve at the mastectomy site.¹⁴⁵

Complications and implications

The intramuscular dissection can be tedious and time consuming but it secures adequate length of pedicle. For the same reason, good exposure is important but aggressive retraction can apply pressure on the sciatic nerve. Especially in the inferior

gluteal artery perforator technique patients may complain about long-term irritation-type symptoms.

In some cases, vessel discrepancy is encountered, most commonly involving the superior gluteal vein (SGV) which has a larger diameter than the SGA.

Despite the fact that it can be a two-team procedure, the positional changes are always time consuming.

Flap shaping into a breast mound is considered easy, but the quality of the subcutaneous tissue of the SGAP flap has less of a ptotic effect compared to a DIEAP flap and initially might look overprojected (Figure 39.4).

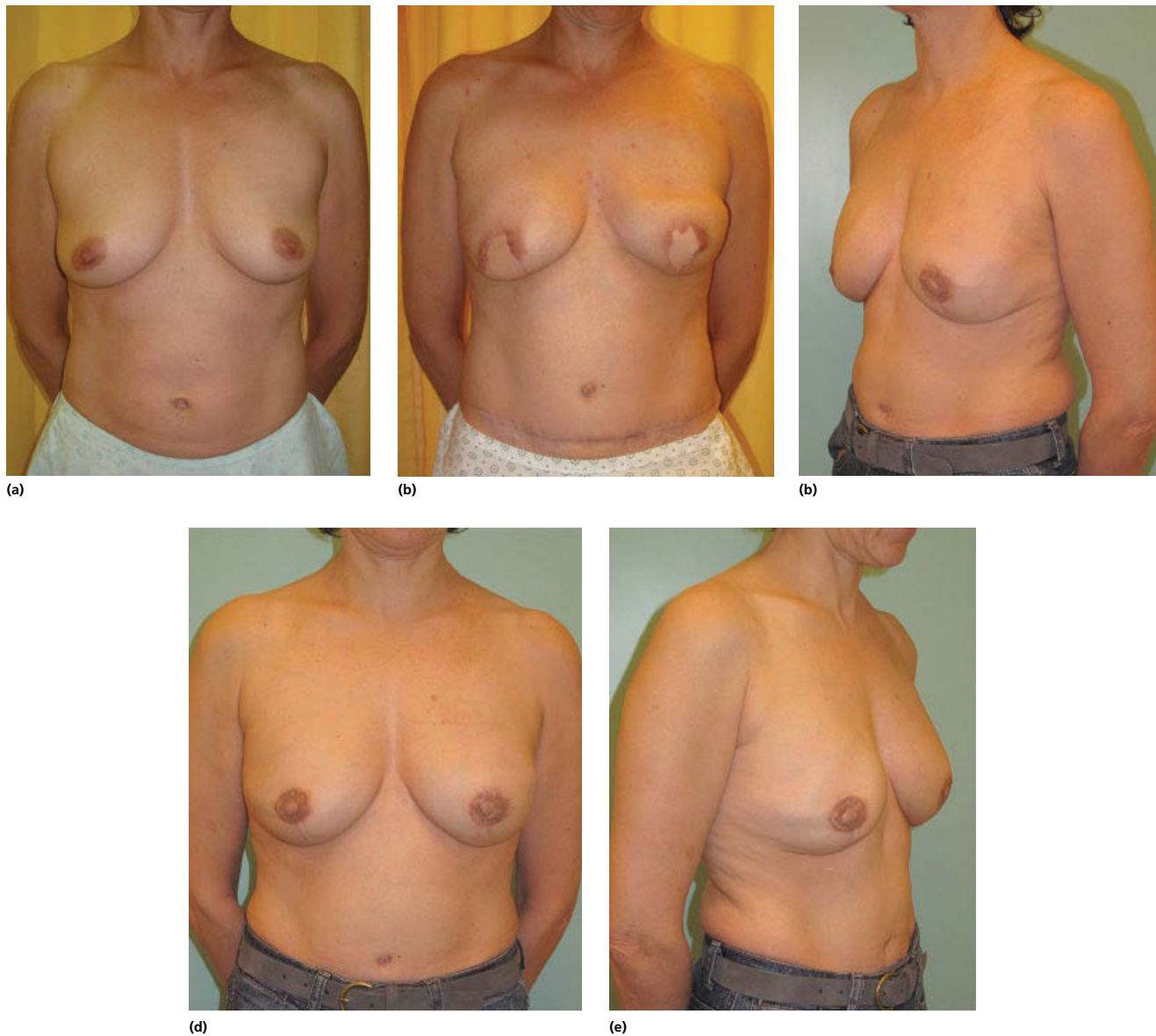


Figure 39.4 Preoperative (a) and postoperative images of a 39-year-old woman diagnosed with a *BRCA2* mutation who underwent bilateral areola-sparing mastectomies and immediate breast reconstruction with a DIEAP flap on the right side and a SGAP flap on the left side. (b) The shortcomings and difficulties in shaping an SGAP flap during the first procedure are clearly shown. Lipofilling and selected liposuction will finally provide the same aesthetic outcome as a DIEAP flap reconstruction (right breast) but additional surgery is always necessary (c, d, e).

Contour deformity of the donor site must be emphasized in the preoperative consultation with the patient. A large amount of bevelling might be needed to achieve the desirable size of breast, leaving a disappointingly hollow contour. Secondary procedures for the correction of the deformity in the form of liposuction and lipomodelling can be performed at the same time as nipple reconstruction.

Inferior gluteal artery perforator flap

The gluteal flaps in the breast reconstruction domain were championed by Shaw¹⁴¹ and Boustred and Nahai.¹⁴⁶ Initial descriptions were of musculocutaneous flaps until the breakthrough of the perforator-based flaps and muscle-sparing concept. The vascular pedicle of the inferior gluteal artery perforator flap (IGAP) exits the pelvis through the greater sciatic foramen in the company of the sciatic nerve, the internal pudendal vessels and the posterior femoral cutaneous nerve.

Indications

The gluteal flaps are used as an alternative type of breast reconstruction in patients who are poor candidates for an abdominal free flap by transferring only skin and subcutaneous fat from the gluteal region. Previously failed free TRAM or DIEAP flaps, inadequate abdominal tissue, abdominal scarring, patient's preference on donor site scarring, adequate gluteal soft tissues in small-breasted women are the main reasons why patient and surgeon would choose this technique over the abdominal type of reconstruction.

Technique

The infrapiriform exit point of the inferior gluteal artery is located halfway between the posterior superior iliac spine and the ischial tuberosity.

Markings are performed in a standing and prone position, and it is very important to design the skin paddle in the best possible aesthetic position, most commonly in horizontal and slightly oblique orientation.

The inferior margin of the flap can be either positioned 4 cm above the inferior gluteal crease or just above the crease in order to utilize excessive saggy tissue in that area. In the former case the scar is hidden in the underwear and in the latter it is disguised by the gluteal fold.¹⁴⁷

The size of the flap that can be harvested is similar to the SGAP flap, measuring 10 cm in width and 20–26 cm in length, but the pedicle is longer, reaching 10–14 cm.

The more lateral the perforators are located on the skin paddle, the longer the pedicle they will provide compared to the more medial perforators.

The muscle-sparing dissection is similar to the SGAP flap and good exposure is achieved by separation of the muscle fibres along their direction. Special caution must be taken to

avoid exposure and damage to the sciatic nerve. In addition, the internal pudendal vessels and posterior thigh nerve must be preserved.

Primary closure of the donor site is achieved with undermining of the skin flaps and by placing a suture to approximate the superficial fascial system.

Complications and implications are similar to the SGAP flap. If excessive tissue harvesting is performed and incisions are not planned correctly, unsightly scars may result.

Transverse upper gracilis flap

Another option in breast reconstruction is the transverse upper gracilis (TUG) flap. It has been used for more than two decades for immediate or delayed reconstruction. The inclusion of a transversely oriented skin paddle along with the gracilis muscle has been described and termed the TUG or transverse myocutaneous gracilis (TMG) flap.^{148–150}

Indications

This kind of flap can provide very good results when a small size of breast is required. In the large series of Schoeller *et al.*, flap weight averaged 330 g, similar to that of other authors.^{151,152}

This flap is ideal for women who have had an abdominoplasty in the past or women who do not want an oblique scar on their gluteal area or back referring to a latissimus dorsi flap for breast reconstruction. One of the advantages of this flap is the thigh lift in bilateral breast reconstructions.

Flap design

The flap is marked with a semilunar skin paddle centred over the longitudinal axis of the gracilis muscle. Tension on the perineal area is reduced by placing the skin island more posterior. By using the pinch technique the borders of the skin paddle can be defined to ensure that the donor site will close free of tension. In this step of the preoperative design, one must take in to account the amount of skin and soft tissue needed to achieve the desired breast volume. The average width of the island is 8–10 cm, or 12 cm in patients with marked weight loss, to ensure that the donor site will close immediately.

Surgical technique

The designed skin paddle has to be harvested with the underlying fascia. Once the edges of the island are incised the tendon of the adductor longus muscle attached on the pubic bone can easily be palpated. The vascular pedicle can be found on the dorsal border of this muscle. Along the axis of the gracilis muscle a minor pedicle is found. This pedicle must be clipped or ligated. When the entire muscle is dissected, first the distal tendon is cut and then its origin from the pubic bone. Care must be taken to preserve the saphenous nerve while dissecting. Once the skin island and the muscle is completely released, the whole complex is transferred to the recipient site. A breast form

can be easily created by folding and suturing together the tips of the paddle. After this step a cone is formed. The apex of that cone can be used to create a nipple as it is on the most projected part of the flap. The transported gracilis muscle can be folded to form the lower pole volume of the new breast after the anastomosis of the vessels has been done. In skin-sparing mastectomy the flap can be totally or partially de-epithelialized.^{153,154}

Complications

The TUG flap has minimal donor site morbidity compared to other free flaps. The TUG flap is not coupled that much with complications as with minor aesthetic results. It has to be said that few authors have focused on the complications of the flap and their management. Shape changes at the level of the inner thighs and donor site scars descending below the groin area may be a problem.

Assuming that the surgeon's experience and microsurgical expertise allows this technique, the downside of this kind of breast reconstruction is the aesthetic outcome due to its small volume. This may not be considered as a complication but it is considered by many authors to be a negative outcome. If volume loss persists then this can be corrected by the classic lipofilling technique to achieve the desired breast volume.¹⁵⁵

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CHAPTER 40

Gynaecomastia and tuberous breast

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Gynaecomastia

Gynaecomastia is commonly described as a benign proliferation of male breast tissue.¹ The incidence of gynaecomastia is reported to be between 32 and 36%^{2,3} of men, and as high as 65% in adolescent boys.⁴ Bilateral disease is reported to occur in 25–75% of cases.⁴ From a surgical point of view it is best to define gynaecomastia as a *persistent* enlargement of male breast tissue. It is this permanent enlargement that motivates patients to seek surgical correction of the condition. From a management perspective, gynaecomastia can be classified as idiopathic, physiologic, pathologic or pharmacologic.

Aetiology

Idiopathic gynaecomastia

Most cases of gynaecomastia are idiopathic.⁵ Oestrogens, androgens and their respective receptors are thought to play a major role in the development of the condition. An imbalance in the concentrations of these hormones, with a relative increase in oestrogens, is thought to bring about breast tissue proliferation.

Physiologic gynaecomastia

This can be further subclassified into neonatal, pubertal and elderly gynaecomastia.

Maternal oestrogens transferred across the placenta into the fetal circulation are thought to contribute to the development of breast tissue in neonates. Treatment is rarely indicated as this usually last weeks to months and is a self-limiting condition. Parental reassurance is usually all that is required.

Transient development of breast tissue is common in adolescent boys. It is usually a failure of this breast tissue to regress that results in emotional distress, motivating surgical correction. Regression is usually seen over a period of months to years. A relative increase in plasma oestradiol compared with testosterone is thought to cause pubertal gynaecomastia. Obesity during teenage years compounds the issue. Increased levels of adipose tissue on the chest add to the problem of enlarged breasts. In addition, obesity results in increased

aromatization of androgens to oestrogens, which further increases breast glandular tissue growth.⁶ These changes in serum oestrogens levels are not usually detectable. As puberty advances, testicular development results in increased androgen levels. This results in regression of breast tissue back to normal in 90% of cases.⁷

With advancing age (>60 years) older men can develop breast tissue as plasma testosterone levels begin to decline. In addition to this, any associated weight gain results in conversion of testosterone to oestrogen (peripheral aromatization). This increased oestrogen-to-androgen ratio results in breast tissue growth.⁵

Pathologic gynaecomastia

Gynaecomastia occurring at the extremes of life is not an unusual finding, however, gynaecomastia occurring suddenly between these two extremes warrants further investigation. Pathologic gynaecomastia occurs as a result of various metabolic and endocrine disorders and acquired and congenital hypogonadal states, leading to an increased overall oestrogen state (Table 40.1).

Pharmacologic gynaecomastia

Drugs are a major aetiological factor in gynaecomastia. There are over one hundred compounds that have been associated with gynaecomastia.⁸ Pharmacologic gynaecomastia is thought to occur by several mechanisms. Increased direct oestrogen activity, decreased testosterone synthesis and decreased androgen sensitivity. In addition, some drugs are also thought to cause gynaecomastia through a prolactin-increasing state (Table 40.2).⁹

Classification

Clinical classifications of gynaecomastia in common use are either based on examination findings or the type of surgical correction required.

Webster¹¹ first proposed a classification for gynaecomastia in 1944. This system grouped patients into three groups:

Table 40.1 Pathologic causes of gynaecomastia

Causes	Examples
Hypogonadism	Primary (trauma, torsion, radiotherapy, infection) Secondary (Kallman syndrome, pituitary failure, hypogonadotrophic hypogonadism)
Tumours	Testicular, adrenal, bronchial
Organ failure	Renal, liver, adrenal, thyroid
Congenital	Klinefelter's syndrome, anorchia
Infective	HIV, herpes zoster
Others	Cystic fibrosis, alcoholism, malnutrition

Table 40.2 Drug-induced gynaecomastia

Class	Examples
Hormones	Oestrogen agonist, anabolic steroids, hCG
Anti-androgens	Bicalutamide, flutamide, nilutamide, cyproterone, goserelin, leuprolide
Antimicrobials	Metronidazole, ketonazole, isoniazid, minocycline
Chemotherapeutic agents	Methotrexate, alkaloids
Cardiovascular drugs	Digoxin, ACE inhibitors, calcium channel blockers, amiodarone, spironolactone, minoxidil
Gastrointestinal agents	Cimetidine, ranitidine, omeprazole
Psychoactive agents	Anxiolytic agents, tricyclic antidepressants, phenothiazines
Others	Metoclopramide, penicillamine, phenytoin, antiretroviral agents

- Glandular
- Fatty-glandular
- Fatty.

Bannayan and Hajdu¹² histologically identified three patterns of gynaecomastia according to the varying amounts of stromal and ductal proliferation:

- Florid – increased number of ducts
- Intermediate – overlap of florid and fibrous types
- Fibrous type – extensive stromal fibrosis with minimal ducts.

Simon's classification of gynaecomastia, described in 1973, is based on subjective assessment of breast volume and skin excess¹⁰ and is the most commonly used system. It is as follows:

- Grade 1: Small enlargement, no skin excess (Figure 40.1)
- Grade 2a: Moderate enlargement, no skin excess (Figure 40.2)
- Grade 2b: Moderate enlargement with skin excess
- Grade 3: Marked enlargement with skin excess (Figure 40.3)

In 2003 Rhorich *et al.*⁴ described another classification system based on the degree of breast hypertrophy, breast ptosis and subtyped them into glandular and fibrous:

- Grade I – Minimal hypertrophy (<250 g of breast) without ptosis
- Grade II – Moderate hypertrophy (250–500 g of breast tissue) without ptosis
- Grade III – Severe hypertrophy (>500 g of breast tissue) with grade I ptosis

- Grade IV – Severe hypertrophy with grade II or III ptosis. Each grade is further subdivided into (A) primarily glandular and (B) primarily fibrous. Glandular and fibrous subtypes are determined using the 'pinch test' medially and laterally and below the nipple–areola complex (NAC).

More recently Cordova and Moschella¹³ have described their own classification system (Figure 40.4). This system takes into account the different structural components of the breast and the relationship with the inframammary fold (IMF) and the NAC:

- Grade I – Increase in diameter and protrusion limited to the areolar
- Grade II – Hypertrophy of all the components of the breast. The NAC is above the IMF
- Grade III – Hypertrophy of all the components, NAC at the same height as or 1 cm below the IMF
- Grade IV – Hypertrophy of all the components, NAC more than 1 cm below the IMF.

Assessment

History

The age of onset, time span of breast growth, pain and any regression or involution should be noted in the patient's history. Alteration in patient weight and its effect on breast growth should also be documented. An enquiry should be made of any systemic symptoms such as weight loss, night sweats and anorexia, which may be signs of pathologic or pharmacologic gynaecomastia. In particular, changes in olfaction or vision should be enquired about as this may suggest a pituitary tumour. Specific questions about medications should include drugs that cause gynaecomastia, including therapeutic and recreational.

Clinical examination

Inspection from the front and side allow the surgeon to appreciate the extent of the disease globally, asymmetry of the breasts and to note any areolar widening and herniation.

The character of the breast tissue is documented by palpating for the presence of any discrete masses. Estimation is made as to the consistency and amount of fibrous subareolar tissue compared to the fibrofatty component. The degree of glandular tissues extension in the subareolar plane is also noted.

It is also important to note how much of the breast contour is due to fatty tissue, and where this fat distribution is most pronounced. Extension of fat under the arm or onto the chest wall must be noted, as this will need to be addressed to create a smooth contour to the chest wall. The chest is also assessed for skin excess.

A testicular examination completes the assessment to exclude possible hormonally active tumours.

Investigations

It is rarely inappropriate to refer a patient with gynaecomastia to an endocrinologist for further investigation. Although pathologic gynaecomastia is uncommonly seen, ruling out underlying

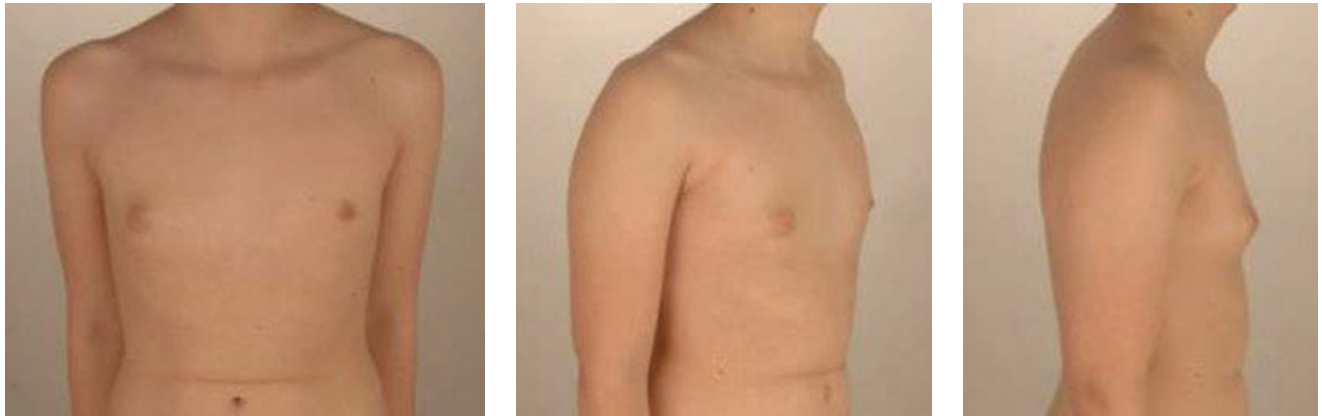
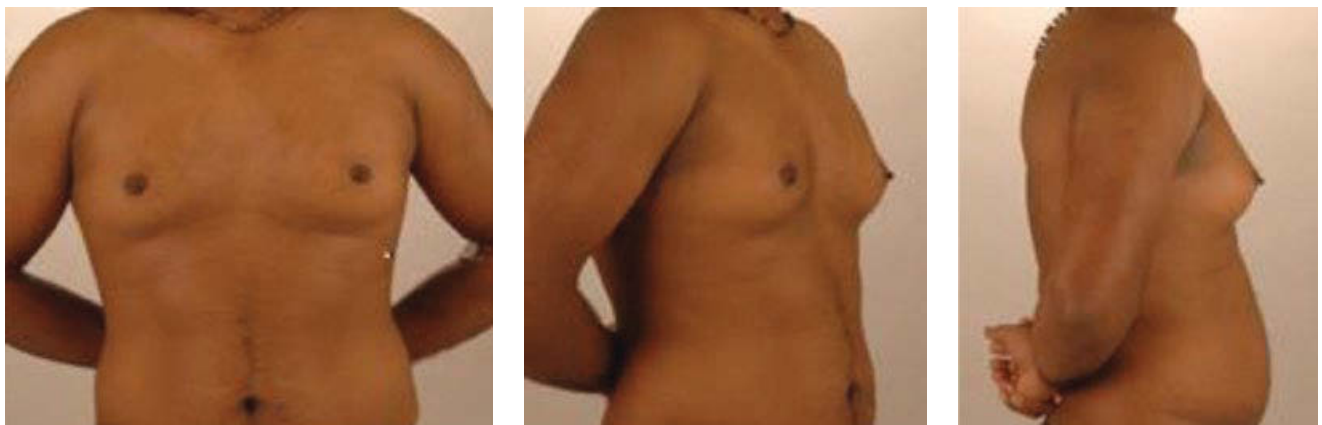


Figure 40.1 Clinical examples of gynaecomastia: Grade 1.



(a)



(b)

Figure 40.2 Clinical examples of gynaecomastia: (a) Grade 2a; (b) Grade 2b.

pathology is paramount to patient care. Investigations should be guided by the examination findings.

General investigations may include evaluation of liver, renal and thyroid function (blood tests), as failure of these organs may cause gynaecomastia. Specific haematological markers may be investigated such as the beta unit of human

chorionic gonadotrophin (β hCG) (testicular tumours), alpha fetoprotein (testicular tumours) and basal serum prolactin levels (prolactinoma).

Imaging can include a chest radiograph to exclude carcinoma of the lung, mammography of the breast to rule out breast cancer and CT head to exclude pituitary tumours.



Figure 40.3 Clinical examples of gynaecomastia: Grade 3.

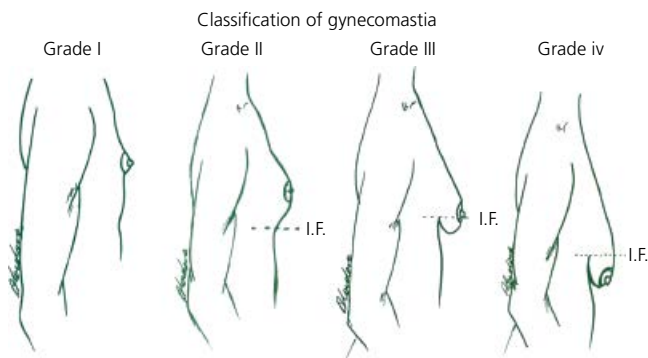


Figure 40.4 Cordova & Moschella classification of gynaecomastia.¹³
Source: Cordova and Moschella, 2008.¹³ Reproduced with permission of Elsevier.

Management

When a recent diagnosis of idiopathic gynaecomastia has been made, it is reasonable to delay definitive surgical management, as spontaneous regression is likely. This is particularly so in adolescent gynaecomastia.

It is also prudent to delay surgical management in those with a body mass index greater than 28 kg/m². Obesity will not only exaggerate the degree of breast enlargement and lead to increased ptosis, but circulating levels of oestrogen are higher

due to adipose tissue aromatization. In addition to this, obesity will increase perioperative risk with regards to infection, wound breakdown and thromboembolic events.

Non-operative treatment

Once a pathological cause of gynaecomastia has been excluded (either clinically or through further investigations) a watch and wait policy is always prudent as some cases may resolve.

The anti-oestrogen tamoxifen has been reported to be effective in some cases of physiological gynaecomastia.^{17–20} Tamoxifen competes with endogenous oestrogen for the oestrogen receptors and inhibits transcription of growth genes. Other pharmacological agents used in the management of gynaecomastia include clomiphene and danazol.

Clomiphene, a non-steroidal agent with a weak oestrogenic activity, acts on the hypothalamic–pituitary axis to increase gonadotrophin-releasing hormone and therefore luteinizing hormone (LH)-releasing hormone and follicle-stimulating hormone (FSH) release. The results of clomiphene treatment however, are disappointing.

Danazol decreases oestrogen production by suppressing the pituitary–ovarian axis by inhibition of the output of both FSH and LH from the pituitary gland. It also has androgenic side effects. It has proved effective in the management of gynaecomastia compared with placebo.²¹ Adverse effects such as weight gain have prevented its general use.

Operative treatment

The aims of surgical treatment are volume reduction with or without retailoring of redundant skin. These aims can be achieved in either a single modal or multimodal treatment strategy. The main surgical options are resection (breast tissue and/or skin) or liposuction (power assisted or ultrasound assisted), or a combination of the two. Again this can be done in a single or staged manner.

Liposuction

Liposuction contouring is indicated in those patients with a predominant fibro-fatty accumulation of tissue. Obese adolescent and elderly gynaecomastia patients are those that benefit the most from liposuction alone. Despite the presence of subareolar parenchymal tissue, liposuction can still reduce its projection and thus yield a satisfactory chest wall contour.

Liposuction can be performed under general anaesthesia or tumescent local anaesthesia and sedation.

Cannula access points can be periareolar, inframammary or laterally on the chest wall. It is important to mark the areas for liposuction preoperatively to ensure a feathered removal of fat and smooth contour to the chest is achieved.

There are several options in terms of type of liposuction technique which can be deployed in gynaecomastia cases.

- *Suction-assisted lipoplasty (SAL)* – Traditional SAL uses blunt tip cannulas with varying diameter attached to tubing with a vacuum as a source of suction. This effectively ‘vacuums’ fat through the cannula. SAL is effective for removal of small volumes of soft fat. It requires considerable effort from the surgeon. Cross-tunnelling must be performed to avoid contour deformities. Bruising is another common sequel of SAL.
- *Ultrasound-assisted lipoplasty (UAL)* – This technique uses ultrasonic waves emitted from the cannula tip to cause micro-mechanical and thermal effects and cavitation on subcutaneous fat. This allows easier removal of the emulsified fat without excess damage to the fibrous septae. This results in greater skin retraction postoperatively. UAL is indicated in areas of fibrous fatty tissue, particularly the breast. Cross-tunnelling may be not be required with UAL as ultrasound energy dissipation at the cannula tip is thought to produce a smooth contour. Bruising is also thought to be reduced with UAL. Thermal injuries and seroma formation can occur with UAL, particularly during prolonged application.
- *Power-assisted lipoplasty (PAL)* – This is similar to SAL but with the addition of an oscillating cannula tip. The cannula tip mechanically breaks down fat and fibrous tissue similar to UAL without the rise in temperature. Cross-tunnelling is required similar to SAL with comparable skin retraction and bruising rates.

Excisional surgery

Patients may present with an isolated fibrous mass beneath the NAC, this may be in addition to diffuse stromal hyperplasia associated with obesity, and there may be excess parenchymal

and skin present. These patients generally require some excisional surgery to address the gynaecomastia. This may be direct excision alone or in concert with liposuction or skin excision (dermoglandular resection).

- *Excision only* – This is indicated in slim individuals with a discrete subareolar mass without any excess skin or fat. It can be performed under general or local anaesthetic. Access is gained through subperiareolar incision at the junction between normal and pigmented areolar skin. The subareolar mass of breast tissue is dissected free from the surrounding breast tissue and NAC. The excision should be tapered peripherally and a small amount of fat should be left behind to avoid a depressed contour deformity beneath the nipple. Postoperatively, a compression support garment should be worn for three weeks to achieve a smooth contour to the chest and prevent haematoma formation.
- *Excision and liposuction* – A combined approach may be required in those individuals where the overdeveloped breast bud is associated with surrounding diffuse stromal fat excess. This can be addressed with excision for the subareolar breast bud and liposuction to the peripheral fatty excess. The combined technique is particularly valuable in those with mild to moderate gynaecomastia. Two strategies can be employed. The patient can undergo a *staged procedure*, which involves liposuction initially, and then removal of subareolar breast bud. Alternatively both procedures can be combined in a *single procedure*. The advantage of the former is that some patients may be satisfied with the postoperative outcome with liposuction alone. This is particularly so when using UAL, as penetration of the breast bud with the cannula can significantly reduce the projection of the breast bud. In addition, the periareolar incision may be avoided. If liposuction alone does not significantly decrease the breast bud intraoperatively, the operation can proceed to direct excision. By then the passage of the cannula through the subareolar breast bud leads to its fragmentation. These can then be removed piecemeal, this has been described by Morselli as the pull-through technique.^{14–16}

Skin excess

In those patients in whom there is excess skin laxity that does not retract after liposuction or excision, or in those with excessive ptosis, then a skin excision is required. It is prudent to wait as long as possible (approximately 6–12 months) to allow as much skin retraction as possible to occur, thus minimizing scars.

Skin excision can be direct, periareolar, circumvertical or horizontal in pattern. Each option is dependent on the degree of skin laxity. In patients with lateral skin excess after liposuction, direct skin excision can be performed with the scar placed in the same line as the lower edge of the pectoralis muscle. This usually leaves a satisfactory scar and addresses the skin excess.

If the NAC requires elevation with excision of skin then a periareolar skin resection can be performed. The NAC position should be between 2 and 3 cm above the IMF. A periareolar doughnut of skin is then de-epithelialized and the defect closed to reduce the skin excess.

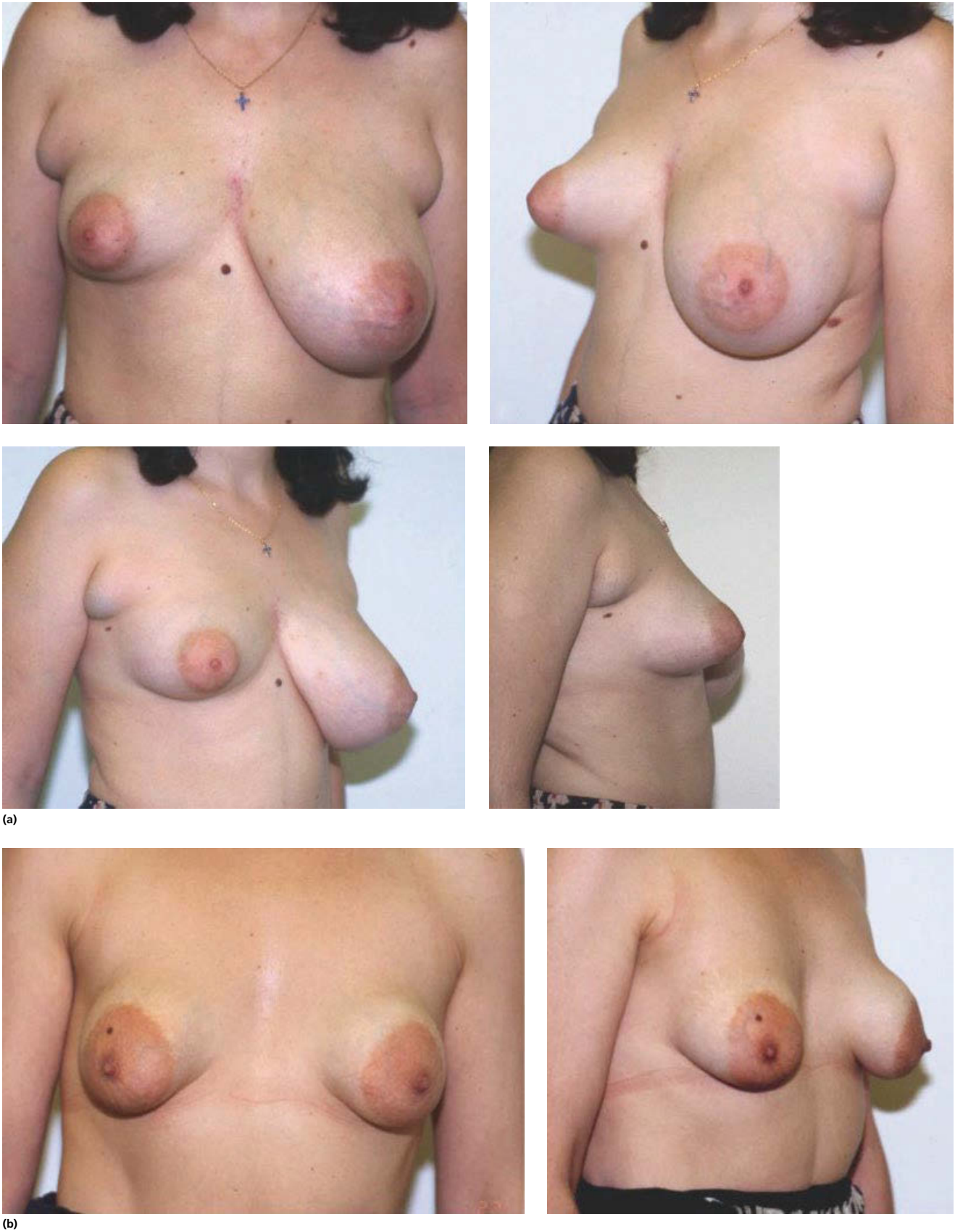


Figure 40.5 Unilateral and bilateral tuberous breast deformity. (a) Unilateral. (b) Bilateral. Source: Shiffman, 2009.²² Reproduced with permission of Springer.

Patients with skin laxity requiring excision in the horizontal and vertical planes would be better suited with a circumvertical pattern of skin excision. The redundant skin in the vertical and periareolar regions can be plicated temporarily to gauge contour correction. Once excess skin has been taken up with plication, excision and definitive closure can be performed.

Horizontal skin pattern excision is used in cases of severe skin redundancy. Blood supply to the NAC is maintained through an inferior pedicle dermoglandular pedicle. Once the skin excess has been removed and the incision closed, the nipple is brought out onto the chest wall in its new position. The resulting scar is placed in the IMF.

Tuberous breast

This rare breast deformity is characterized by a deficiency in the vertical and/or horizontal dimensions of the breast, underdevelopment of the breast and frequently pseudoherniation of breast tissue into the NAC. These characteristics give rise to a breast with a high IMF, constricted base and herniation of the NAC. It can be unilateral or bilateral (Figure 40.5) with its true incidence unreported.^{23,24}

Pathoanatomy

To better understand the mechanism behind tuberous breast formation it is important to appreciate the breast's embryology, described in the breast development chapter. Briefly, the breasts originate from the mammary ridge, an ectoderm derivative, during the fifth week of gestation. The area of the mammary ridge around the thoracic region begins to invaginate into the underlying mesoderm between weeks 7 and 14. Until puberty no further development occurs.

During the invagination of the ectoderm into the mesoderm, the breast tissue is surrounded by superficial fascia. Synonymous with the abdomen the superficial fascia is composed of two layers. A fatty layer superficial to the breast parenchyma, and deeper fascial layer posterior to the breast exists throughout. This fascial layer is absent in the subareolar region.

Histological studies have demonstrated that there is thickening of the deep fascial layer around the areolar in the form of a 'fibrous ring'. This fibrous ring prevents the breast tissue from spreading out onto the chest wall. Therefore, instead of growth posteriorly the breast parenchyma grows anteriorly. Since there is no fascia beneath the NAC, we see pseudo herniation of breast tissue through it.

The severity of the deformity depends on the constricting effect of the fibrous ring. This can range from slight underdevelopment of the inferior and medial quadrants to major hypoplasia.

Classification of tuberous breast

There are two major classification systems in use for tuberous breast. In 1996 von Heimberg described four types of tuberous breast (Figure 40.6) deformity based on a study of 68 tuberous breasts in 40 women.²⁵

- Type I – Hypoplasia of lower medial quadrant
- Type II – Hypoplasia of lower medial and lateral quadrant
- Type III – Hypoplasia of the lower medial and lateral quadrant with skin deficiency in the subareolar region
- Type IV – Severe breast constriction with a minimal breast base.

In 1999 Grolleau²⁶ introduced another classification (Figure 40.7). He modified von Heimberg's classification by arguing that the difference between a type II and III tuberous breast was not clinically or anatomically objective when planning therapeutic options. Therefore, Grolleau's classification consisted of three types:

- Type I – Lower medial quadrant deficiency
- Type II – Both lower quadrants are deficient; areolar points downwards with a short subareolar skin segment
- Type III – All breast quadrants are deficient with a breast base that is constricted in the horizontal and vertical axis.

Assessment

History

Patients may present with either unilateral or bilateral breast deformity. This may have both a functional and psychological impact, such as difficulty wearing certain clothing or relationship issues. The patient with unilateral tuberous breast will have noticed her problem post puberty as breast development occurs.

It is important to ascertain whether the patient has breastfed or plans to breastfeed and counsel then on the potential impact on breastfeeding post surgical correction.

Examination

The breasts should be examined to ascertain the presence of bilateral or unilateral deformity. The key clinical findings are as follows:

- superiorly malpositioned IMF;
- constricted lower pole skin envelope; and
- widened areolar with herniation of breast tissue.

An assessment must be made as to whether or not the constricted lower pole skin can be expanded to allow primary placement of an implant to create a natural lower pole contour. One should also determine whether the old IMF could be overcome by the primary implant once the new lower IMF has been created. In a fully developed tuberous breast a flattened lower pole with breast skin adherent to the chest wall will mean that a primary implant may not correct the deformity adequately.

A general examination of breast for masses and axillary palpation for lymphadenopathy should be performed routinely to exclude malignancy.

Management

The principles in correcting tuberous breast are: (a) the constricted skin envelope and malpositioned IMF should be corrected as much as possible, (b) areolar pseudo herniation must be addressed and the areolar size reduced to normal range, and (c) aesthetic breast volume and symmetry should be achieved.

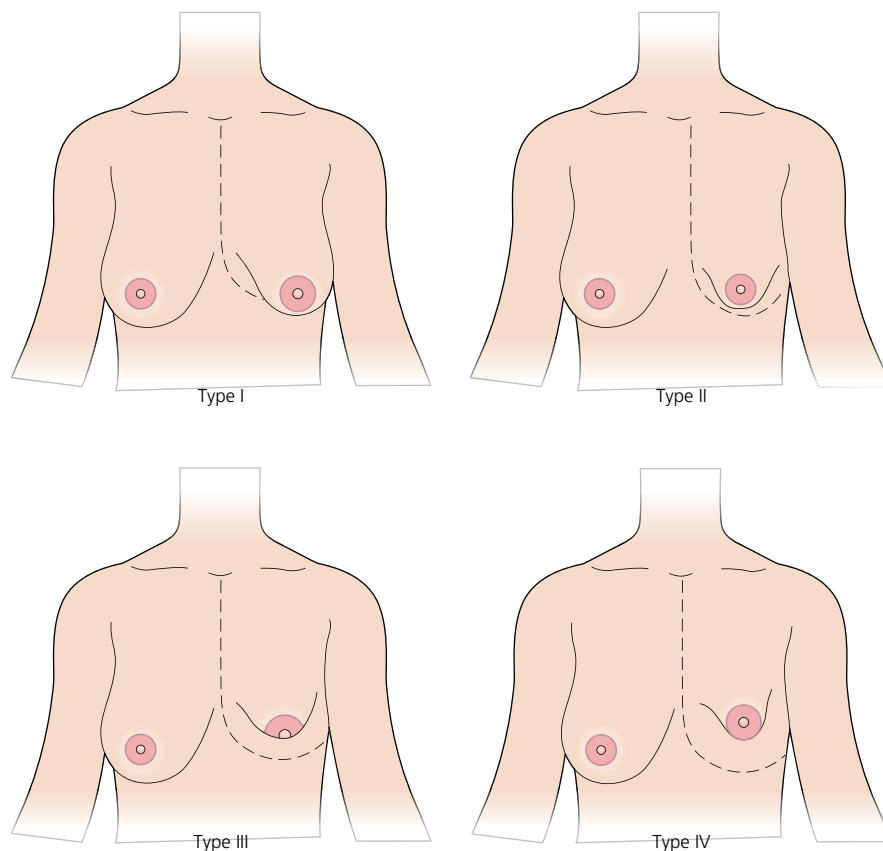


Figure 40.6 von Heimburg classification of tuberous breast. Source: von Heimburg *et al.*, 1996.²⁵ Reproduced with permission of Elsevier.

Constricted skin envelope

Correction of the tight skin envelope and high IMF play a large part in the final aesthetic outcome following surgery for tuberous breasts. A constricted skin envelope will limit the final volume of the breast. Therefore a tight skin envelope will limit the size of any potential implant used to add volume to the breast. This tight skin envelope also means that the IMF is abnormally high.

The constricted skin envelope in mild forms of tuberous breast can be managed with primary placement of a breast implant. However, this is difficult decision to make as a persistent IMF skin crease may be source of patient dissatisfaction and may not soften and relax with time.

A tight skin envelope and high IMF can be stretched and lowered over time using a tissue expander. This can be followed at a second stage with exchange of the expander for an implant.

Areolar size and pseudoherniation

Reducing the areolar size in cases with areolar pseudoherniation has the effect of evenly distributing the forces of the underlying breast tissue on the entire skin as well as adding symmetry to the breast appearance. Areolar size can be matched to the contralateral normal side if possible or in cases of bilateral disease an areolar diameter of 4–4.5 cm can be created depending on patient choice.

Breast volume and symmetry

If bilateral disease exists then both breasts are treated with skin envelope expansion, lowering of the IMF, correction of areolar diameter and the addition of volume. However, many patients present with asymmetry. They usually have a hypoplastic tuberous breast and ptotic contralateral breast (Figure 40.5). In addition to this, the contralateral ptotic breast may also display tuberous characteristics with a high IMF.

It is prudent to treat the more severe side first, usually the tuberous side, followed by symmetrization at a later stage. It is important to counsel patients appropriately that although perfect symmetry is the ultimate goal, realistic expectations are that perfect symmetry cannot be achieved in many patients.

Immediate reconstruction

Periareolar technique

In this approach, using a standard mastopexy technique, the NAC can be reduced in size, the constricting ring divided and the remaining breast parenchyma rearranged and positioned in the breast envelope to achieve a more pleasing aesthetic shape. Additional volume can also be achieved with an appropriate implant. This single-stage approach is appropriate in those patients with adequate skin envelope with mild to moderate (grade I–II) tuberous breast. Adequate breast volume is highly

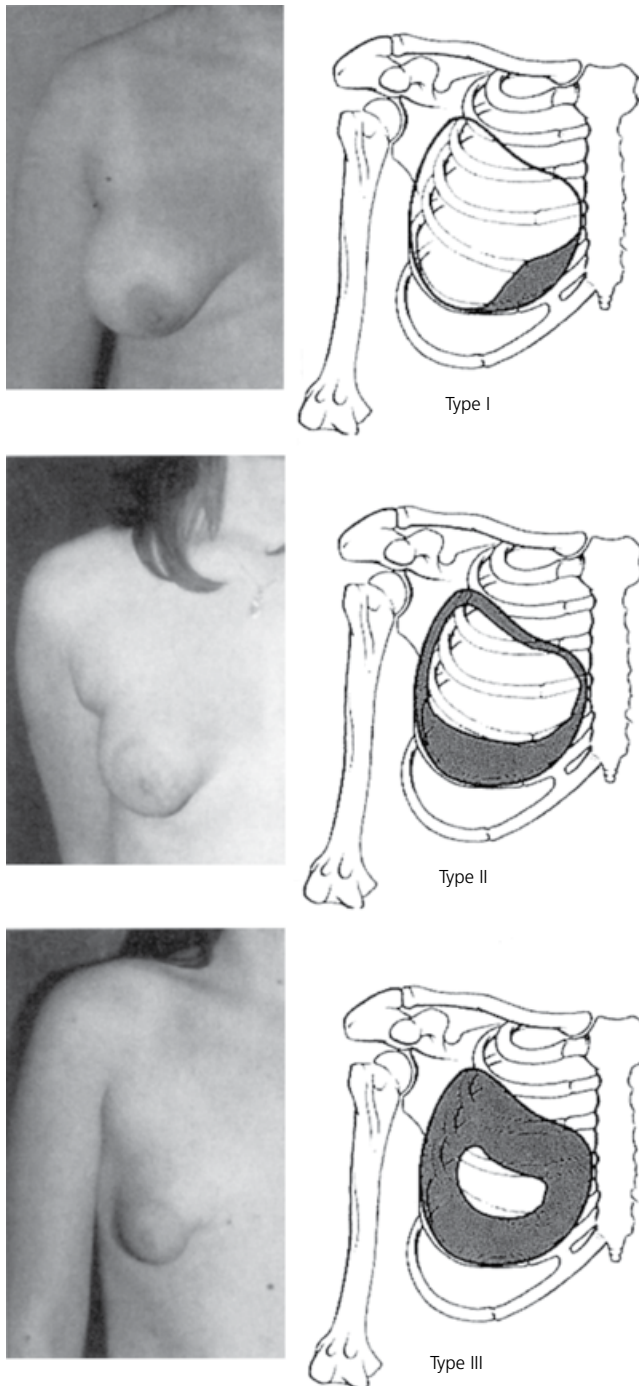


Figure 40.7 Grolleau's classification of tuberous breast. Source: Grolleau *et al.*, 1999.²⁶ Reproduced with permission of Lippincott, Williams & Wilkins.

desirable to allow breast tissue reshaping. When severe skin inadequacy is an issue with a tight lower pole contour then a tissue expander is indicated.

A periareolar ring of skin is de-epithelialized to reduce the size of the areolar (Figure 40.8). The inferior half of the breast is dissected to the IMF and continued behind the breast

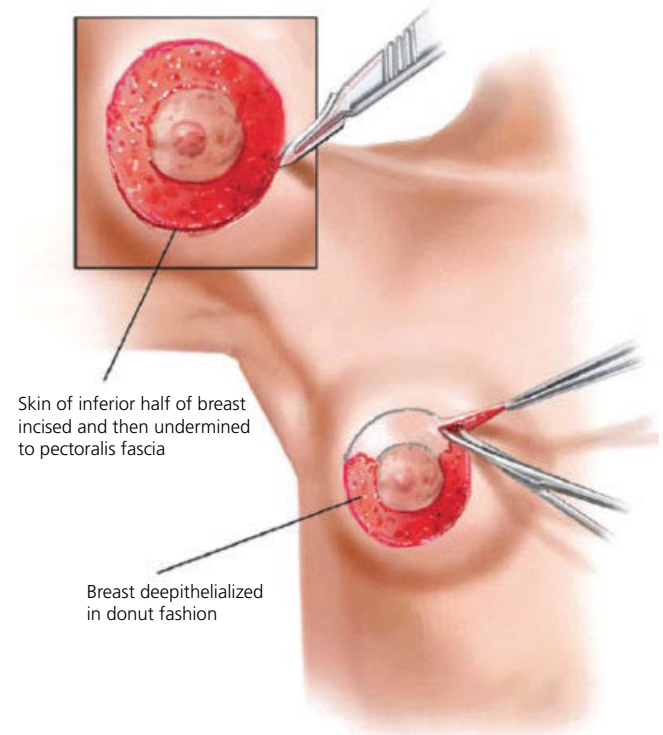


Figure 40.8 Areolar reduction technique. Source: Mandrekas and Zambacos, 2010.²⁷ Reproduced with permission of Elsevier.

parenchyma superficial to the pectoralis fascia. This manoeuvre dissects the breast off the chest wall. The breast is then exteriorized through the periareolar incision (Figure 40.9).

Once the breast parenchyma is exteriorized it can be transected radially or bisected up to the dermis if required or vertically divided in its lower pole (Figure 40.9). This releases the constricting ring and increases the breast width. However, this manoeuvre may result in loss of projection. The two pillars of breast tissue can be loosely approximated or folded onto themselves to create a more desirable breast shape and an implant used if required. Next the IMF can be lowered to the desired position and the periareolar incision closed in layers.

A disadvantage of the periareolar single-stage technique includes a stubborn old inframammary crease that may or may not improve with time. Placing the implant in a subglandular as opposed to a submuscular plane will help counteract this by placing direct pressure on the inframammary crease and lower pole skin.

The technique described above utilizes a superior pedicle to the NAC. An inferior pedicle technique can also be used to achieve the same goals. This is in the form of the short scar periareolar inferior pedicle reduction (SPAIR) technique. This again allows internal release of the constricted breast base. Once the breast is reshaped the widened areolar is reduced and a circumvertical pattern used to reduce the skin envelope.

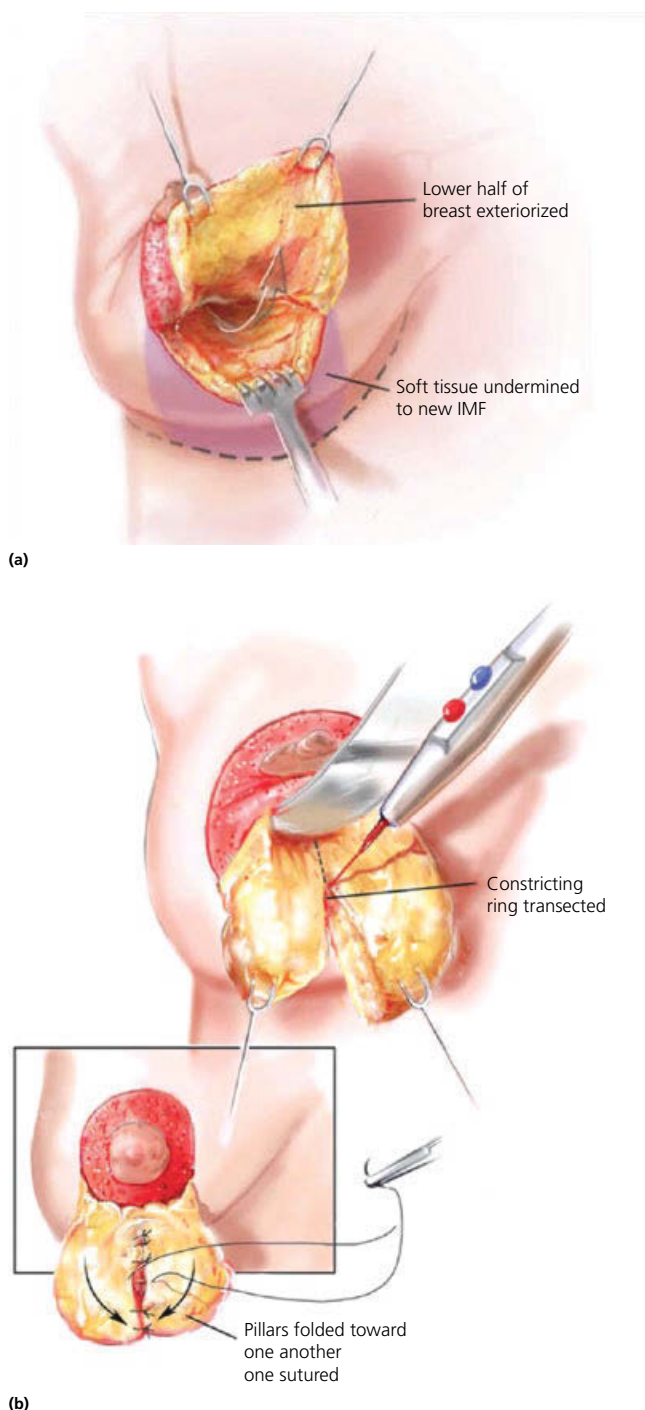


Figure 40.9 Dissection of breast parenchyma off the chest wall and creation of breast pillars. Source: Mandrekas and Zambacos, 2010.²⁷ Reproduced with permission of Elsevier.

Implant technique

If there is inadequate breast parenchyma, and the skin envelope is compliant enough to accept an implant, then a primary breast augmentation can be utilized. A subglandular pocket is ideal as this would better fill out the skin envelope. Access incisions

depend on whether there is areolar herniation and enlargement. If this exists then the previously described periareolar technique can be utilized. If the areolar is a normal size an inframammary incision will allow pocket creation and soft tissue release. This is the case in patients with hypoplastic breasts.

Staged reconstruction

Staged reconstruction is indicated in those patient who require additional volume yet the soft tissue envelope is inadequate to accept a suitably size implant (grade III and IV tuberous breasts). In these patients a staged reconstruction with tissue expansion is required to prepare a sufficiently sized implant pocket.

The optimal plane for expander placement is the same as that for an implant – a subglandular pocket. It is important to select an expander with suitably broad base to fill out the width of tuberous breast and to match the contralateral normal side, or in bilateral tuberous breast the desired breast width.

Expander placement is through a mammary fold incision. The constricted base of the breast can be divided to provide soft tissue release. The expander is placed with its inferior pole at the position of the new IMF. Tissue expansion can begin two weeks after surgery and be continued until the desired volume is achieved.

Definitive implant placement is dependent on the outcome once full expansion has been achieved. In some cases an implant expander may be sufficient to correct the deformity. If areolar reduction is required then this may be the only procedure that is subsequently required to achieve breast symmetry.

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CHAPTER 41

Mastopexy and breast reduction

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The best way to approach breast reduction, mastopexy, mastopexy–augmentation and augmentation is to remove tissue where it is in excess and add tissue where it is deficient. Although this sounds simplistic, it forms the basis of the principles of aesthetic breast surgery.¹ Although in some cases the skin brassiere can be used to shape the breast, it is better to rely on parenchymal reshaping for a consistent, long-lasting result. The parenchyma can be reshaped most easily by following this ‘excess/deficiency’ principle.

The surgeon needs to understand what various surgical manoeuvres can achieve and what our limitations are as surgeons. With that knowledge, we can then manage surgical results (and therefore patient expectations) more appropriately (Figure 41.1).

Because the upper breast border will not change with either a breast reduction or mastopexy, the result can be predictable (but not always what either the surgeon or the patient desires).² Adding an implant will raise the upper breast border on average 2 cm, which has the effect of making the breast footprint look higher on the chest wall. Some patients are high breasted and some are low breasted. Once the patient (and the surgeon) understands this concept, it becomes clear what can be ‘removed’ in a breast reduction or ‘moved’ in a mastopexy.

The surgeon can transpose the drawings in Figure 41.1 onto the patient’s photographs and the decision-making process becomes simplified. Remove (or move) any ptotic gland below the ideal breast shape. Determining the ideal nipple position then becomes simple once the shape is outlined.

This involves an understanding of the inferior wedge principle.^{3,4} The inferior ptotic gland needs to be removed in a breast reduction. It can be separated and moved up to the centre of the breast (not the upper pole) with a mastopexy. Filling the upper pole usually requires either an implant or the addition of tissue (e.g. fat injections). The inferior wedge is that tissue below the Wise pattern. When Robert Wise⁵ deconstructed a brassiere and developed the ‘Wise pattern’, it was designed to be used for the skin to shape and hold up the breast (Figure 41.2). Unfortunately,

skin is often not a good brassiere and it stretches with tension (as it does with skin expansion procedures). If the surgeon, on the other hand, uses the Wise pattern for the parenchyma and leaves behind a Wise pattern (with no tension on either the parenchyma or the skin), the result will be predictable and long-lasting. The Wise pattern shown in Figure 41.2 starts flat but then develops a nice conical shape when put together with the inferior wedge removed.

Because the upper breast border is the more stable (and predictable) landmark, the inframammary fold (IMF) becomes less important. In the past, I marked the new nipple position at the level of the IMF until I realized how variable it can be. Some patients not only have a variable footprint location on the chest wall, the distance between the upper breast border and the lower breast border (IMF) can vary from a few centimetres to over 15 cm.²

Because of the variability of the IMF, the Regnault classification of ptosis can be misleading. Paule Regnault⁶ classified breast ptosis based on the relationship of the nipple to the IMF and breast parenchyma into three grades (Table 41.1). If the nipple was well below the level of the fold, then a mastopexy would be required. Unfortunately, sometimes the nipple is in a good position on the breast mound, but the fold is abnormally high (especially in tuberous breasts) and the approach should instead be to fill the lower pole of the breast and give a better breast shape by centralizing an implant behind the existing nipple position. If the breast is large and the nipple is high then the best approach would be to remove some of the ptotic gland. The nipple should only be moved if it is too low on what will be the final breast mound.

We should instead look at the amount of glandular ptosis or nipple ptosis in relation to the upper breast border and upper pole of the breast. Once the inferior wedge of glandular ptosis is removed, the new nipple position can be easily determined. The ideal nipple position on an average ‘C’ cup breast is about 10 cm down from the upper breast border and about 10 cm from the chest midline (drawn straight – not around the breast).

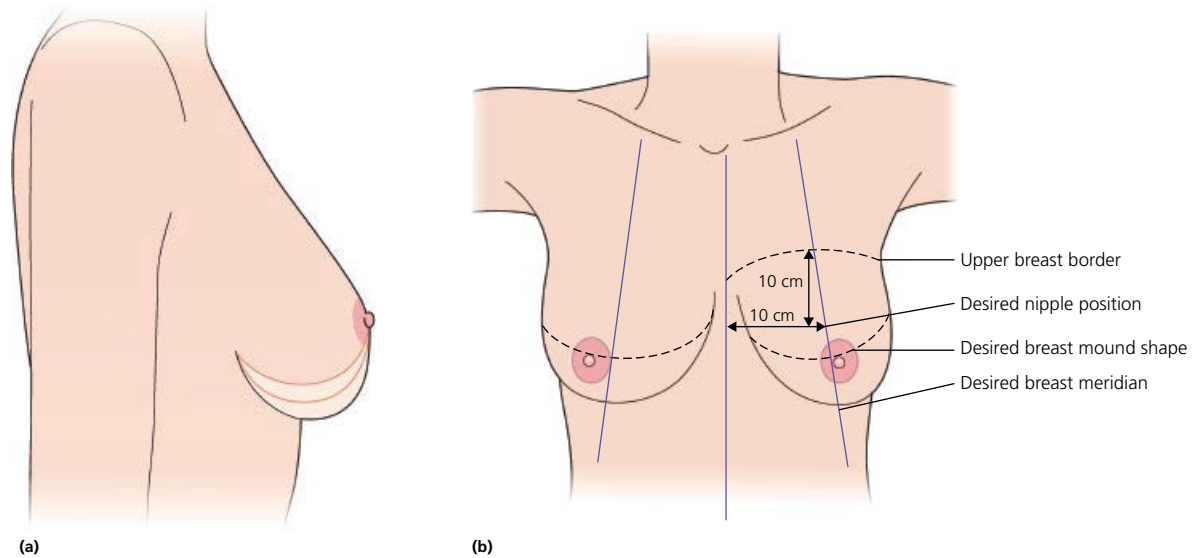


Figure 41.1 Principles. (a) The upper breast border does not change in a breast reduction or mastopexy. The key to a good result is for the surgeon to understand that the excess in the lower pole needs to be 'removed' in a breast reduction or 'moved' in a mastopexy. (b) The surgeon can visualize the shape of the final result by marking what is desired. The excess beyond that shape needs to be removed in a breast reduction. The ideal nipple position should be determined at about 10 cm below the upper breast border (since this remains unchanged) in an average 'C' cup breast. The new breast meridian should be drawn where it 'should' be, not where it exists and the ideal breast meridian is about 10 cm from the chest midline (straight, not around the curve of the breast) in the 'average' patient. Source: Hall-Findlay et al., 2010.¹ Reproduced with permission of Taylor & Francis.

The upper breast border remains at the preoperative level with a breast reduction or mastopexy but it can be elevated on average 2 cm with an augmentation or mastopexy–augmentation.²

It is easier to plan surgery if the surgeon first assesses glandular ptosis and then nipple ptosis. The level of the IMF is the least important landmark. Sometimes it is better to fill the lower pole of the breast (and lower the IMF) with an implant to centralize the breast mound behind the existing nipple position than it is to move the nipple.

Macromastia

Assessment

History

It is important for the surgeon to first assess patient desires in terms of size. Any previous biopsies, mammogram results, and personal and family breast history should be outlined. The patient should be asked about past experience and wishes related to pregnancy and breastfeeding.

Then details of other patient requests, such as shape and upper pole fullness, should be determined.

Examination and photographs

The consultation is far easier if photographs are taken. The surgeon can then mark on the photos to illustrate what kind of result can be achieved. The upper breast border is marked (dotted line in Figure 41.3a). The distance of 'flat' chest wall is marked with the vertical arrow on both frontal and lateral views.

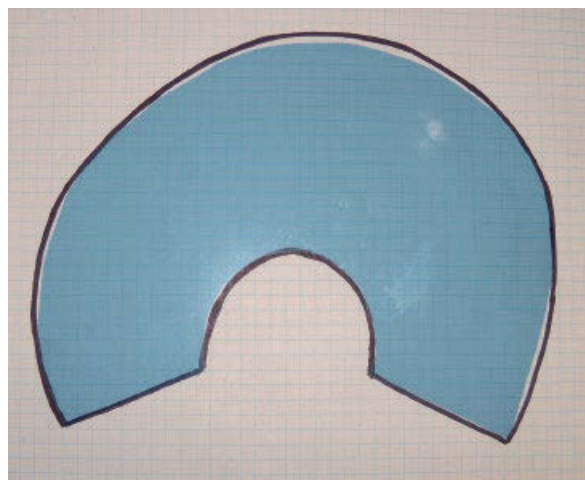
Note that the drawing shows removal of the glandular ptosis – removal of both inferior and lateral breast tissue where it is in excess. Once the desired inferior border of the breast is determined, the new nipple is placed about one-third up the breast mound. Note that the IMF is not marked – and not needed.

There is still a bit of overhang in the final result because it is difficult to lift the breast completely up onto the footprint without some overhang. Having the breast shape completely confined to the breast footprint is difficult without lowering the IMF (as often happens with an inverted T inferior pedicle breast reduction).

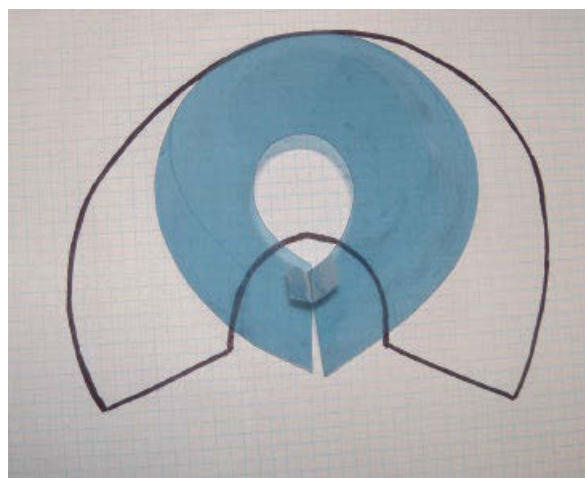
Patients need to be warned that they will not achieve the 'double push-up brassiere effect' without clothes. There is normally a vertical distance of flat chest wall (this can vary from about 9 cm to 16 cm in an average height patient) between the clavicles and the upper breast border. The breasts do not normally come up close to the clavicle and the upper breast border should never be higher than the preaxillary crease. This has become an important part of managing patient expectations over the last few years with the new fashions in brassieres.

If a patient has very poor upper pole fullness, some of the inferior wedge can be moved up^{7,8} (separated from all surrounding breast tissue but left attached as a chest wall-based flap) to provide better projection, but any attempt to try to fill the upper pole is usually doomed to failure. The tissue that is mistakenly pushed up will inevitably fall down, resulting in unattractive glandular ptosis.

Patients often see more in the photographs than they do in the mirror and they can then understand the limitations of the



(a)



(b)



(c)

Figure 41.2 The Wise pattern was adapted from a deconstructed brassiere by Dr Robert Wise⁵ and it forms the basis of an ideal breast shape. Once the flat pattern is coned (by removing or moving the inferior wedge) a good shape with good projection is achieved. (a) The Wise pattern. (b) The Wise pattern coned. (c) The Wise pattern is an excellent design for what should be left behind to achieve a good breast shape rather than using it as a skin brassiere pattern. Source: Elizabeth Hall-Findlay. Reproduced with permission.

Table 41.1 Regnault's ptosis classification

Grade	Description
I	NAC at the level of IMF
II	NAC below the level of IMF but above the most dependent part of the breast parenchyma
III	NAC well below the IMF as well as the most dependent part of the breast parenchyma
Pseudoptosis	NAC is at or above IMF but the majority of the breast parenchyma has descended below the IMF

procedure. Patients need to understand that they cannot achieve either a higher footprint or more upper pole fullness. They can, however, achieve better projection.

Management principles and operative options

Reduction/mastopexy

There are numerous designs and labels for various breast reduction and mastopexy techniques. The design should be divided into two categories: pedicle design and skin resection pattern.

Pedicle design

Pedicle design is based on blood supply. If the surgeon does not believe that there is a safe pedicle available, then a free nipple graft may be an option. Usually, however, there are good pedicle designs that can be used. The main choices are inferior (and central), superior, medial, lateral or a combination (Figure 41.4).^{9,10}

The breast is a superficial structure attached to the skin at the nipple. Most of the blood supply to the breast is superficial. It starts out in a deep location around the periphery of the breast and then both the arteries and veins travel around the breast in the subcutaneous tissue.

Most of the blood supply to the breast comes from the internal mammary system. The inferior or central pedicle is supplied by the deep artery and vein that penetrates through the intercostal muscles and the pectoralis muscle from the fourth interspace. That this artery (and venae comitantes) comes from the fourth interspace makes sense because the breast is a fourth interspace structure.¹

The superior pedicle is supplied by the internal mammary system by the descending branch of the artery from the second interspace and the medial pedicle is supplied by the third interspace. There is clearly some variability but a true superomedial pedicle can be designed to include these two axial arteries.

There is also some blood supply from the superficial branch of the lateral thoracic system and this artery supplies a lateral pedicle.

Most of the arteries are within the first centimetre deep to the skin surface at the level of the areola. The veins travel separately from the arteries and they are more superficial – just under the dermis. They can often be seen through the skin when examining

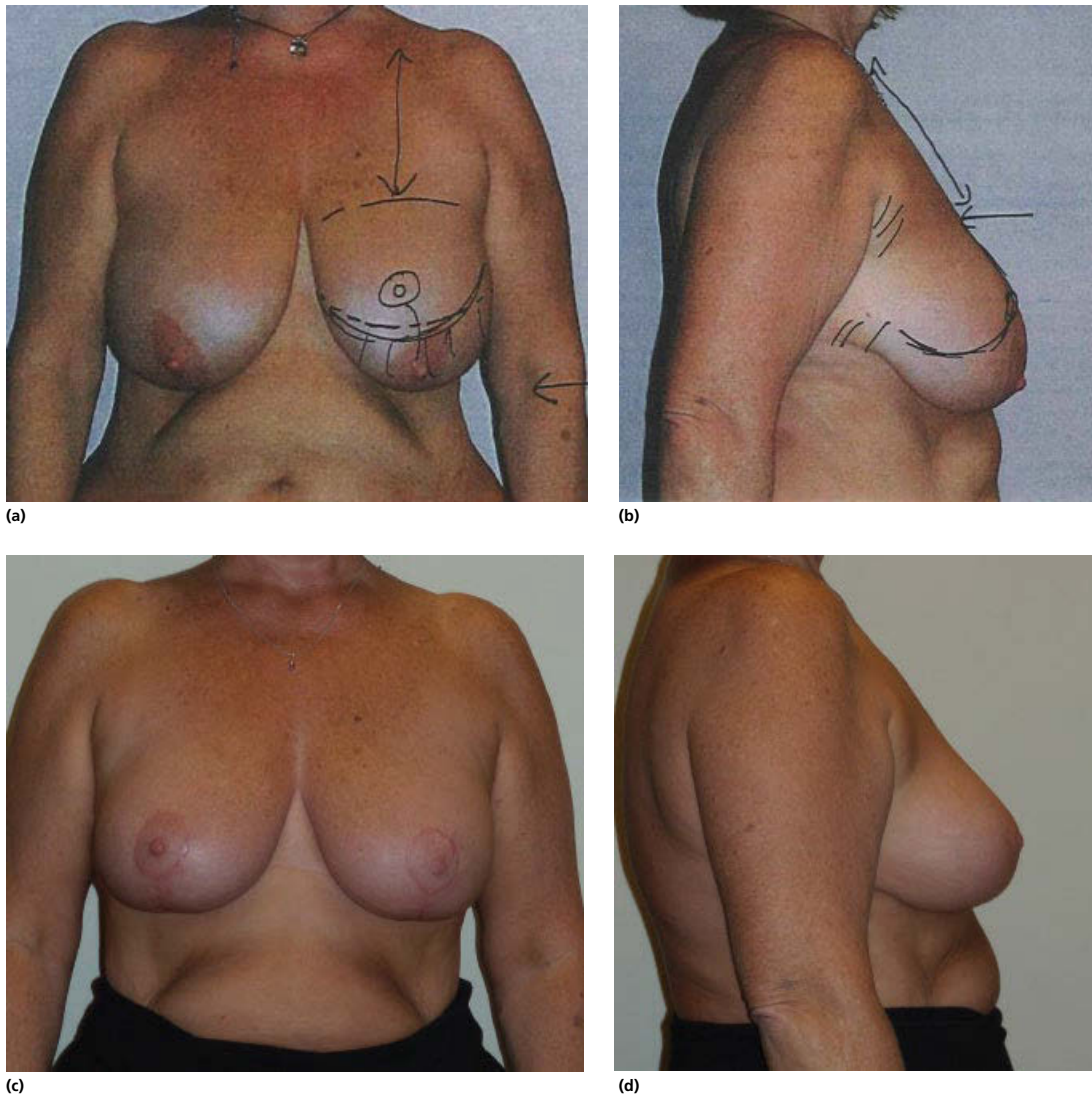


Figure 41.3 Managing patient expectations through consultation photographs. Patient photographs taken at consultation can be used to manage patient expectations. Once the surgeon understands that the upper breast border will not change, the expected result can be drawn on the preoperative photograph. More projection can be achieved by coning the breast tissue after the inferior wedge is removed. The breast cannot be elevated on the chest wall and the upper pole cannot be filled, but the angle (projection) can be increased. The surgeon will also know if tissue needs to be removed in the upper pole in the occasional patient where reduced projection is desired. (a) Preoperative frontal breast reduction candidate showing the desired final breast shape with inferior and lateral tissue removed. (b) Preoperative lateral of same breast reduction candidate showing the patient that the upper breast does not change but the lower pole is removed. (c) Postoperative view of same patient showing the postoperative result. The frontal view matches the preoperative drawing. (d) The lateral postoperative view matches the preoperative drawing. The breast footprint remains unchanged but the glandular ptosis is corrected and the nipple repositioned to the most projecting part of the breast. Source: Elizabeth Hall-Findlay. Reproduced with permission.

the patient and they can be seen to drain mainly superomedially (Figure 41.5).

Because the blood supply to an inferior or central pedicle comes up through the chest wall, the pedicle must be full thickness. Because the blood supply to the superior, lateral and medial pedicles is in the subcutaneous tissue, the pedicles can be created as dermoglandular pedicles. The pedicle should be thick where the vessels are deep at the breast periphery and then can be thinned out closer to the areola where the vessels are found

usually within the first centimetre of tissue depth beneath the skin. Figure 41.6 shows the artery and vein to the inferior wedge that is about to be removed in a vertical breast reduction using a superomedial pedicle.

Innervation to the nipple comes from various sources.¹¹ There are nerves that come from all directions. There are several medial branches but also a major nerve from the lateral fourth intercostal nerve. This nerve splits with a superficial branch that courses up in the subcutaneous tissue and a deep branch that

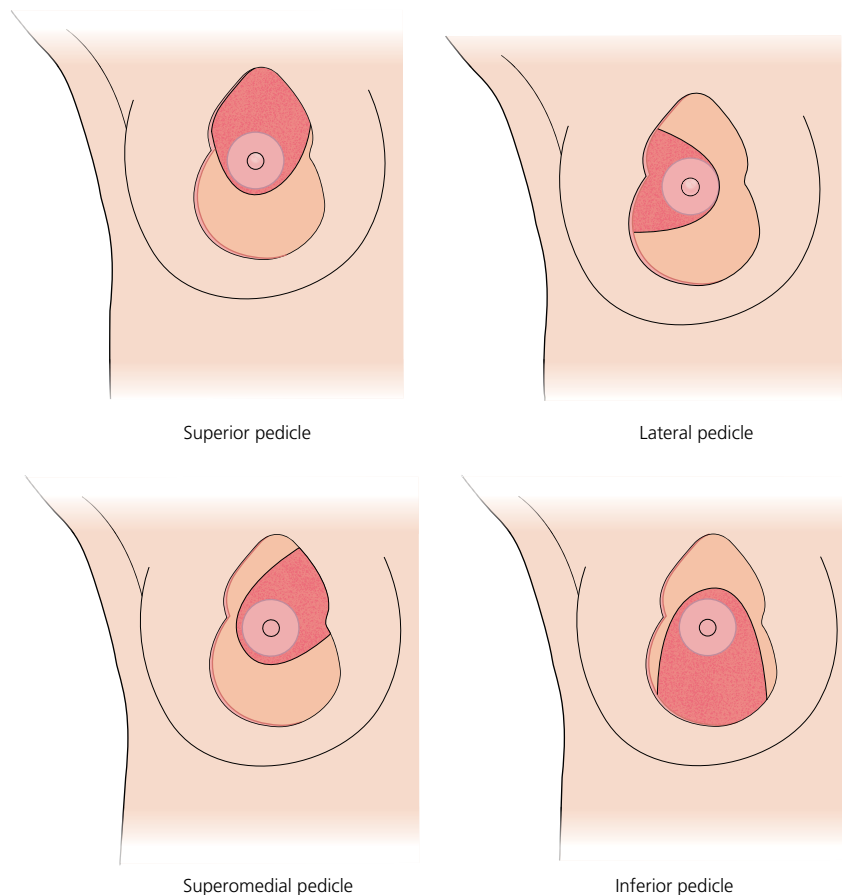


Figure 41.4 The various pedicle designs for the nipple and areolar complex. A true superomedial pedicle combines both superior and medial pedicles to include two axial arteries. The majority of the venous drainage is superomedial. Source: Elizabeth Hall-Findlay. Reproduced with permission.

travels along the pectoralis fascia. The deep nerve branch then turns upward at the breast meridian and can be included in a full-thickness pedicle. Evaluation of my patients has shown that the medial pedicle actually has better return of sensation than either lateral or superior pedicles.¹ Other studies have shown that there is not much difference in sensation recovery among the various pedicles.

Skin resection pattern

The pedicle design determines the design of the parenchymal resection pattern, but the skin pattern can vary. The classic inverted T (Wise pattern) skin resection pattern^{12,13} was designed to use the skin brassiere to hold up the remaining breast parenchyma. In those cases where the skin has good elasticity, it can be used to shape the breast.

Unfortunately, skin tends to stretch under tension and many surgeons are now using the inferior wedge resection principle to remove the heavy inferior breast tissue, then closing the pillars together following the Wise pattern⁵ for the parenchymal resection and closure instead of using the Wise pattern for the skin resection pattern. The skin resection pattern will then depend

on how much skin redundancy there is because it is used to redrape around the breast parenchyma instead of being used as a handle or support.

Not all 'vertical' (or 'inverted T') breast reductions are the same. They can be designed with various pedicles. Usually, however, the term 'vertical' is associated with the superiorly (superior, medial or lateral) based pedicles and the term 'inverted T' is associated with inferior or central pedicles.^{14–26} The excess breast tissue is removed inferiorly (and laterally) with the superiorly based pedicles and the excess breast tissue is removed superiorly (and laterally and medially) with the inferiorly based pedicles.

Periareolar skin resections patterns (where the outer doughnut skin edge is sutured to the inner doughnut hole skin edge) are only used rarely because there is not much skin resected. If the patient has a significant amount of redundant skin, then more skin needs to be resected along either a vertical or inverted T pattern. The breast is not well reshaped when the skin alone is tightened – surgeons need to be aware that Benelli's description²⁷ of a periareolar mastopexy did not just tighten the skin; he reshaped the underlying breast parenchyma as well. Failure to follow this advice has resulted in unsatisfactory results.

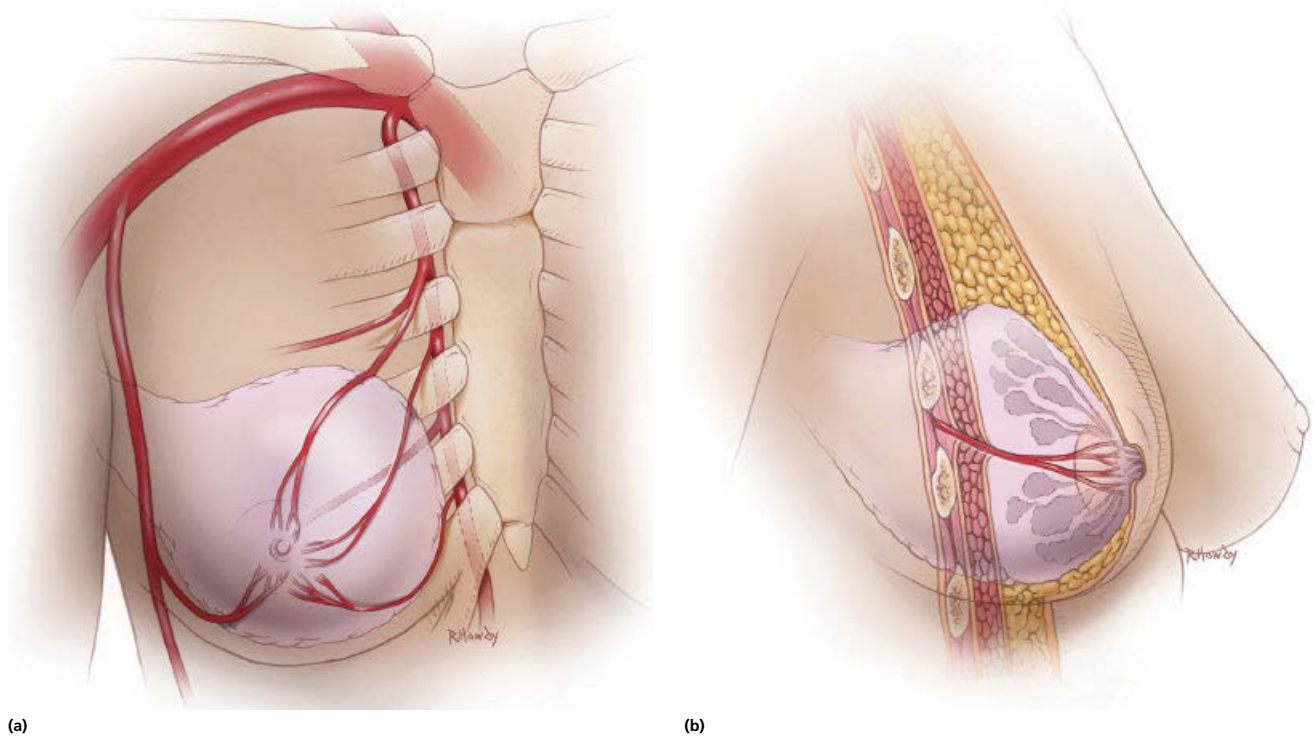


Figure 41.5 The major blood supply to the breast comes from the internal mammary system. There is also some contribution from the superficial branch of the lateral thoracic artery. (a) The arteries penetrate up from the chest wall around the periphery of the breast and then they pass around the breast parenchyma itself up in the subcutaneous tissue. The arteries are superficial as they course toward the areola. The veins are even more superficial as they course just under the dermis (and are often visible through pale skin) with the majority draining superomedially. (b) The branch of the internal mammary artery from the fourth interspace actually penetrates directly up into the breast parenchyma just medial to the breast meridian just above the fifth rib. This artery has accompanying venae comitantes. This makes sense because the breast is a fourth interspace structure attached to the skin at the nipple and sliding loosely over the pectoralis fascia. The breast is held in place by skin–fascial structures at the inframammary fold and over the sternum. The superior and lateral aspects of the breast slide freely over the underlying tissues. Source: Hall-Findlay, 2014. Reproduced with permission of Sage.

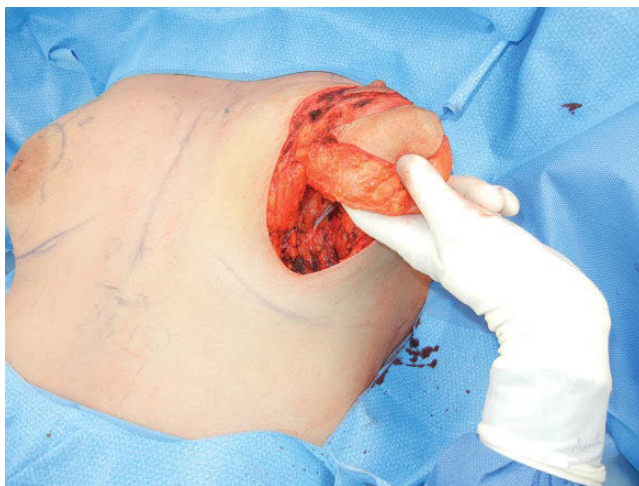


Figure 41.6 This photo shows the artery and vein to the inferior wedge that is about to be removed in a vertical breast reduction using a superomedial pedicle. Source: Elizabeth Hall-Findlay. Reproduced with permission.

Indications

Some surgeons believe that the inferior pedicle is a safer pedicle for the very large breast reduction. I personally believe that the true superomedial pedicle has the best blood supply because it includes two axial arteries. I have pencil dopplered over 85 patients and the descending artery from the second interspace is usually just medial and occasionally just lateral to the breast meridian.

When the breast enlarges, the blood vessels also enlarge and travel down with the nipple. The risk in a longer pedicle is not that the blood vessels do not travel to the nipple but that creation of the pedicle might not actually include those particular vessels.

An inferior pedicle (using the inverted T skin resection technique) is the easiest to inset but the weight of the breast is left inferiorly. The medial and lateral pedicles are easy to inset because they rotate into position. The superior pedicle can be harder to inset because it needs to be folded or forced up. Superior pedicles often need to be thinned in order to allow an easier inset without compression and this leaves an empty concave lower pole.

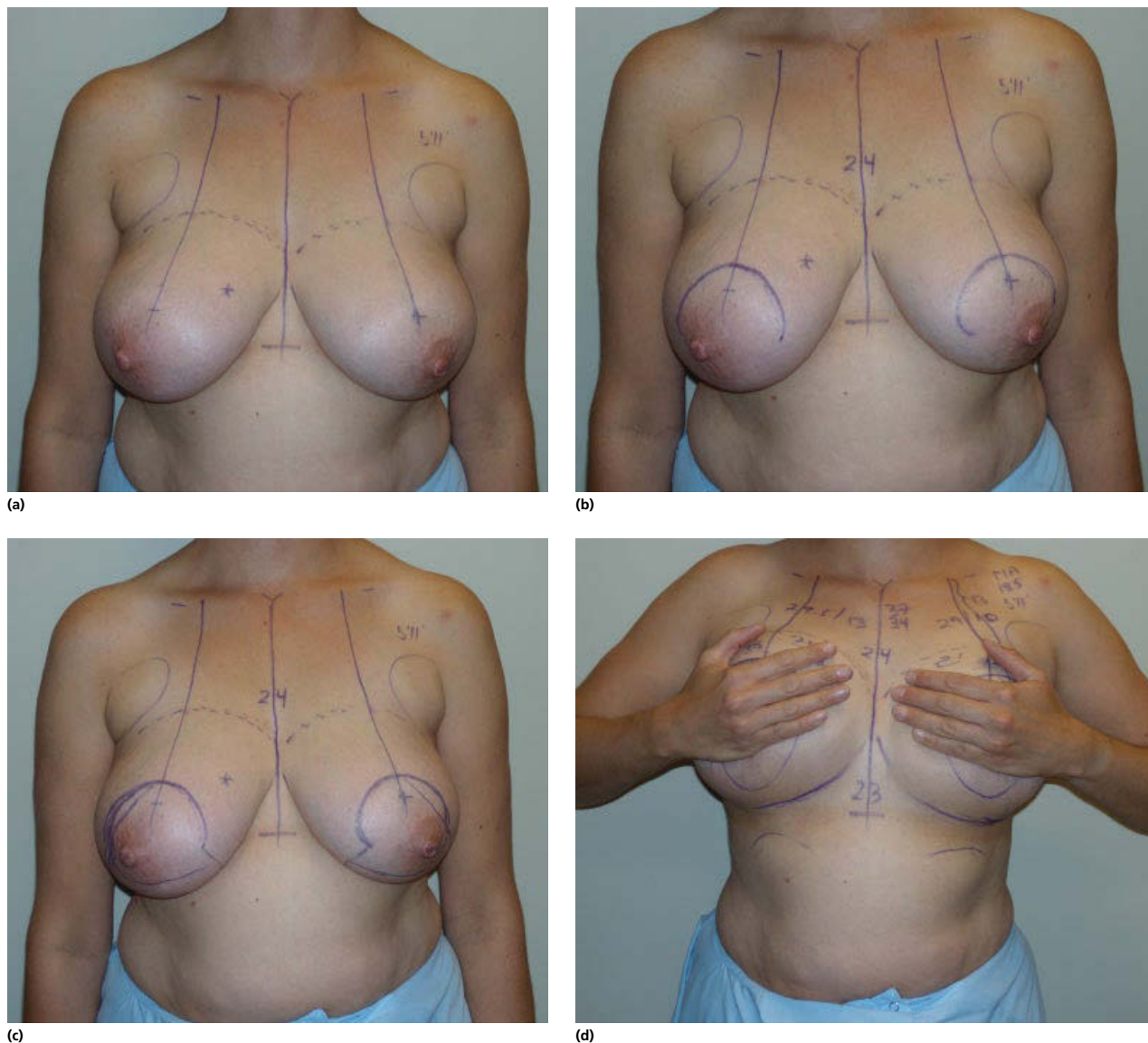


Figure 41.7 Markings for a vertical breast reduction using a superomedial pedicle. (a) The upper breast border and desired breast meridian are marked. The new nipple position is marked about 10 cm below the upper breast border. The inframammary fold is marked at the chest midline (it is often different from one side to the other) but it is not used to determine the new nipple position because it is often too high or too low. Because the upper breast border remains unchanged from the preoperative position to the postoperative result, it is the better landmark to use to determine the new nipple position. (b) The areolar opening is drawn freehand about 2 cm above the new nipple position. The drawing should be performed so that the final result is a circle. A large paperclip is 16 cm long and it can be folded out and used as a template if desired. The skin opening will determine the new areolar diameter because the areolar skin is so elastic that it will stretch to fit the skin pattern. A 16-cm-circumference matches a 5-cm-diameter areola and a 14-cm-circumference matches a 4.5-cm-diameter areola. (c) A true superomedial pedicle is drawn. A pure medial pedicle has a base that is about 4 cm up into the areolar opening and about 4 cm below along the vertical limb. A true superomedial pedicle has an extension of the base up and slightly across the breast meridian to capture the descending artery from the second interspace. A true superomedial pedicle contains the arteries from both the second and third interspaces and still allows the lower border of the pedicle to become the medial pillar (giving an elegant shape to the lower pole) while avoiding some of the difficulties inherent in inseting a superior pedicle. (d) The lower skin resection pattern is drawn while rotating the breast medially and then laterally to match the desired breast meridian drawn on the breast and continued down onto the chest wall. The pattern should be joined usually about 4 cm above the inframammary fold to take into account the fact that the fold often rises but also to avoid the scar extending onto the chest wall. Closure of an ellipse ends up with a longer scar at each end. The pattern should be designed only to remove the excess, not to use the skin as a handle or brassiere. The key is to reshape the parenchyma (by removing the inferior wedge) and then allowing the skin to redrape. Source: Elizabeth Hall-Findlay. Reproduced with permission.

The true superomedial pedicle (with the main base medial but extending upward and across just lateral to the breast meridian) can be harder to inset than a true medial pedicle. But

if the surgeon understands that the blood supply is superior, debulking deep tissue which is causing the restriction can be performed safely. This not only provides a good reliable blood

supply but it also gives a nice elegant curve to the lower pole of the breast on the table because the inferior border of the medial pedicle becomes the medial pillar.

The lateral pedicle can be a good pedicle but it is often difficult to remove some of the lateral bulk in a breast reduction because the tissue that is in excess (laterally) forms the base of the lateral pedicle. Although there are some veins that drain superolaterally, the majority of the venous drainage is superomedial.

With a mastopexy, breast tissue needs to be rearranged because tightening the skin brassiere alone is not effective. Patients who have developed ptosis have already demonstrated that their skin envelope cannot hold up the breast parenchyma. A small breast reduction is the easiest mastopexy, especially when an inferior wedge of breast tissue is removed. When the patient wants to maintain their volume, then an inferior flap can be created, moved up and the pillars closed around caudal to the flap.^{7,8} When this method of a mastopexy is used, the pedicle is therefore based superiorly (superior, superomedial or superolateral). This method of 'moving' breast tissue (that is 'removed' in a breast reduction) is a good way of reshaping the breast when the skin brassiere has lost elasticity.

Operative technique for superomedial vertical breast reduction

Assessment

The surgeon needs to assess the footprint – whether the patient is high or low breasted. Then he or she needs to visualize what needs to be removed – usually inferiorly and laterally. The patient needs to be consulted on their preferred breast size (and of course warned that an exact size and shape cannot be achieved).

The superomedial pedicle can be used with either the vertical or inverted T skin resection pattern.²⁸ This will depend on the desired final size of the breast and the elasticity and amount of excess skin. A vertical distance from the lower border of the areola to the IMF is usually about 7 cm in a 'B' cup breast, 9 cm in a 'C' cup breast and 11 cm in a 'D' cup breast. Surgeons are often warned to keep the vertical scar length at 5–7 cm in an inferior pedicle inverted T breast reduction because the weight of the breast stretches out the skin. In a vertical type of breast reduction, however, the extra vertical length is needed to accommodate projection.

Markings

First the surgeon should mark the *upper breast border* because this is a landmark that does not change. This is best determined by finding the preaxillary indentation laterally and marking the upper breast border medial to that indentation (Figure 41.7). The breast can be pushed up to see where the fold between the breast and the chest wall occurs.

Next the surgeon should draw the *chest midline* and *breast meridian*. The 'ideal' breast meridian should be marked and not the 'existing' breast meridian. The surgeon can then draw what

the resultant breast shape would be (see Figure 41.3). This can help to mark the new nipple position.

The *ideal nipple position* on an average 'C' cup breast on an average patient should be about 10 cm below the upper breast border and 10 cm from the chest midline (not drawn around the curve of the breast but with a straight ruler on a line parallel to the chest wall). If a surgeon places the new nipple (for example) at 24 cm from the suprasternal notch (SSN), that distance will not change postoperatively. It will remain at 24 cm. An arbitrary SSN–nipple distance can be misleading, leading to too high a nipple placement. The surgeon needs to understand that the footprint cannot be changed and a low-breasted patient will remain low breasted.

The *new areolar opening* should be marked about 2 cm above the new nipple position. The opening can be drawn freehand to be about 14–16 cm in length. A 14 cm circumference matches a 4.5 cm diameter areola and a 16 cm circumference matches a 5 cm diameter areola. A large paperclip is 16 cm long and can serve as a good template when unfolded. There is no need to draw out the opening laterally in a mosque-type shape. It is in fact better to take out length vertically rather than horizontally.

In order to mark the *skin resection pattern* the breast can then be rotated laterally and the vertical meridian marked to match the breast and chest wall meridians. The breast is then rotated medially and the lateral meridian marked. The breast can be held up with the surgeon's thumb of the opposite hand and the two lines can be joined in a 'U' or 'V' shape, but the bottom of the design should remain at least 2–4 cm above the IMF. The surgeon can then test the skin resection pattern by pinching the skin.

A vertical breast reduction using the superomedial pedicle does not rely on the skin brassiere so there is no need to design a wide skin resection pattern. There should be no tension on the vertical skin closure. If an inverted T design is needed, the medial and lateral extensions are drawn about 7 cm down the vertical limbs. These extensions are then drawn to meet the IMF both medially and laterally. The skin resection pattern should not result in a tight closure in any direction; it is used only to allow the skin to redrape loosely.

A pure *medial pedicle* is designed with the base about 4 cm below the areolar opening along the vertical skin resection pattern and 4 cm above into the areolar opening. A true *superomedial pedicle* takes the medial pedicle base and extends it up and across the breast meridian. This allows it to include two axial arteries (as opposed to one artery in a medial pedicle) but it does make it a bit harder to inset occasionally. If it does prove to be too stiff to inset easily then excess tissue can be safely debulked deep to the pedicle because the blood supply is superficial.

Finally the areas that may need to be liposuctioned (preaxillary fullness and lateral chest wall areas) are marked.

Positioning

The patient is supine on the operating table with the arms outstretched to the sides.

Infiltration

It is not a good idea to infiltrate along the incisions because the veins can be damaged. Lidocaine with adrenaline is used in the areas to be suctioned. If the patient has a higher body mass index, then tumescent type of infiltration may be indicated for the lateral chest wall in particular.

Operative technique²⁹

De-epithelialization

The incision lines are incised around the areola and vertically, making sure that the vertical skin resection pattern remains about 4 cm above the IMF. A lap pad is then placed around the base of the breast and held with a Kocher clamp to tighten the breast skin and facilitate de-epithelialization.

The veins are just deep to the dermis, so whatever method is used, the surgeon should keep the dermis intact during de-epithelialization. The skin is easier to remove in a superomedial pedicle than in an inferior pedicle (Figure 41.8a).

Pedicle creation

The pedicle is created initially as a full thickness pedicle cutting straight down toward the chest wall. Most of the bleeding occurs in the first few centimetres as initially the veins are encountered just under the dermis and then the arteries are severed in the subcutaneous tissue. There is not much bleeding from the breast parenchyma itself (Figure 41.8b).

No alteration is made at this stage for inseting the pedicle. If the pedicle is stiff then deep tissue can be debulked later to allow for an easier inset.

Parenchymal resection

The inferior wedge of parenchyma is then removed, cutting straight down to the chest wall. Once a surgeon is more familiar with the technique an en bloc resection including lateral tissue can be performed but a 2-cm-thick lateral pillar should be created before undermining the lateral flap. It is important to remove tissue down toward the pectoralis fascia, but there will be less bleeding if the fascia itself is left undisturbed. The surgeon will encounter the deep perforators coming just up above the fifth rib (fourth interspace) just medial to breast meridian. There are some branches that perforate more laterally as well but all from the internal mammary system (Figure 41.8c).

The excess parenchyma in a breast reduction is inferior and lateral. Once the inferior wedge of breast tissue is removed, then more tissue can be bevelled out under the lateral flap. The lateral parenchymal pillar should be about 2 cm thick and about 7 cm in length. If the tissue is thick glandular tissue (as in a teenager) the surgeon needs to carefully carve out all tissue until a good Wise pattern is left behind. If the lateral excess tissue is composed mainly of fat, it can be removed later with liposuction, but any 'white' glandular tissue needs to be directly excised (Figure 41.8d).

Rarely does any parenchyma need to be removed medially except just above the IMF. It is good to leave behind some parenchyma in the lateral aspect of the areolar opening to serve as a platform so that the areola does not retract once it is inset. Even a full-thickness pedicle will appear to be undermined once it is created.

The surgeon should consciously leave behind a Wise pattern of parenchyma. This will mean not only removing the inferior wedge of tissue and tissue under the lateral flap but also tissue caudal to the Wise pattern above the IMF. This will result in some elevation of the IMF – another reason to keep the lower border of the vertical skin resection pattern about 4 cm above the original IMF.

The surgeon who is less familiar with the vertical approach will initially remove less parenchyma than required – the breast looks smaller on the table than it is when sitting.

Pillar closure

It is best to close the areolar opening first to best assess the shape and to effectively close the pillars. The dermis does not need to be undermined or released for this suture (usually a 3–0 Monocryl). Once the areola is closed, the pedicle can be easily rotated up into the opening. If there is any resistance, the deep tissue of the pedicle can be debulked because the blood supply is superficial (Figure 41.8e).

If the pedicle is pulled up in a cephalad direction, the pillars will automatically come together. The inferior border of the medial pedicle becomes the medial pillar and the first suture should be placed at the caudal end of the lateral pillar (about 7 cm down from the areolar opening as shown by the arrows on the photograph) and where the inferior border of the medial pedicle meets the skin (i.e. at the caudal end of what is now the medial pillar) (Figure 41.8f).

Deep bites are not necessary (and can actually lead to tissue necrosis). It is only necessary to make sure that parenchyma (not fat) is apposed on each side so that healing can occur. I usually use only 3–4 sutures of 3–0 Monocryl to hold the pillars in contact with each other. Any tension on parenchyma (which I mistakenly tried with lateral pedicles in a vain attempt to pull lateral tissue inward) will eventually release and the tissue will return toward its original position (Figure 41.8g).

Skin closure

The deep dermis is then closed with interrupted 3–0 Monocryl sutures again with the goal of only achieving dermis-to-dermis contact so that the circulation to the skin edges is not compromised. The vertical skin is then closed with a subcuticular 3–0 or 4–0 Monocryl but it is important not to 'cinch' or 'gather' the vertical incision. It was initially thought that it was important to shorten the vertical incision because of the fear of bottoming-out that occurred when the skin stretched with the inferior pedicle, inverted T breast reduction.

Not only does the gathering compromise blood supply to the skin edges, but it is ineffective in shortening the vertical skin

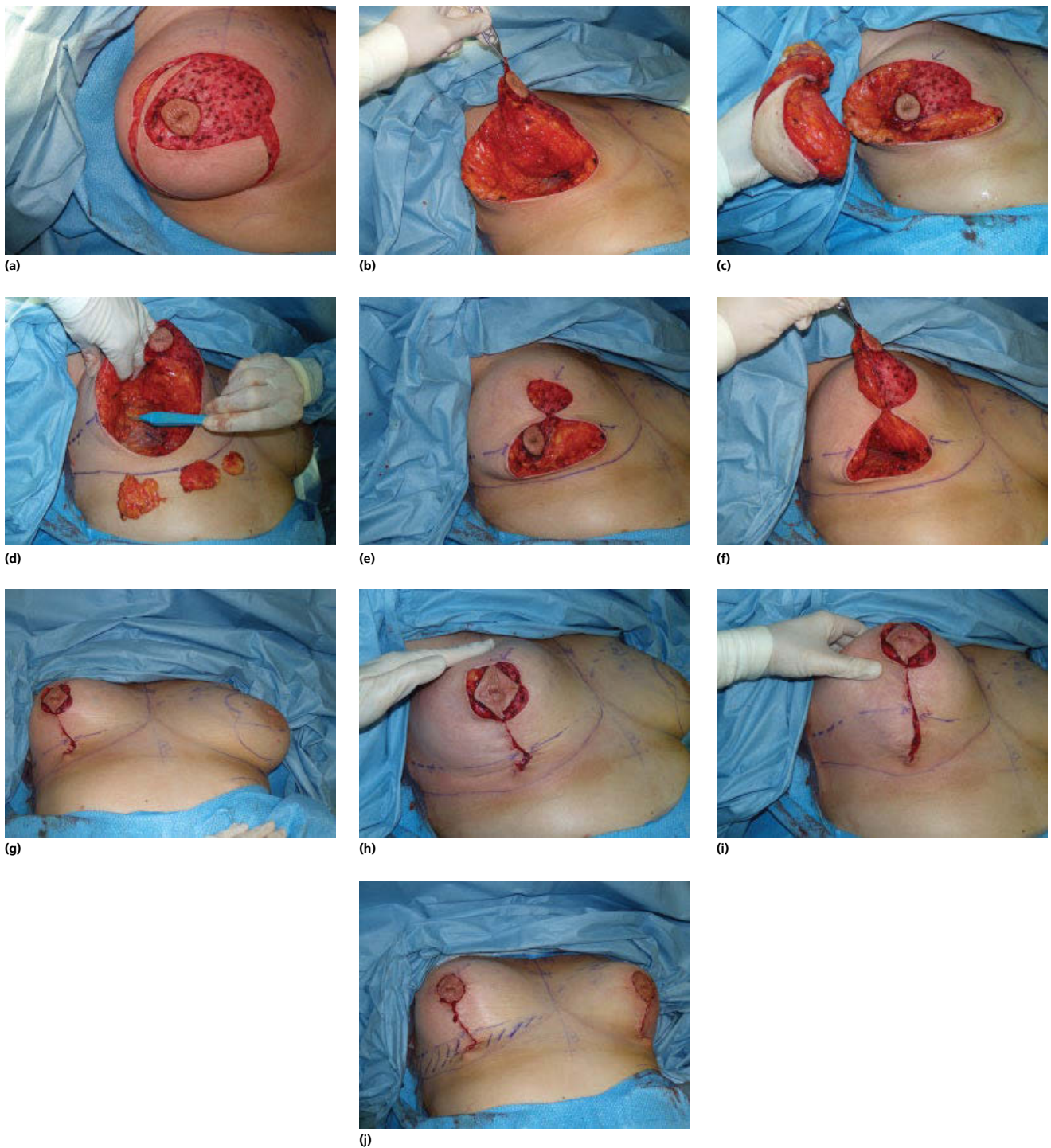


Figure 41.8 Operative technique for superomedial vertical breast reduction. (a) De-epithelialization of a superomedial pedicle. Note that the base of the pedicle extends along the vertical limb but that the upper base extends across to just lateral to the breast meridian. This allows the pedicle to include two axial arteries (from the second and third interspaces). (b) The superomedial pedicle is created as a full-thickness pedicle to include the ascending branch of the fourth intercostal nerve that travels above the pectoralis fascia and then turns upward toward the areola at the breast meridian.¹¹ (c) Resection of the parenchyma is performed initially around the pedicle but mainly as an inferior wedge resection. An initial *en bloc* resection is performed while leaving a 2-cm-thick lateral pillar (about 7 cm long). The medial pillar will be formed by the inferior border of the medial pedicle. (d) Extra breast tissue is removed inferiorly above the IMF and below the Wise pattern (which is marked in blue on the skin). This is performed to prevent bottoming-out and to prevent an inferior pucker (which is usually a problem of lack of subcutaneous tissue excision rather than a skin redundancy problem). Aggressively removing this tissue will result in a higher IMF level. The blue forceps are pointing to the excess parenchyma that needs to be removed under the lateral flap. In a very glandular breast (young women) this tissue needs to be carved out under the flap but also to remove any tissue lateral to the desired Wise pattern. In older patients, this excess can be liposuctioned but it must be completely removed or else the breast will end up too wide and full laterally. Tissue needs to be removed where it is in excess. (e) The lower border of the areola is closed first to allow the surgeon to assess the extent of resection. No dermal undermining is needed for this closure. (f) The superomedial pedicle is easily rotated up into position. This manoeuvre of pulling the areola cephalad allows the medial and lateral pillars to fall together. The arrows marked on the skin show the lower edges of the pillars. This is usually about half way up the vertical

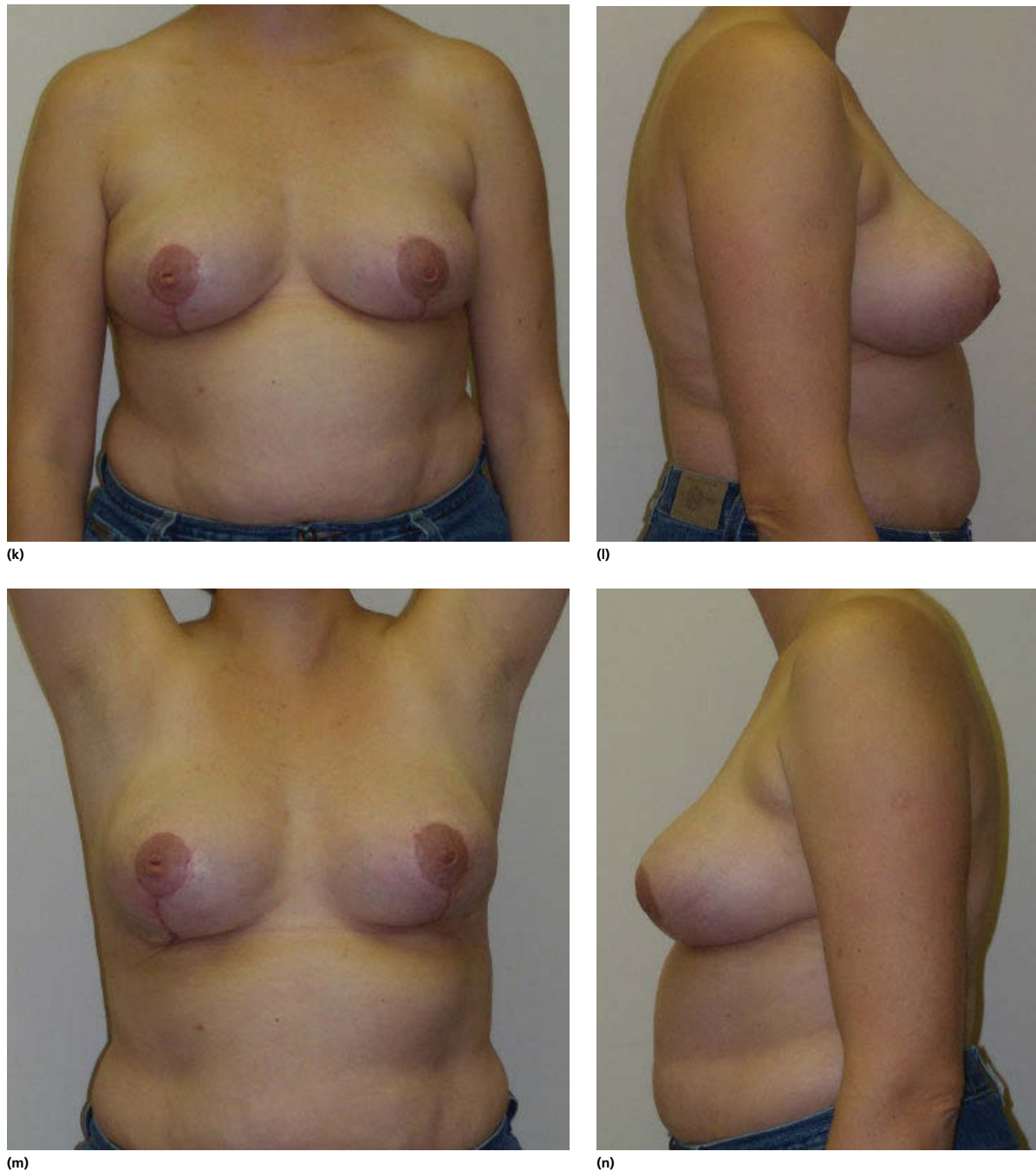


Figure 41.8 (Continued) skin resection with tissue removed inferiorly. The pillars do not extend as far down as the skin opening or as far down as the IMF. The pillars are closed with a few relatively superficial sutures with no tension. The pillars should be sutured only enough to allow them to stay together during the postoperative period so that they can heal together. Deep bites are not necessary and may lead to tissue necrosis. Tension should not be relied upon to shape the breast because it will inevitably fail over time. The key is to remove all tissue where it is in excess. (g) The dermis is closed loosely both vertically and around the areola. Note the shape of the right breast. Good projection is achieved along with narrowing of the breast base as compared with the unoperated left breast. (h) The photograph shows that the breast does not settle by dropping as shown. (i) The final breast shape and extent of excess skin can be shown by using this manoeuvre where the breast is elevated and pushed down so that it slides down the chest wall to its original position. This allows the surgeon to determine if any more subcutaneous tissue or parenchyma needs to be removed but also to assess whether a horizontal skin excision needs to be added. The excess inferior skin in this example will just curve up under the parenchyma around the pillars and does not need to be modified. (j) Final closure. The cross-hatched area shows where the liposuction has been performed (usually after deep dermal closure before subcuticular closure). Liposuction is also performed along the lateral chest wall and (not to excess) in the preaxillary areas. (k, l, m, n) Postoperative photographs at five months of the patient shown above. She was 41 years old, 180 cm tall, weighed 82 kg and wore a 34G brassiere. She had 346 g removed from the right breast and 325 g from the left breast. She had 400 mL of fat suctioned from the inferior breast along the IMF as well as along the lateral chest wall and in the preaxillary areas. Source: Elizabeth Hall-Findlay. Reproduced with permission.

length. It is important for surgeons to change their thinking and realize that a longer vertical length is actually needed to accommodate the increased projection that occurs with the vertical approach. Bottoming-out occurs with the inferior pedicle procedures because the weight of the tissue is left in the lower pole and the skin brassiere stretches; bottoming-out occurs with vertical procedures with inadequate resection of the lower pole (inferior wedge). The skin is allowed to redrape with a vertical approach it is not used as a handle to hold the breast up. Any procedures that use skin (or dermis) as slings tend to fail over time because skin (and dermis) stretches.

There is some tissue overhang in a vertical breast reduction and the vertical length from the lower border of the areola to the IMF is usually about 7 cm in a 'B' cup breast, about 9 cm in a 'C' cup breast and about 11 cm in a 'D' cup breast. It is important to warn patients that they will still be able to 'hold a pencil' underneath their breasts.

The decision to add a 'T', a 'J' or and 'L' can be a difficult one. It will depend on the quality and amount of excess skin. As a guideline, a vertical incision length of up to 12 cm can be tolerated in patients with poor-quality skin and up to 15 cm with good-quality skin for an average 'C' to 'D' cup breast. The way to test to see if the inferior end of the incision will tuck up underneath the breast is to hold the breast up and push it down by lifting and sliding (see Figure 41.8h,i) and if the end of the incision tucks up under the pillars, no further correction is necessary.

Areolar closure

The pedicle is allowed to sit comfortably in the areolar opening and it is usually rotated slightly to fit. Debulking of deep tissue in the superomedial pedicle to allow unrestricted inset can be performed at any time during the procedure.

Four deep 3–0 Monocryl sutures are used to close the dermis and the skin is closed with a subcuticular 3–0 or 4–0 Monocryl. If the skin opening is not completely circular, the skin can be trimmed prior to closure.

The areola will eventually stretch out to match the skin opening circumference. A 16 cm circumference (of the skin opening, not the areola) will match a 5 cm diameter areola. A 14 cm circumference matches a 4.5 cm diameter areola. Areolar skin is quite elastic and will stretch to fit the skin opening. A permanent Goretex type of suture can be used in a circumvertical type of breast reduction (which is used to shorten the vertical length) but at the expense of some stretching that can occur beyond the Goretex pursestring.

Liposuction for tailoring

After the deep dermis is closed, liposuction is easier to perform because there is some resistance to the tissues and because it is easier to visualize the final Wise pattern. Liposuction is performed beyond the Wise pattern to remove any excess tissue (this must be carved out and excised directly in young patients who have more parenchyma and less fat). Liposuction can also

be used to reduce fat in the lateral chest wall and preaxillary areas (Figure 41.8j).

Drains

Drains can be used if desired. If a patient has a large lateral dead space or had an unusual amount of bleeding during surgery then drains might be indicated. Seromas do occur and drains would need to be left in for a long time to prevent seroma formation. The advantage of the vertical approach is that seroma fluid can easily work its way through the tissues with gravity without being blocked by an inframammary incision.

Antibiotics

The breast is often considered 'clean' surgery, but it is well known that breast ducts contain bacteria and it may be best to give a prophylactic dose of antibiotics before surgery (usually a cephalosporin). When I gave patients a full week of postoperative antibiotics, almost all my problems with suture spitting disappeared. Some of those problems recurred when I began using only a single prophylactic dose. The use of triclosan (an antibacterial, not an antibiotic)-impregnated sutures also helped reduce the incidence of suture spitting.

Taping and garments

The incisions are best managed by covering them with paper tape. Steristrips can be used but if placed across the incision, blisters can occur from the shear forces as the breast swells. Tape should be placed along the incision and glue is not necessary. The incisions are then covered with gauze and the patient is placed in a non-compressive surgical brassiere. It is unnecessary (and ineffective) to tape all the breast skin in an attempt to shape the breast.

Patients are allowed to shower the next day. The paper tape will stay in place (without being changed) for 3–4 weeks.

The pucker at the lower end of the vertical incision may swell and protrude for a few weeks and then it will tuck in under the pillars. Some patients may need a revision but this should not be performed for at least six months. Because the parenchyma (and skin) is removed as a vertical ellipse, there is a dog-ear that disappears into the areola and one that can be a nuisance inferiorly. The inverted T, inferior pedicle type of breast reduction results in a horizontal type of excision of skin and parenchyma with a medial and lateral dog-ear – both of which can be a nuisance.

Another example of vertical breast reduction in a 16-year-old patient is shown in Figure 41.9.

Operative technique for vertical mastopexy

It is important to understand that the skin will not act as a good brassiere in a patient who has already developed breast ptosis. An inferior wedge of breast tissue needs to be removed in a breast reduction or small mastopexy. If a patient likes her breast size, then the inferior wedge of breast tissue needs to 'moved'

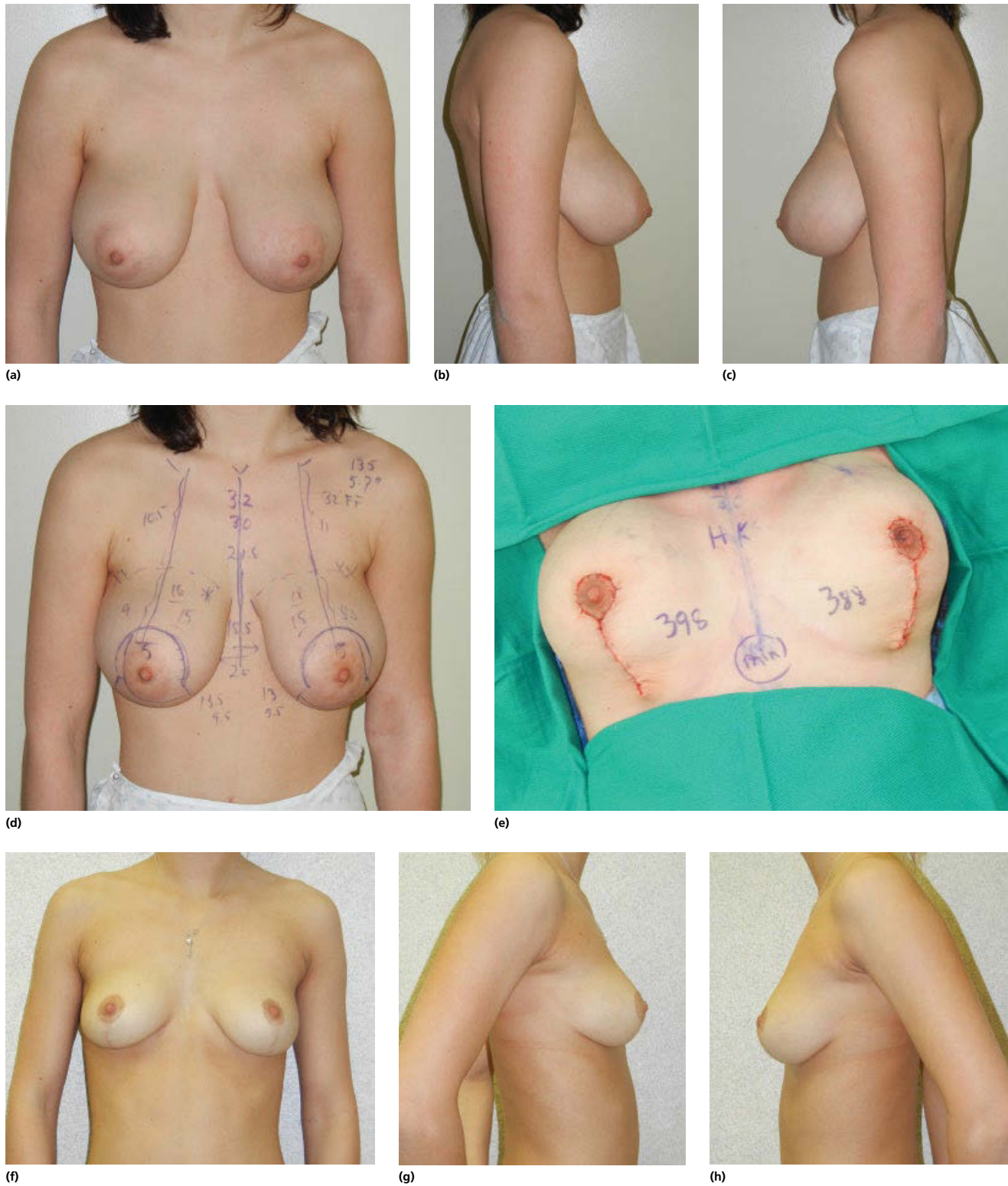


Figure 41.9 Vertical breast reduction. (a, b, c, d) A 16-year-old patient who was 170 cm tall, weighed 61 kg and wore a 32FF brassiere. (e) She had a true superomedial pedicle vertical breast reduction with 398 g removed from the right breast and 388 g removed from the left breast. She only had a minimal amount of fat removed with liposuction. (f, g, h) She is shown just over a year after the surgery. Source: Elizabeth Hall-Findlay. Reproduced with permission.



Figure 41.10 The inferior wedge that is normally removed in a breast reduction is shown as an inferior flap left attached to its chest wall-based blood supply but separated from breast and skin on all four sides to allow good mobilization. Source: Elizabeth Hall-Findlay. Reproduced with permission.

rather than 'removed'. Any attempt to push and hold up the parenchyma with the skin will be doomed to failure.

Ribeiro^{7,8} describes an inferior flap along with a superior pedicle. The flap needs to be separated on all four sides and left as a chest wall-based flap. The flap can be easily mobilized and moved up the chest wall and the medial and lateral pillars can then be closed caudal to the flap to hold it up. The flap is sutured up to the chest wall long enough that breast tissue will heal to breast tissue. The flap does not heal to pectoralis fascia even when sutured up but it will heal to the overlying breast tissue (Figure 41.10).

Any attempt to suture breast tissue to pectoralis fascia will either fail or result in scar contracture that will cause the breast to move unnaturally every time the muscle is contracted. In normal circumstances, this flap can provide extra breast projection and can prevent recurrent glandular ptosis but it will not result in increased upper pole fullness.¹

The blood supply to this inferior flap is the deep artery and vein from the internal mammary system at the fourth interspace. It is important to create the flap while being careful to preserve the attachments to the chest wall just above the fifth rib and just medial to the breast meridian. The flap is de-epithelialized not to maintain blood supply but to serve as a something somewhat substantial to help suture the flap upwards. Either a superior or superomedial pedicle can be used with this inferior flap.

A superior pedicle is often used because a concave inferior aspect to the breast will not occur because the central aspect of the breast is filled with the flap (Figure 41.11). A superomedial pedicle can be used if the nipple and areola have a long distance to travel – where rotation is better than folding.

Graf³⁰ has described a similar mastopexy but using a strip of pectoralis muscle to act as a handle to hold the flap up higher on the chest wall. This does attach the flap to the chest wall but the remaining breast will still tend to drop down around the flap

back into its original position. This flap can improve upper pole fullness to some degree but it can cause a problem in weight-loss patients when the flap stays up and the breast drops down. Some surgeons complain that this procedure puts breast tissue behind the pectoralis muscle, but in reality all that happens is that a strip of muscle acts as a handle over the breast tissue.

Operative technique for vertical augmentation–mastopexy (Figure 41.12)

The problem with the inferior flap in a mastopexy–augmentation is that the blood supply to the inferior flap is damaged when the implant is placed in either a subglandular or subpectoral location. The artery and vein to this flap comes up through the intercostal muscles just above the fifth rib and they are cauterized with insertion of the implant. The procedure can be performed while preserving the flap but it is difficult and skin closure can be a problem.

It is best to perform a 'reduction–augmentation' or 'plus–minus' procedure^{31,32} when both a mastopexy and an augmentation are desired. Some surgeons believe that the mastopexy and the augmentation involve opposing forces, but that is not really the best way to approach the problem.^{33–35} The implant is used to add volume to the upper and central poles of the breast and the inferior wedge resection is used to take care of the glandular ptosis.

A superior pedicle is often used because a concave inferior aspect to the breast will not occur because the central aspect of the breast is filled with the implant. A superomedial pedicle can be used if the nipple and areola have a long distance to travel, where rotation is better than folding.

Closing the skin under tension is not only unnecessary but can lead to wound-healing problems. The skin will not act as a good brassiere and skin tension should not be relied upon to hold the implant up. Even a subfascial inferior sling fails to hold up the implant in the long term.¹ The ptotic gland needs to be removed and patients need to understand that a large implant will be subject to gravity.

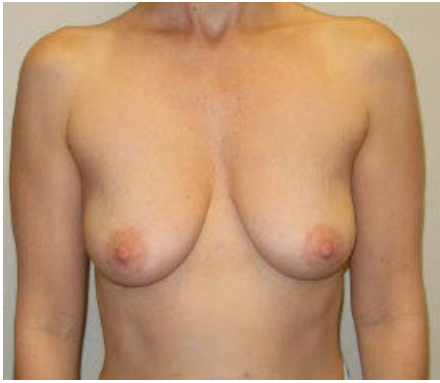
Some surgeons will insert an implant into the subpectoral pocket, first keeping inferior muscle and fascia intact to close up the pocket completely. They then perform the mastopexy so that there is no contact between the implant and the bacteria present in the breast ducts.

Complications

The potential complications are basically the same for all breast procedures: blood loss, infection, necrosis, hematoma, seroma, dehiscence and unsatisfactory aesthetic results.

Blood loss

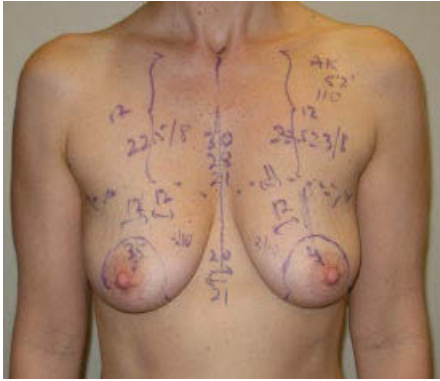
It is unusual to need a transfusion or even an autologous transfusion for most aesthetic breast surgery. Blood loss is higher for the inverted T inferior pedicle breast reductions (about 300–400 cm³) than a vertical reduction (about 100–200 cm³). A vertical



(a)



(b)



(c)



(d)



(e)



(f)

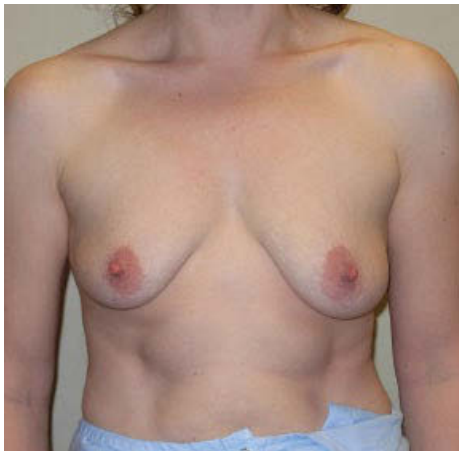


(g)



(h)

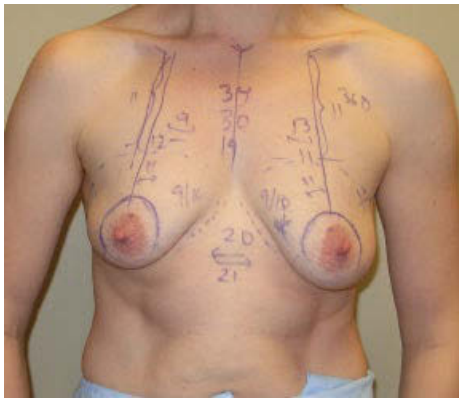
Figure 41.11 Vertical mastopexy. (a, b, c, d) Patient who had a mastopexy using an inferior flap and a superior pedicle to rearrange (rather than remove) the inferior glandular ptosis. (e, f, g, h) She is shown just over a year after her surgery with better projection and a long-lasting shape. Source: Elizabeth Hall-Findlay. Reproduced with permission.



(a)



(b)



(c)



(d)



(e)



(f)



(g)



(h)

Figure 41.12 Vertical mastopexy–augmentation. (a, b, c, d) Patient shown with a superior pedicle vertical mastopexy–augmentation with the implant (Allergan Style 15, each 339 mL) placed in a subglandular pocket. She did not have much glandular ptosis and only 15 g was removed from the right breast and 25 g from the left breast. (e, f, g, h) She is shown 3 years after her surgery. Source: Elizabeth Hall-Findlay. Reproduced with permission.



Figure 41.13 Nipple necrosis. Once established, sometimes nipple and areolar necrosis heals surprisingly well without intervention. The photos are taken at two weeks, six weeks and three months postoperatively. Source: Elizabeth Hall-Findlay. Reproduced with permission.

mastopexy as described here has a blood loss of about 100 cm³ and a vertical mastopexy–augmentation rarely exceeds 100 cm³.

Infection

The breast ducts contain bacteria and breast surgery should not be considered completely ‘clean’. A single dose of prophylactic antibiotics is indicated but postoperative antibiotics should probably only be considered when an implant is used. Even this is controversial. In my experience, suture spitting (festering) is a low-grade infection and it can be prevented to some degree by using antibacterial-impregnated sutures such as Monocryl Plus.

It appears that larger breast reductions and a higher BMI may lead to a higher infection rate.

Necrosis

Nipple necrosis is a definite risk inherent in all breast procedures that involve a transposition of the nipple and areola complex (Figure 41.13). The rate is usually less than 1%. A

good understanding of the blood supply to the breast will help minimize the risk but it is probably inevitable in any large-volume practice. Although there are many recommendations on what to do with incipient necrosis, it is probably best to only intervene if it is possible that there is compression causing obstruction on either arterial inflow or venous outflow. The latter may develop obvious venous congestion. Once it is clear that some necrosis is likely, it is best to leave things alone and let the extent of necrosis declare itself. The NAC seems to be a ‘privileged’ construct and it often recovers far better than expected with no intervention.¹ There are recommendations to remove the nipple–areola and apply it as a free nipple graft but this is probably not indicated in most cases. Removing sutures or taking the patient back to the operating room to inspect the pedicle for compression may be indicated but the surgeon needs to weigh the potential consequences of action versus inaction. Inaction is often a good choice.



Figure 41.14 Dehiscence. Sometimes a dehiscence is best left alone for healing by secondary intention. Source: Elizabeth Hall-Findlay. Reproduced with permission.

Haematoma

All plastic surgery procedures have a risk of hematoma. The rate is probably close to 1% of cases. The best way to prevent a haematoma is to understand the main arterial supply and to check those areas for secure haemostasis before closure. There is an artery that comes up at the fourth interspace just medial to breast meridian along with some branches appearing laterally as well at the fourth interspace. There are some vessels coming up in the subcutaneous tissue at the level of the IMF and there is the superficial branch of the lateral thoracic artery that enters the breast laterally and courses up into the breast, initially deep peripherally and then in the subcutaneous tissue. The veins are even more superficial and usually lie just beneath the dermis. Drains do not prevent or treat haematomas but they may be indicated if there is an unusual amount of 'ooze' that is evident during surgery.

Seroma

Seromas occur where there is a lot of dead space and where there is some friction between the overlying flap and underlying chest wall. Drains are needed for as long as shearing movement allows friction to occur. This can range from several days to

weeks. Quilting sutures are very effective in the abdomen but somewhat harder to use along the lateral chest wall.

Dehiscence

This can occur when sutures fail to hold. More frequently the problem is excess tension on the closure, which leads to constriction of blood supply to the skin edges (Figure 41.14).

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PART VI

Trunk and lower limb

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CHAPTER 42

Chest wall reconstruction

Anatomy and physiology of the thoracic cage

The thoracic cage consists of 12 pairs of ribs articulating with the facet joints of the 12 thoracic vertebrae posteriorly and, via their respective costal cartilages, to the manubrium and sternum anteriorly. The costal cartilages of ribs 1–7 ('true' ribs) articulate directly with the manubrium/sternum, whereas those from ribs 8, 9 and 10 ('false' ribs) unite as a synchondrosis joining the 7th costal cartilage to articulate with the inferior part of the sternum. Ribs 11 and 12 have no anterior articulation and are known as 'floating' ribs. Between the ribs are three layers of intercostal muscles, external, internal and innermost, with a neurovascular bundle running just below each rib between the internal and innermost layers. The thorax is bounded inferiorly by the diaphragm and superiorly by the clavicle.

This arrangement of the thoracic cage confers both strength, to provide protection for the thoracic and upper abdominal viscera, and flexibility, to allow the movements required for ventilation. During inspiration, contraction of the external intercostal muscles elevates the ribs, which, given their curved shape and fixed points anteriorly and posteriorly, allows the thoracic cage to swing up and out (analogous to the oft-quoted movement of a bucket handle). This, together with depression of the diaphragm, increases the thoracic volume, thus creating the negative pressure to draw air in. As the diaphragm and the external intercostals relax, thoracic volume decreases and air is expelled. The internal intercostals, which actively depress the ribs, are only used during forced expiration.

CONGENITAL DEFECTS

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Congenital chest wall anomalies may present at any age up to adolescence and may occur in isolation, or represent one marker in a spectrum of defects.

Pectus deformities

In this spectrum of malformations, there is an abnormal contour of the anterior chest wall. It is thought the underlying problem lies with the relative rate of growth of the costal cartilages in relation to the growth of the ribs and sternum. If the elongated cartilages cause the anterior chest wall to buckle inwards, pectus excavatum results. If there is buckling outwards, the result is pectus carinatum.^{1,2} However, some have proposed that the defects arise from abnormal growth of the diaphragm.³

Both conditions may be symmetric or asymmetric in nature, with a varying composite of anomalies, such as sternal angulation, tilt and rotation about the long axis – all of which need to be taken into account during cosmetic correction. Within the broad spectrum, some patients may even have a mixed defect.

Pectus excavatum

In this condition, there is a depression in the anterior of the chest wall of varying shape and severity (Figure 42.1).

Morphologic varieties

In most cases, the depression is symmetrical on either side of the midline and limited to the lower half of the sternum. More severe forms are deeper and extend further upwards toward the manubrium. At the more severe end of the scale is the so-called 'Grand Canyon' variety, where there is a deep and long depression. It can be a challenge to treat successfully, particularly since many of these are asymmetrical. In about 5% of cases, there is a combined excavatum/carinatum deformity.

Epidemiology

The incidence of the defect is 1 in 150–1000 live births.¹ Although the defect may present any time after birth, most patients present around the time of puberty during their phase of rapid body growth. There is also an association with connective tissue disorders such as Marfan's and Ehlers–Danlos syndromes, where scoliosis is also more frequent.⁴

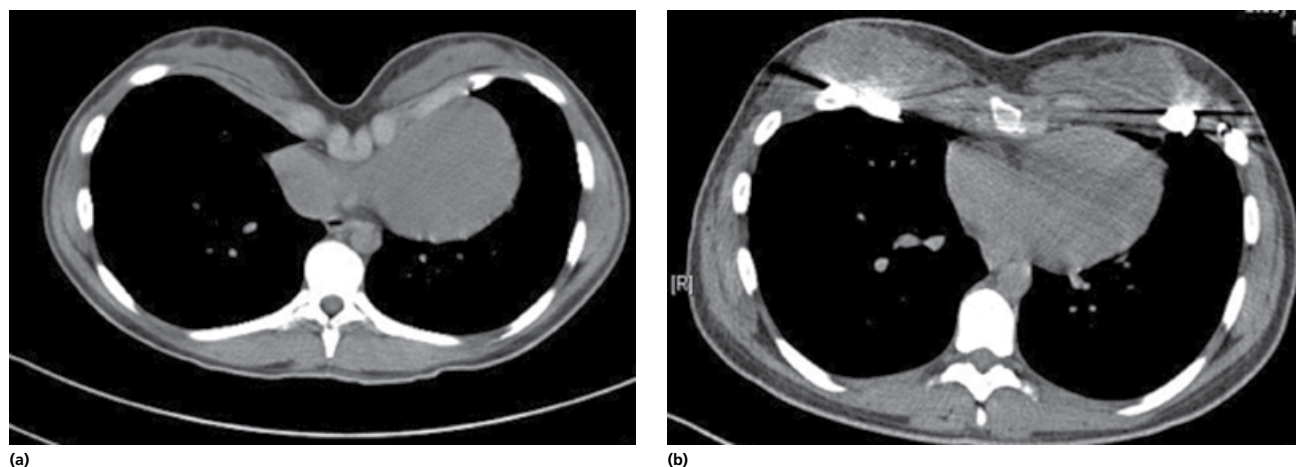


Figure 42.1 Transverse section of a CT scan of a patient with pectus excavatum, before (a) and after (b) Nuss correction. Note the cardiac compression seen before correction.

Clinical evaluation

Prior to surgical correction, we undertake a number of investigations to determine the severity and extent of morphology, as well as impact on physiology.

Imaging

We routinely perform a plain chest radiograph that demonstrates the overall shape of the thoracic cavity. This is followed by CT imaging, which can be used to construct the 3D anatomy of the thoracic cavity.⁵ The Haller⁶ index of severity can be calculated from this. This is the ratio of the transverse diameter and the anteroposterior diameter of the rib cage. A normal ratio is 2.5. Of special importance is the shape and axis of the body of the sternum. Sternal torsion and asymmetry have an impact on the type of surgery. Imaging can also show the extent of cardiac compression.

We also routinely perform transthoracic echocardiography to assess overall ventricular function. There is also an association with mitral valve prolapse, which may be seen with connective tissue disorders.⁴

Functional assessment

As part of our initial evaluation, we also perform exercise testing of the patient. This is following some reports of a modestly reduced functional capacity arising from the combination of right ventricular compression and reduced pulmonary capacity in some patients.^{7,8}

Surgery for pectus excavatum

Timing

There are advantages and disadvantages to both early and late repair.⁹ Each case is considered individually. Important factors to consider include the age at presentation, symptoms, severity of the deformity and type of surgical approach. As a general rule, younger patients have a more malleable chest, which will make the minimally invasive Nuss procedure an attractive

option here. However, this has to be tempered with the fact that some of these children will outgrow their bar and need further bar insertion. Also, early bar removal in younger patients may result in recurrence as the chest continues to remodel with age. Nevertheless, the severity will reduce the age at intervention.

In the literature, surgery has been performed successfully from the ages of 1 year to 50.^{10,11} In our experience, for the reasons noted above, the ideal is around the age of puberty.

Influence of morphology

We reserve the minimally invasive Nuss procedure for symmetric cases.¹² If the symmetry extends to a so-called Grand Canyon type deformity,¹³ we may either use two Nuss bars, or elect to perform an open sternochondroplasty.¹⁴ If it is likely that any second bar needs to be inserted high up, we favour an open approach due to the risk of placement over the great arteries in the mediastinum. In some cases with mild asymmetry, the Nuss bar can be moulded appropriately.

Nuss procedure

The patient is usually positioned supine on the operating table with the arms abducted following induction of anaesthesia and epidural catheter placement.

Once positioned, necessary landmarks are drawn on the chest: the deepest part of the chest which indicates the level where the bar will lie, the point beneath the nipples at the level of where the bar will lie, and the two lateral points where the vertical skin incisions are made for the introduction of the bar and stabilizers.

The two lateral skin incisions are made, and the subcutaneous plane is developed to the marked point beneath the nipples at the level of where the bar will lie. These will be the points at which the bar will exit the chest wall on either side. Then, the thoracoscope is introduced into the right pleura through a port placed either just beneath or into the lateral skin incision. Carbon dioxide insufflation permits exposure. Next, the bar

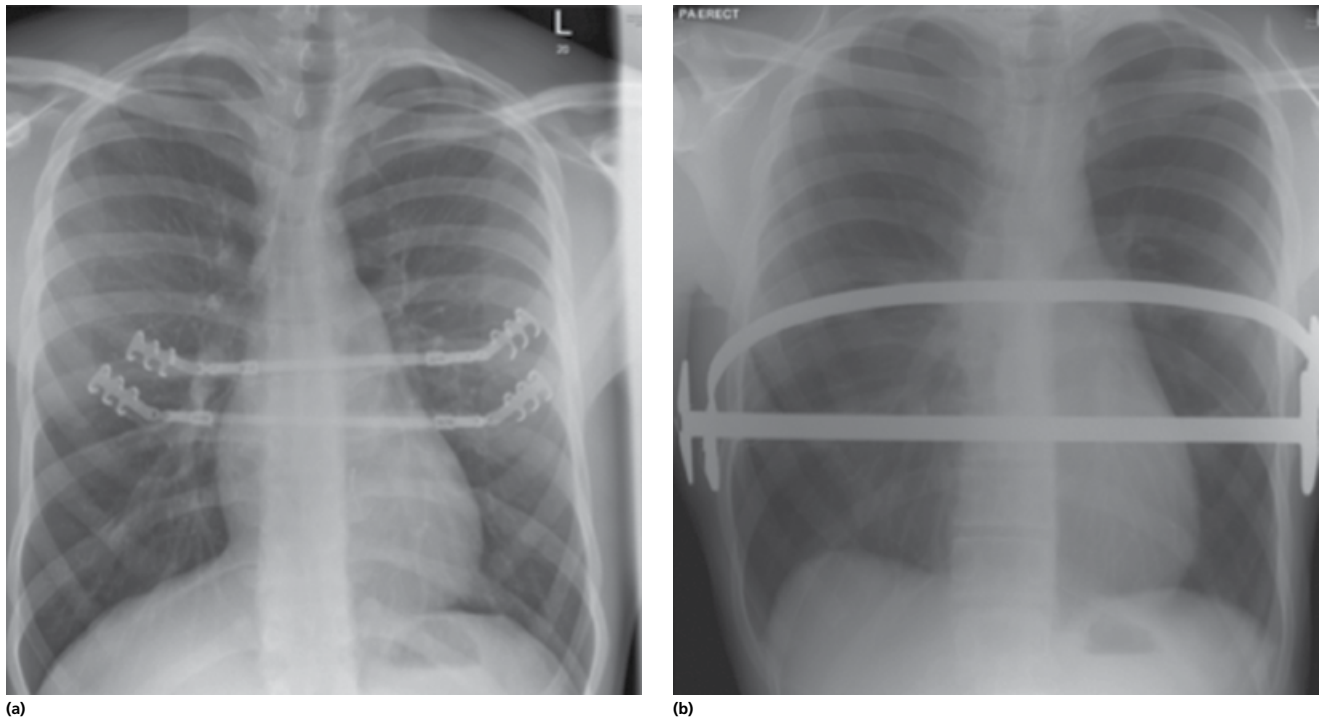


Figure 42.2 (a) STRATOS bars in place for stability following repair of pectus carinatum. (b) Two Nuss bars *in situ* following repair of pectus excavatum.

introducer creates a subcutaneous tunnel from the skin incision to the point of entry into the chest. Entry through the chest wall is observed via thoracoscopy. The introducer is passed to the pleural cavity on the other side, anterior to the pericardium under direct vision. It is guided through the chest wall. Once safely out of the skin, it is tied by umbilical tape to a moulded Nuss bar of appropriate size. As the introducer is removed, the bar is carried with it into the chest. The bar is then finally flipped with a specially designed flipper and secured to the chest wall by bilateral stabilizers secured to it by steel wire (Figure 42.2b).

Following the procedure, the patient is extubated on table and a chest radiograph is taken immediately to check for pneumothoraces and bar position.

Once on the ward, the patient is observed in the high-dependency unit for the duration of the epidural catheter – usually 4–5 days. Adequate analgesia is very important here once the epidural is removed. Mobilization aided by physiotherapy is the final step before discharge. A final PA and lateral chest radiograph check the bar position.

Bar removal

We elect to remove the bar after 3 years. Although the bar can be left in for 4 years without problems, the longer it is left, the greater the degree of calcification around the bar, which can make removal more challenging. Removal is performed as a day-case procedure under general anaesthesia. The greatest risk comes from pneumothorax, and there are also isolated reports of injury to great vessels.¹⁵

Pectus carinatum

This is also known as pigeon or keel chest. In this condition there is variable protrusion of the chest.¹⁶ It is less common than pectus excavatum, being seen in about 0.06% of all live births¹⁷ (Figure 42.3).

Morphologic varieties

Depending on the exact extent of the protrusion, there are a number of variations:

- Symmetrical – The sternum is arched forwards. If there is some depression of the costal cartilages, the sternal prominence is exaggerated. In some cases, the body of the sternum fuses with the xiphoid at a 90° angle.
- Symmetrical deformity with a normal sternum – Here, the anterior prominence comes from protrusion of the costal cartilages with a relatively normal sternal contour.
- Asymmetry with combined carinatum and excavatum – Protrusion of costal cartilages on one side only causes rotation of the sternum about its long axis, causing a relative excavatum on the contralateral side.
- Pouter pigeon chest – There is prominence of a thickened maubrium so that the deformity is limited to the upper sternum only.

Timing of operations

This deformity becomes most apparent during adolescence, when the infantile potbelly recedes to make the anterior chest more prominent during the period of rapid body growth.

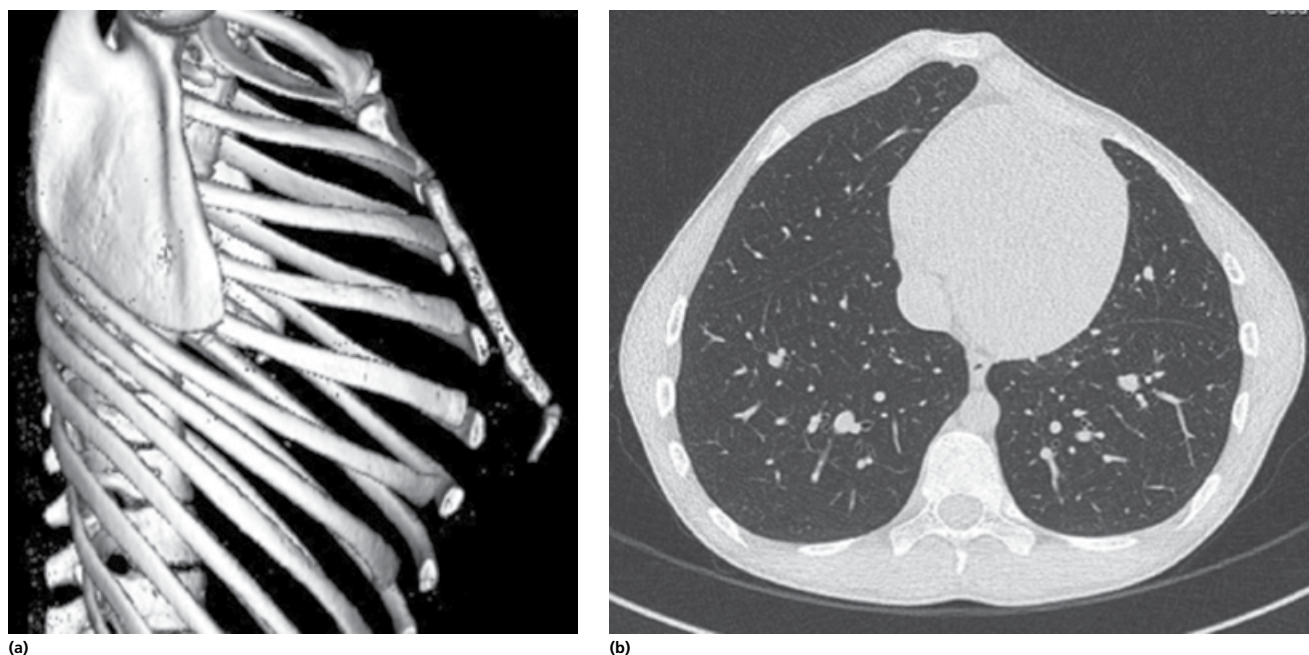


Figure 42.3 Pectus carinatum as seen on 3D CT reconstruction (a) and transverse CT scan (b). Note the 90° angulation between the body of the sternum and xiphisternum.

Although we do not have a distinct cut-off age, surgery is mainly reserved after this final adolescent growth-spurt has begun and chest wall natural chest wall modelling is complete.¹⁸

Reconstructive aims

The main aim of surgery is to remodel the anterior chest wall to the normal contour through trimming the elongated costal cartilages and returning the sternum to its natural alignment. Surgery will provide an immediate result, but in order for the remodelling to be complete, we encourage physiotherapy and muscle-building exercises to enhance the muscular contribution to chest wall shape.

Technique of repair

Our method of repair is based on that of Ravitch¹⁴ but with the addition of chest wall stabilization using the Strasbourg Thorax Osteosyntheses System, MedXpertGmbH, Heitersheim, Germany (STRATOS™) system of costal bars^{19,20} (Figure 42.2a).

The patient is positioned in the supine position, with the arms by the side. We prefer to mark the skin first – indicating both the line of incision and the most prominent areas of cartilage/sternum. The incision is horizontal at the level of the xiphisternal junction, being some 10 cm with a gentle upwards curve. The subcutaneous plane is developed, at first superiorly to the highest cartilage involved then inferiorly to the xiphisternum. This is seldom above the 3rd rib. Then the pectoralis major muscles are detached, sparing their clavicular origins. Care must be taken not to enter the intercostal spaces, especially in thin patients. Next, the plane inferior to the incision is developed. The uppermost origins of the rectus abdominis muscles

may have to be detached if the underlying cartilages are involved. At this point, the xiphisternum is also detached from the body of the sternum with diathermy.

Once the underlying involved costal cartilages and ribs are exposed, resection can begin. The cartilages are initially detached at the sternocostal joints through a combination of diathermy and sharp dissection. Care must be taken not to damage the underlying internal thoracic vessels, or parietal pleura. In order to preserve growth potential in the juvenile sternum, we try to preserve the underlying perichondrium when shelling out the exuberant cartilage. Generally, the lower the cartilage level, the greater the lateral extent of resection required. The lateral extent of resection will be determined by the final position of the sternum.

A sternal osteotomy is performed at a level determined by the angle and position of protrusion. This allows the sternum not only to be straightened, but also rotated appropriately to the normal anatomic plane. The sternum is fixed in the correct position using absorbable suture, or stainless steel wires depending on the size of the patient.

Next, the free ends of ribs are fixed to the lateral edge of the sternum with a figure-of-eight absorbable suture, making sure that they lie flat without protrusion. The xiphisternum is also reattached to the body of sternum with absorbable suture.

If there has been multilevel involvement with the risk of chest wall instability, we add STRATOS titanium rib bridges to provide final stability.

The muscles are reapproximated, ensuring that the two pectoralis major muscles on either side are brought together in the midline. This will help determine the final contour of the

chest, and allow better healing of the bone beneath. Two drains are inserted and placed in the subcutaneous plane. If the pleural cavity has been entered, a suction catheter is placed at the end of the case to evacuate air. The rest of the tissues are closed in layers and dressing applied. Once in the recovery room, a chest radiograph is taken to ensure there is no pneumo- or haemothorax.

Poland's syndrome

Poland's syndrome is a rare congenital anomaly characterized by hypoplasia of the breast and nipple, absence of the costosternal portion of the pectoralis major muscle, absent pectoralis minor, aplasia or deformity of the costal cartilages and ribs 2–4 or 3–5, alopecia of the axillary and mammary region, and unilateral brachysyndactyly.²¹

Epidemiology and aetiology

The incidence of Poland's syndrome ranges from 1 in 7000 to 1 in 100 000 live births. Males are affected more frequently with a 2:1 to 3:1 ratio. The right side of the body is involved in 60–75% of patients.²²

There are a number of theories as to why it develops. One suggests that there is hypoplasia and interruption of the subclavian artery during the 6th week of development.²³ Another theory suggests a disruption of the lateral plate mesoderm affecting the development of the upper limb girdle.²⁴ There is an association with Klippel–Feil syndrome²³ and Möbius syndrome, with bilateral congenital facial nerve palsy and paralysis of the eye abductors.²⁵

Clinical manifestations

Although there is some variation upon the theme, the most consistent feature is the absence of the sternocostal head of the pectoralis major muscle. The underlying pectoralis minor may be absent altogether. Other chest wall muscles, such as the latissimus dorsi and serratus anterior, may also be absent – which is important when considering surgical soft tissue coverage. In up to a third of females there is absence of breast development.

Absence of underlying ribs and costal cartilages causes the ipsilateral chest to be sunken. The most frequently absent or hypoplastic ribs are 2–4 or 3–5. As a result, upwards of 10% of patients may also have lung herniation and chest wall instability, leading to paradoxical movements during respiration.²¹ Hand abnormalities in Poland's syndrome include shortness of the middle phalanges with cutaneous webbing (or even full syndactyly), to complete absence of the hand (ectrodactyly). These tend to be ipsilateral to the chest wall anomalies.

Reconstructive aims and indications for surgery

Surgical intervention may be indicated for the following reasons:¹⁷

- unilateral depression of the chest wall with cosmetic implications;
- lack of adequate protection of the heart and lung with lung herniation;

- chest wall instability with resulting paradoxical chest movement;
- hypoplasia or aplasia of the female breast.

Surgical management

This will be influenced by the age and sex of the patient, as well as pattern of involvement. Both the chest wall contiguity and muscular coverage need to be addressed. Many have advocated a two-stage approach in children – first repairing the chest wall defect, followed by a second procedure, which provides chest wall soft tissue coverage and sculpting of the absent anterior axillary fold.^{26,27} A single-stage approach may be reserved for older children and adolescents.

The approach is via a transverse incision, which will provide excellent access not only to the rib defects, but also to the sternum, which may be rotated as part of the constellation of defects.¹⁷ A subperichondral resection of the costal cartilages is performed of the affected rib ends. A sternal osteotomy is performed to allow it to be rotated to the corrected plane. Where there is rib aplasia, rib grafts may be used to bridge the defect. Synthetic mesh has also been used to provide a wider coverage.²⁷ We have, however, in more recent cases been using artificial bone or STRATOSTM bars to bridge the defect and provide chest wall stability.

A musculocutaneous flap of the latissimus dorsi can be used for coverage and provide reconstruction of the absent axillary fold. In our centre, such cases are performed as joint procedure between the thoracic and plastic surgeons.

Sternal cleft

This is a rare malformation characterized by failure of fusion of the sternum in the midline. It may be partial, where there is a U-shaped dip at the upper end of the manubrium, to a more extensive defect extending all the way to the xiphisternum, where there are two bars of bone to which attach the costal cartilages.²⁸

This defect in the sternum may occur in isolation, but it may also herald associated anomalies, such as cardiac malformations.²⁹

Surgical approach and timing

Our preference is to repair this defect in the neonatal period, or at least as early as possible, as the more pliable tissues allow for a better chance for primary repair with autologous tissues.

The repair is carried out via a midline vertical incision and the subcutaneous plane developed over the sternum and adjoining costal cartilages. A wedge osteotomy is performed of the bony bridge between the two sternal bars. This will allow better mobilization when bringing the two sides together without tension.

If the two sides of bone can be brought together, they may be sutured together with either stainless steel wire, or else a heavy absorbable suture. If there is tension laterally, the sternocostal joints may have to be released and resutured or bridged with STRATOS bars.

We close the soft tissues over the bone by suturing the pectoralis major, rectus abdominis and sternocleidomastoids to their counterparts in the midline. The subcutaneous tissues and skin are then closed primarily to complete the operation.

Jeune syndrome: asphyxiating thoracic dystrophy

This autosomal recessive condition leads to characteristic skeletal abnormalities in the form of generalized chondrodysplasia, limb defects, mild dwarfism and, importantly, a constricted chest. This latter feature makes the resulting physiological problems particularly difficult to manage.³⁰

Epidemiology and clinical features

The incidence is 1:100 000 to 1:130 000 live births. Since 2006,³¹ this has been classified under the umbrella of short rib skeletal dysplasias with or without polydactyly. The natural history of the condition is that the majority of patients do not live beyond the age of 10, mainly from the complications of respiratory or renal failure, which are some of the extraskeletal manifestations of the disease.³²

Chest wall morphology

Failure of normal costal cartilaginous growth leads to cessation of normal chest wall expansion and a persistently narrowed, bell-shaped rib cage. The result of the defect is that the ribs and costal cartilages are shortened, with a fixed horizontal orientation. This results in failure of normal lung development with resulting respiratory failure and pulmonary hypertension, recurrent chest infections and restrictive lung disease.³³

Chest wall surgery

There is no easy surgical solution to the chest wall defects seen in this condition. A number of approaches have been attempted, often through staged interventions.

Lateral thoracic expansion was first outlined in 1995 by Davis *et al.*³⁴ as a staged approach for patients who are older than 12 months. Dissection is carried down to the beds of the 4th to 9th ribs. Those ribs are divided along their shaft, and their adjoining intercostal muscle released. This will permit the rib to be connected to the rib below by a titanium plate in order to expand the chest. Additional chest wall support is obtained by uniting the subperiosteal tissue of rib bed to its counterpart below. In those in whom this technique has been used, new bone formation has been demonstrated.³⁵

Less frequently, there are reports of the modified Nuss technique being used to provide expansion. In the former technique, two bars are implanted bilaterally to provide expansion in the lateral plate. It has been reserved for older children and is limited by the need for a bar change.³⁶

More recently, thoracoplasty using the vertical expandable titanium rib technique has been described.^{37,38} A vertical titanium device is anchored bilaterally to ribs 2 and 10. Ribs 3–8 are then transected and pulled laterally, and finally connected to the titanium device. Growth is allowed by progressively adjusting the device.³⁸

ACQUIRED DEFECTS

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Pathology of chest wall defects

A 'flail segment' or paradoxical chest movement occurs when a segment of chest wall is discontinuous with the remainder of the thoracic cage (classically two or more ribs fractured in two or more places). As the rib cage swings up and out during inspiration, this segment will be 'sucked in' by the increasing negative pressure in the thorax. A similar effect is produced with larger chest wall defects as the overlying soft tissues move paradoxically in comparison with the rest of the chest, compromising ventilation.

Acquired defects of the chest wall invariably result from one of four pathologies: sternal dehiscence following cardiac surgery, tumours, radiation necrosis or trauma. The aetiology has important reconstructive implications both locally in terms of resection margins and quality of local tissue, and in terms of the patient's general health and planned adjuvant treatments.

Sternal dehiscence following median sternotomy Background

Fifty years ago it was the high mortality rate of sternal wound dehiscence, over 50% in early series, that contributed to a delay in the midline sternotomy incision³⁹ gaining universal acceptance amongst cardiac surgeons. At that time, treatment involved debridement and packing, leaving the sternal wound open to granulate. These patients required prolonged mechanical ventilation as a result, with large numbers succumbing to bypass graft failure due to exposure.

The advent of early, aggressive wound debridement and obliteration of the dead space by importing fresh, well-vascularized tissue was introduced by in 1976 by Lee *et al.*,⁴⁰ who transposed greater omentum into the sternal defect. In 1980, Jurkiewicz reported the successful use of muscle flaps in a small series of sternal dehiscences where closed catheter irrigation had been unsuccessful.⁴¹ This became the basis for modern-day treatment and has resulted in the attendant mortality falling to 8–15%.⁴²

Aetiology and classifications

Wound dehiscence occurs in 1–3% of median sternotomies and, although often attributed to an infective aetiology, the underlying problem is devascularization of a hemisternum secondary to the harvest of the internal mammary artery (IMA). Bilateral IMA harvest increases the risk of sternal wound complications significantly up to 7%.⁴³ Those patients with concurrent vascular compromise, such as diabetes, are the least tolerant of the reduction of hemisternal blood supply as a result of IMA harvest. Conditions such as obesity, immunosuppression, previous chest wall irradiation, chronic obstructive airways

disease, congestive cardiac failure and smoking all elevate the risk of sternal wound dehiscence.

The commonest organisms cultured in infected sternal dehiscences are *Staphylococcus epidermidis* and *Staphylococcus aureus*, although a range of gram-negative bacteria have also been isolated.

The two most widely used classifications of sternal defects are outlined in Table 42.1. Like most classifications that attempt to rigidly categorize clinical presentation, neither is perfect, but both can aid management decisions. Although it supplies useful clinical information, a drawback of Pairolero and Arnold's classification⁴⁴ is it fails to specifically identify those early wounds where there is a significant degree of devascularization, or those that are longstanding but with reasonable vascularity.

Starzynski's classification⁴⁵ is anatomical, defined by the location of the sternal defect and thus can only be made after intraoperative assessment. The authors use the classification to predict the degree of physiological compromise in terms of loss of mechanical integrity of the thoracic cage, though it can also be useful in guiding the choice of flap for reconstruction.

Management principles – planning

When assessing a patient with sternal wound problems the reconstructive surgeon should have a clear understanding of the cardiac procedure performed and the implications of that procedure for chest wall vascularity, subsequent debridement and reconstruction requirements. A full history of any thoraco-abdominal procedures that might influence the reconstructive options should be taken. It is particularly important to carefully assess the general wellbeing of the patient with formal anaesthetic consultation. On occasion, patients will not be fit enough for immediate reconstruction, or even for radical debridement, and immediate washout and period of optimization may be the only option.

Patients with sternal wound breakdown will have many complex medical requirements and many will have been hospitalized for prolonged periods of time, so an assessment of the patient's nutritional status and haemoglobin level is vital.

Table 42.1 Pairolero's classification and Starzynski's classification of sternal defects

Pairolero	Presentation	Timing
Type I	Serosanguinous discharge only	0–3 days
Type II	Cellulitis and purulent mediastinitis	3 days–3 weeks
Type III	Chronic sinus and osteomyelitis	Months–years
Starzynski	Anatomical involvement	Physiological deficit
1	Upper sternal body and adjacent ribs (2–5)	Mild
2	Entire sternal body and adjacent ribs (2–7)	Moderate
3	Manubrium, upper sternal body and ribs (1–5)	Severe

In septic patients, specimens of wound fluid and pus should be sent for assessment at the earliest opportunity. Patients should also be carefully screened for coexisting sepsis with appropriate culture of sputum, urine and blood. Plain chest radiographs, while giving limited information about the state of the sternum, can identify a coexisting empyema or pneumonia. In patients where an extensive debridement and reconstruction is contemplated, the benefits and implications of the ongoing use of anticoagulant therapy should be considered and discussed with the cardiac surgeons and the anaesthetists.

CT scans can provide useful information in selected cases, especially those of a chronic nature, with regard to extent of bony necrosis.

Management principles – debridement

Following a washout the state of the tissues should be carefully assessed. The pathophysiology of this condition is reflected in the relative health of the two hemisternums in cases where one side has lost its internal mammary vessel while the other retains its blood supply. While the vascularized segment remains healthy and supports the sternal wires, the devascularized segment will frequently be soft and fragmented; there is often evidence of pus in the tissues and the wires will easily cheesewire through the bone. A thorough debridement of all devascularized tissue should be performed. This will typically involve removing the involved segments of the sternum and associated costal cartilages. It is very rare that there is a need to undertake any soft tissue debridement, although in cases where fat sutures have been used to close the original sternotomy it may be necessary to remove small areas of fat necrosis.

When undertaking a debridement, the reconstructive surgeon must be aware of the position of any grafts and should take great care when mobilizing the sternum. At the completion of debridement it is important that the bone edges should be assessed for sharp spicules that would have the potential to cause damages to the underlying tissues. If a negative pressure suction dressing is to be used, the surgeon must be aware that once suction is applied the bony edges are stabilized by the dressing and that any reorientation of the leading edge has the potential to cause damage to the underlying pericardium and myocardium. Care should be taken to protect the myocardium from the edges of the debrided bone by careful orientation of the sponge. During debridement, multiple soft tissue and bone samples should be sent for microbiological culture.

Surgical tip: Healthy sternum is difficult to debride, any bone that is removed with relative ease or crumbles in the jaws of a pair of bone nibblers is not healthy.

The patient's physiological state at the time of surgery, the extent of the debridement and the surgeon's confidence of its completeness will all influence whether reconstruction is undertaken immediately or whether it is felt wise to delay. In patients who are deemed too unfit for an extensive reconstructive procedure or in cases where there is some doubt as to the extent of devascularization (often the case of those on large doses of pressor support), the introduction of the negative pressure

vacuum dressing has transformed management. If used with care, this dressing allows those patients to be optimized while the wound is mechanically stabilized and oedema and bacterial loads are reduced. When used in open chest wounds, vacuum dressings should only be used by those with experience and an understanding of the risks involved.^{46,47}

Management principles – negative pressure therapy

Negative pressure therapy, in addition to its wound management properties, also serves as a remarkably effective splint for the sternal defect, restoring some degree of mechanical integrity to the thoracic cage and allowing early mobilization and physiotherapy.

If negative pressure therapy is employed, a suitable foam (e.g. Whitefoam (polyvinyl alcohol, KCI)) or an interface dressing, such as Mepitel (Molnlycke Healthcare, Sweden), should be used. This reduces the direct pressure on the thin-walled right ventricle and helps to prevent adherence of the sternum to the exposed myocardium (implicated as a causative factor in the rare instance of myocardial rupture). Following cardiac surgery, adhesions will develop between the myocardium in areas where the pericardium has been reflected and the anterior mediastinum, it is important to be aware that when suction pressures are applied, shearing forces are introduced to the tethered areas. To minimize the resultant damage we free as many of the adhesions as possible, and typically limit suction pressure to 75 mmHg.

Chest wall tumours

Tumours account for a large proportion of cases of chest wall defects, and can be broadly split into primary chest wall neoplasms, invading adjacent tumours (e.g. lung, breast, skin) and metastatic lesions. Advances in medical therapies for these cancers in both the adjuvant and neoadjuvant setting has led surgeons to undertake increasingly large surgical resections. These extensive procedures are only possible with adequately robust chest wall reconstruction.⁴⁸

Management principles – planning

Patients with complex chest wall tumours are best managed in a multidisciplinary team (MDT) setting. Prior to reconstruction it is important to consider the prognosis of the disease and plans for the use of adjuvant therapy. The medical fitness of the patient and the state of the local tissues, particularly following previous surgery or radiotherapy, will all influence reconstructive options.

Management principles – resection

Whether the resection is performed by the thoracic surgeon or the reconstructive surgeon, it is helpful if the surgeon planning to undertake the reconstruction is closely involved with this process to ensure that the flap and its vascular supply are protected.

Osteoradionecrosis

Advances in oncological management, in particular in the treatment of breast cancer, have led to an increasing number of patients undergoing irradiation of the chest wall. Although

more sophisticated techniques have improved therapeutic targeting, radiotherapy still damages surrounding bone and soft tissues, both through direct cellular effects (particularly damaging fibroblasts and osteocytes) and through impairment of tissue vascularity. It is worth noting that osteoradionecrosis can present years after radiotherapy, often as non-healing ulcers or pathological rib fractures.

Chest wall reconstruction associated with radiation necrosis provides considerable challenges as both the compliance and vascularity of local tissues is compromised. The extent of radiation injury is often extensive and good judgement is required to determine how to balance the need to resect compromised tissue and yet maintain stability and minimize reconstructive requirements. The quality of both local flaps and local blood vessels for free tissue transfer should be carefully assessed prior to the procedure.

Trauma

High-energy injuries involving blunt trauma to the chest wall can lead to a flail segment that may require stabilization, however it is relatively rare that a segment of chest wall requires formal reconstruction. The priorities in these patients are the assessment and treatment of life-threatening injuries, in particular any concomitant lung or mediastinal injury, but the reconstructive principles remain the same.

Reconstruction of chest wall defects

When planning reconstruction the surgeon should consider the following:

- Is the thoracic cage stable, does it require support to restore mechanical integrity?
- Which flap (or combination of flaps) will best achieve obliteration of dead space and durable soft tissue coverage for the least donor site morbidity?

Thoracic cage stability – restoring mechanical integrity

Classical teaching states that resection of four or more ribs or the creation of a defect >5 cm diameter will require bony support to prevent a flail segment and preserve pulmonary compliance. In these large defects, bony reconstruction has the added advantage of protecting the underlying viscera. This principle has been successfully applied in most major series⁴⁸ and has stood the test of time, although there are certain anatomical areas that require specific consideration. The scapula stabilizes the upper part of the posterior chest wall and larger defects (up to 10 cm) in this area may not necessarily need further rigid support. Defects in the anterior chest wall in the region of the first three ribs are splinted by the sternum, clavicle and surrounding musculature, so larger resections may likewise be tolerated without the need for stabilization. An example is a defect resulting from partial or complete sternectomy, where infection typically contraindicates

Table 42.2 Prosthetic materials used to restore thoracic cage mechanical stability

Synthetic meshes/composites	Prolene (polypropylene)
	Marlex (polypropylene and high-density polyethylene)
	Gore-tex (expanded polytetrafluoroethylene)
	Vicryl mesh (polyglactin 910)
	Composite Merlex/Prolene and methylmethacrylate sandwich
Biomaterials	Alloderm (allograft)
	Strattice (porcine xenograft)
	Permacol (porcine xenograft)
	Surgimend (bovine xenograft)

the introduction of support with prosthetic materials or grafts. In these cases, the mechanical integrity of the chest wall is rarely permanently affected, although the patient respiratory capacity is frequently, but temporarily, compromised until a fibrous union has re-established stability.

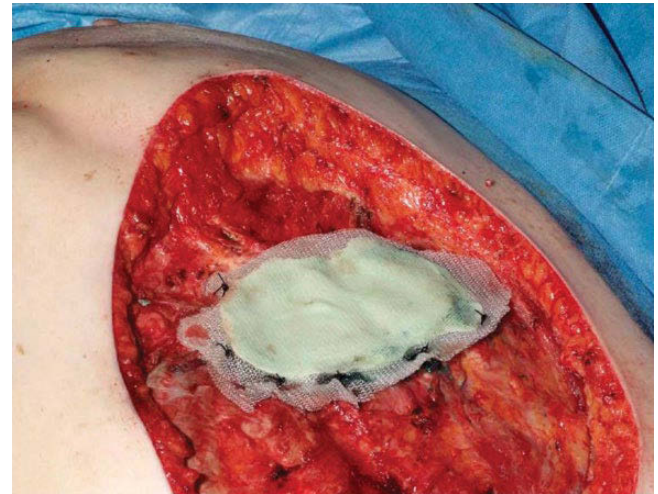
Choice of bony support

It is the re-establishment of the mechanical integrity of the thoracic cage that is pivotal for chest wall reconstruction. The use of prosthetic materials aptly demonstrates this and the development of these products has transformed the reconstruction of the chest wall skeleton. These prosthetic implants conform well to the contour of the chest wall defect, avoid the additional morbidity of a donor site and enable reconstruction of much larger composite defects than could be achieved with autologous material alone. They can be broadly split into synthetic meshes/composites and biomaterials and are outlined in Table 42.2.

Both Prolene and Marlex are non-absorbable, synthetic meshes with good tissue incorporation. For added rigidity, two layers can be combined with methylmethacrylate cement to form a composite 'sandwich'. This can be moulded to the contour of the chest wall defect as the cement sets. Gore-tex mesh is another permanent implant which provides less tissue ingrowth due to its limited porosity, but contours well and obtains a better water-tight seal. The rate of complications, such as infection and seroma, along with long-term outcome, are similar for the different synthetic meshes and use generally comes down to surgeon preference. If infected, these implants invariably need removal. Metallic support, in the form of transverse titanium plates, has been used (normally in combination with a synthetic mesh) for some anterior defects where additional rigidity is required (Figure 42.4).⁴⁹

Biomaterials – acellular dermis

The use of acellular dermis for chest wall reconstruction is relatively new. These dermal matrices can be either allograft (Alloderm) or xenograft (Strattice, Permacol) and have been denuded of their cellular components to prevent an antigenic response from the patient. Their structure allows cellular

**Figure 42.4** Composite Prolene and methylmethacrylate 'sandwich'.

infiltration and, more importantly, vascularization, which makes them more resistant to infection. A small series of acellular dermis in chest wall reconstruction has shown promising results.⁵⁰ These materials lack rigidity but they help to stabilize tissues and reduce paradoxical movement.

Autogenous

Following the development and increased use of prosthetic materials, autologous bone grafts are now seldom used. In situations where autologous bone is necessary it may be harvested as split rib grafts or taken from the iliac crest. However autogenous bone is limited in the size of defect it can reconstruct as there must be a large degree of cancellous bone overlap at the wound edge to enable the graft to survive.

Soft tissue coverage

Flap reconstruction

There are a variety of flaps that can be used for chest wall reconstruction, but familiarity with the following four flaps will enable the plastic surgeon to tackle the majority of chest wall defects: pectoralis major, omentum, rectus abdominis and latissimus dorsi.

Pectoralis major

Pectoralis major flaps are the workhorses for reconstruction of sternal wound dehiscence following median sternotomy. As a type V muscle,⁵¹ pectoralis major can be advanced and rotated on the thoracoacromial axis or raised on its secondary segmental pedicles, the perforators of the internal mammary artery (provided this vessel has not been harvested) and used as a 'turnover' flap. Combining one pectoralis turnover flap (Figure 42.5) with one pectoralis advancement flap (Figure 42.6) is an effective way of obliterating the dead space and providing adequate coverage of the inferior part of a sternal defect. Once the musculotendinous insertion is divided, the muscle is 'turned



Figure 42.5 Pectoralis major raised as a turnover flap based on IMA perforators. The turnover flap obliterates the dead space of the sternal defect, allowing the advancement flap to double breast over the top.

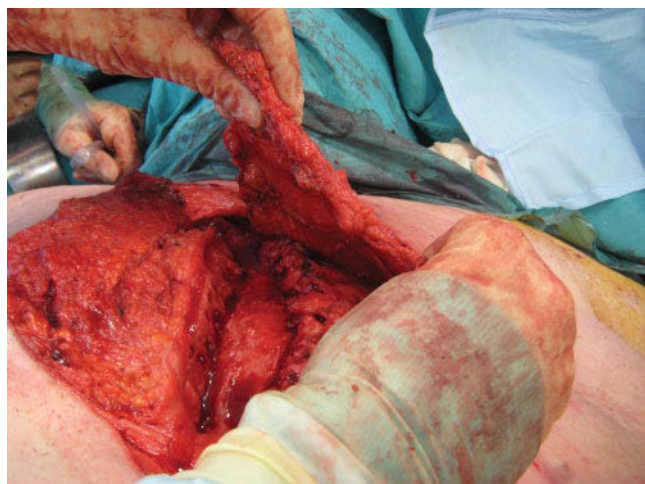


Figure 42.6 Pectoralis major raised as muscle only advancement flap based on thoracoacromial axis.

over' into the defect, and provides useful bulk that effectively fills the dead space and covers vulnerable tissues.

We would suggest that the post-sternotomy wound breakdown should be termed the 'IMA harvest sternal necrosis', as significant problems that require reconstruction are rare if the IMA has not been harvested. If the IMA is used to revascularize a coronary artery the pectoralis major muscle would not typically be raised on its medial blood supply. The left IMA is frequently used in bypass operations and, in these cases, the left pectoralis major is typically mobilized on the thoracoacromial axis. Cardiac revascularization with bilateral IMAs is rare and so it is usually possible to mobilize the right pectoralis major muscle on its mammary perforators.

The pectoralis major flap may also cover anterior and central defects following tumour resection. However, in cases of

osteoradionecrosis the flap has frequently been involved in the irradiated field.

Surgeons should be cautious of mobilizing the pectoralis major in a female patient with a large breast volume, particularly those patients who perfusion pressures are compromised by sepsis or organ failure. In extreme circumstances the combined removal of mammary perforators and the thoracoacromial axis supply will devascularize the fat of the breast and patients may experience considerable fat necrosis.

Omentum

Although not a muscle, the greater omentum can be classified as having a type III blood supply, with two codominant pedicles (left and right gastroepiploic arteries). Previous abdominal surgery or episodes of peritonitis may complicate or preclude its use.

Omentum is well suited to reconstruct large sternal defects; it is richly vascularized tissue and its bulk is effective at obliterating dead space. It also appears to be highly effective at encouraging the firm fibrous union that is important for the ventilatory rehabilitation of patients with large defects. The benefits and versatility of the omental flap make it particularly useful in reconstruction of osteoradionecrosis defects. The omentum provides volume, vascularity and, crucially, is derived from a non-irradiated area.

The greater omentum is accessed via an upper midline incision, although laparoscopic techniques have been described.⁵² The flap is usually raised on the larger right gastroepiploic artery and can be delivered into the sternal defect through the anterior tendinous portion of the diaphragm or through the wound and over the costal margin via a subcutaneous tunnel. Care must be taken with both of these approaches to ensure that the defect created is of adequate size so that the pedicle is not compressed, however it is also important to fashion the defect to minimize the risk of herniation of abdominal viscera. The concept of a tight defect compressing the pedicle is, in most cases, a theoretical one as the omental flaps tend to be very bulky and a generous defect is usually required to allow flap transposition. The greater risk is therefore of gastrointestinal herniation through a relatively large defect.

We would suggest the use of a technique that enables the surgeon to create a generous transdiaphragmatic window while minimizing the risk of herniation. Our preferred technique involves creating a secondary omental flap which can be wrapped around the pedicle and tacked to the edges of the diaphragmatic defect on the abdominal side (Figures 42.7 and 42.8). The flap acts as a plug that softly obliterates the defect and helps prevent the herniation of abdominal viscera into the chest.⁵³

Surgical tip: Transillumination helps identify the gastroepiploic vessels that lie close to the greater curvature of the stomach.

Rectus abdominis

A type III muscle, rectus abdominis is raised on its superior pedicle, the superior epigastric vessels (a continuation of the internal mammary vessels), when used in chest wall reconstruction.

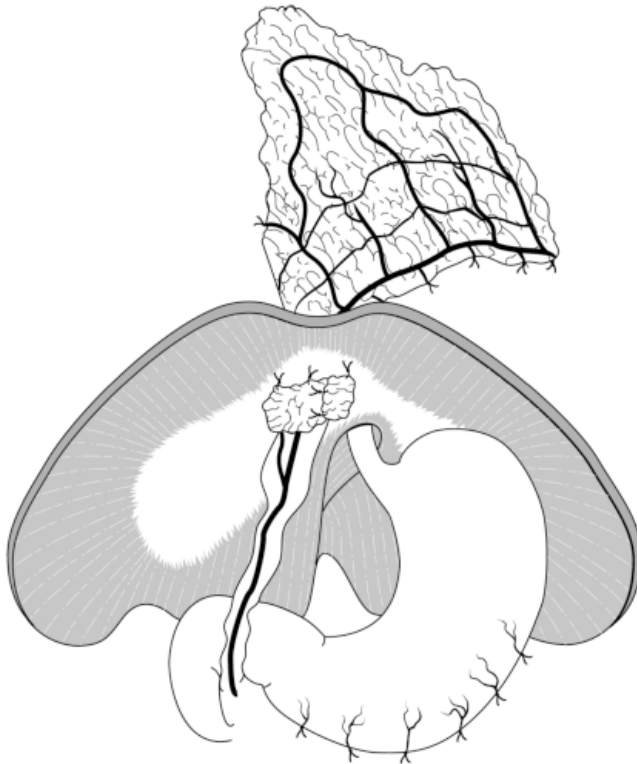


Figure 42.7 The 'omental wrap'. Source: Tansley *et al.*, 2006.⁵³ Reproduced with permission of Lippincott, Williams & Wilkins.



Figure 42.8 The omental flap raised with secondary flap isolated.

A myocutaneous or muscle-only flap, it is transposed into the defect with a pivot point at the costal margin and provides coverage for lower chest wall defects. As a myocutaneous flap the skin paddle can be orientated vertically (VRAM) or transversely (TRAM).

The rectus abdominis flap has been reported in cases where both IMAs have been harvested. Here the residual IMA is perfused through communication with intercostal vessels, however, clearly in these instances flap reliability is unpredictable.

Surgical tip: Preserving a length of the deep inferior epigastric artery and vein enables supercharging of the flap if rectus perfusion is inadequate.

Latissimus dorsi

The latissimus dorsi (LD) flap provides robust and reliable coverage for most anterolateral and lateral chest wall defects. It has a type V blood supply, the main pedicle being the thoracodorsal vessels, a branch of the subscapular system.

As with pectoralis, the humeral attachment of the LD can be divided to aid mobilization. The thoracodorsal vessels leave the subscapular axis and run anterior to the LD for about 5–6 cm before entering the underside of the muscle. The tendinous portion must be divided above this point to avoid injuring the pedicle.

The LD muscle can also be advanced or transposed postero-medially to cover central defects on the back. The LD can be raised on its secondary segmental lumbar perforators, and mobilized as a turnover flap.

Summary – flaps

This is by no means an exhaustive list and there are a number of other flaps that may be used for specific chest wall defects in specific situations (trapezius, external oblique, serratus anterior), although for the majority of cases one of the aforementioned flaps will be applicable. For very large defects a combination of flaps may be employed, or in some instances free tissue transfer may be required.

Adult presentation of congenital chest wall defects

Although congenital defects have been covered in the previous section, it is worth highlighting that patients with congenital chest wall deformities may present in adulthood. Concerns are generally aesthetic, as severe symptomatic cases have usually been addressed earlier. A detailed history to identify the patient's concerns, and careful examination of the chest wall are essential. The treatment modalities used to camouflage the defect include custom-made silicone prosthesis and autologous fat transfer.

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CHAPTER 43

Abdominal wall reconstruction

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Anatomy

The abdominal wall is a multilayered structure that wraps around and protects the abdominal viscera. In addition, it is involved in numerous other functions including trunk movement, respiratory effort (active expiration) and an adjunctive force in micturition and defecation. Anatomically, it is bound by the xiphoid process and costal margin superiorly and the iliac crests and pubis inferiorly.

The abdominal wall consists of skin, superficial fat, muscles, transversalis fascia and parietal peritoneum.

Skin, subcutaneous fat and fascia

The skin envelope of the abdomen is compliant and of intermediate thickness. Traditionally, two distinct layers of fascia are recognized – Camper's fascia and Scarpa's fascia. Camper's fascia represents the superficial layer, which contains variable amounts of fat. In males it is continuous with dartos fascia in the penis. Scarpa's fascia is deeper and more fibrous. It merges with the deep thigh fascia and superficial perineal fascia to contribute to fascia lata and Colles' fascia, respectively. In reality, it is more practical to think of three layers between the skin and abdominal musculature – superficial fat, membranous fascial layer (Scarpa's) and deep fat. The superficial fat layer is evenly distributed throughout the abdomen and the more vascular of the two. The deeper fat layer is of variable thickness, being thicker in the upper abdomen compared with the lower abdomen.¹

Muscles

The central abdominal wall is formed by the two paired rectus abdominis muscles and pyramidalis muscles inferiorly.

The paired rectus abdominis muscles are long, flat muscles involved in trunk flexion, forced expiration and in creating increased abdominal pressure such as in coughing. They originate from the pubic symphysis and pubic crest and insert into the xiphoid process and costal cartilages 5–7, interdigitating with the inferior fibres of pectoralis major. The two paired muscles are enclosed within the rectus sheath and separated from

each other by a midline band of connective tissue, the linea alba. The vertical muscle fibres are separated by three or four tendinous interconnections, which are horizontal bands of connective tissue. It is a type III muscle with a dual codominant blood supply (deep inferior and superior epigastric arteries). It is segmentally innervated from the 7th–11th intercostal nerves.

The pyramidalis muscles originate from the pubis and insert into the linea alba.

The lateral abdominal wall muscles include three sets of paired muscles – external oblique, internal oblique and transversus abdominis, which all contribute to the rectus sheath medially (Figure 43.1).

The external oblique is the most superficial and largest of the three lateral muscles. It originates from ribs 5–12 and runs in an inferomedial direction to insert into the linea semilunaris, where it contributes to the anterior rectus sheath, and iliac crest. The aponeurosis of external oblique forms the inguinal ligament inferiorly. It is innervated and nourished by the intercostal neurovascular bundles (5–12).

The internal oblique muscles originate from the thoracolumbar fascia and iliac crest and run in a superomedial direction, perpendicular to the external oblique fibres. It becomes aponeurotic medially, contributing to both anterior and posterior rectus sheath before inserting into the linea alba and ribs 10–12. It is innervated by the lower intercostal nerves and the first lumbar spinal nerve. There are multiple source vessels feeding the muscle including the deep circumflex iliac artery, deep inferior epigastric artery and lower intercostal vessels.

The transversus abdominis is the deepest of the three lateral muscles and runs transversely towards the midline from the iliac crest and thoracolumbar fascia. The transversalis fascia is a continuous fascial sheet that lies on the deep surface of transversus abdominis.

Blood supply

The blood supply of the abdominal wall is robust, with multiple independent sources and much collateralization. Huger considered the skin and subcutaneous components of the

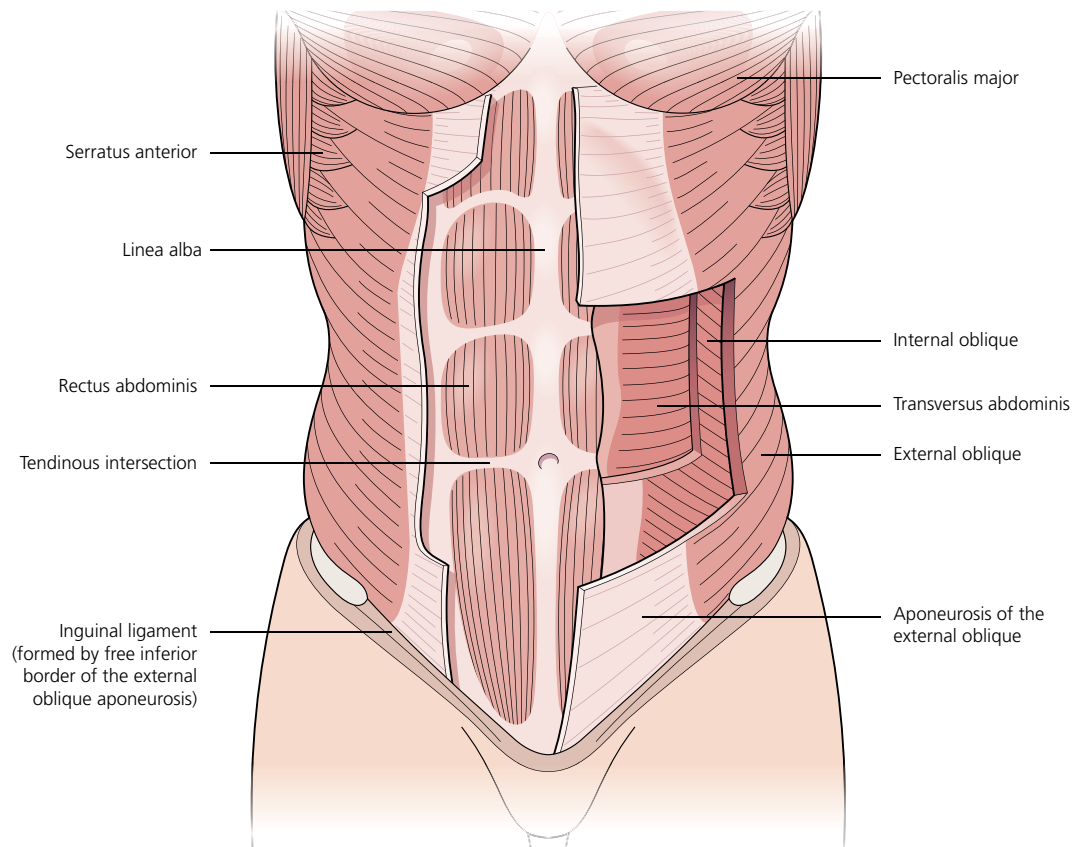


Figure 43.1 Anteroposterior view of abdominal wall muscles (lateral and central components). Source: <http://lyndseydesjardins.com/>

abdominal wall in terms of three zones of perfusion as illustrated in Figure 43.2.²

Zone I encompasses the central abdominal tissue overlying the paired rectus abdominis muscles. It is bound by the xyphoid process, pubic symphysis and lateral edges of the rectus sheath. The skin and subcutaneous tissue of zone I is perfused by musculocutaneous perforators from the deep inferior epigastric (branch of external iliac artery) and to a lesser extent the superior epigastric arteries (branch of internal mammary artery). The perforators are predominantly arranged in two rows – medial and lateral. The highest concentration of perforators is within 3 cm of the umbilicus. The medial row perforators perfuse across the midline to a greater extent than the lateral row perforators, a feature that is important in deep inferior epigastric perforators (DIEP) flap breast reconstruction.⁴

Zone II refers to the lower abdominal wall lying below the line of the anterior superior iliac spines. It is perfused by three different branches of the common femoral artery – superficial circumflex femoral artery, superficial inferior epigastric artery and the external pudendal artery. Zone III refers to the flank and abdominal wall lateral to the rectus muscles. It is supplied by the lower intercostal arteries and first lumbar artery – a

robust blood supply that is able to perfuse the entire abdominal wall after division of Huger's zone I and II as demonstrated after an abdominoplasty.

Innervation

The motor and sensory innervation of the anterior abdominal wall is derived from the anterior rami of the 7th–12th intercostals and first lumbar root. The intercostal neurovascular abdominal bundles pierce the transversus abdominis and travel in a plane deep to internal oblique. Preservation of the motor supply is essential for maintaining the integrity of the abdominal wall to prevent hernia formation or bulging.

Fascia

A detailed understanding of the fascia of the abdominal wall muscles is essential in abdominal wall surgery (Figure 43.3). The rectus sheath is formed laterally at the linea semilunaris by the condensation of the aponeuroses of external oblique, internal oblique and transversus abdominis. The anterior rectus abdominis is covered by sheath throughout its course. Posteriorly, the rectus is covered by sheath only above the arcuate line, a transverse line at the level of the anterior superior

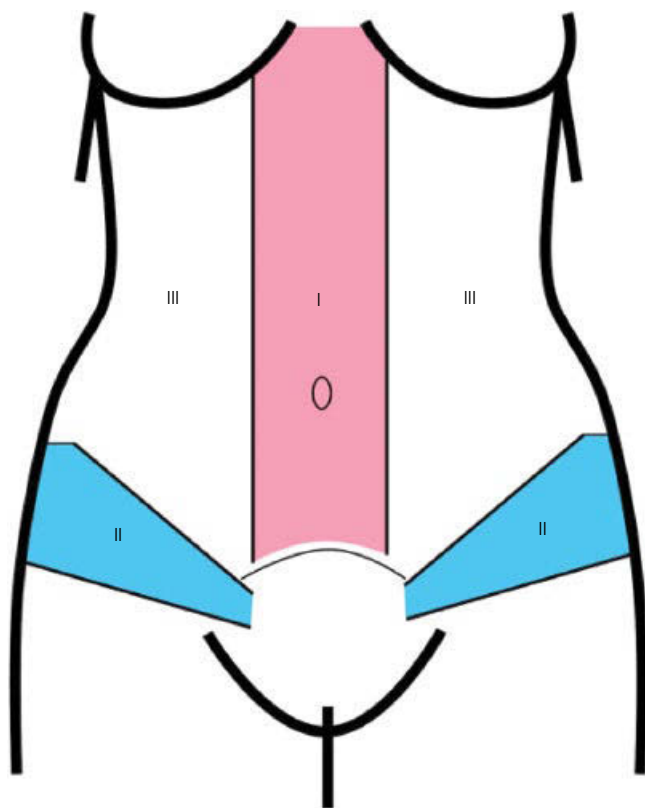


Figure 43.2 Huger's abdominal skin perfusion zones. Source: Karthikesalingam *et al.*, 2011.³ Reproduced with permission of Elsevier.

iliac spine. Below the arcuate line, only transversalis fascia and parietal peritoneum lie deep to the rectus.

Above the arcuate line, the anterior rectus sheath is composed of external oblique and the superficial leaf of the internal oblique fascia, whereas the posterior rectus sheath is formed by the condensation of deep leaf of internal oblique and transversus abdominis fascia. Below the arcuate line, the transversus abdominis fascia and deep leaf of the internal oblique fascia also contribute to the anterior rectus sheath, whereas the posterior rectus sheath is devoid of muscular aponeurosis. The rectus abdominis muscle is tightly adherent to the sheath at the tendinous intersections.

Aetiology and classification of abdominal wall hernia defects

Abdominal wall hernias can be congenital or acquired. Congenital causes include umbilical hernias, periumbilical hernias, Spigelian hernias (between rectus and linea semilunaris), lumbar hernias and epigastric hernias. Gastroschisis and omphalocele represent two severe types of congenital abdominal wall defects that are commonly associated with other

developmental anomalies. In gastroschisis, the abdominal wall defect is usually smaller and the abdominal wall contents freely protrude through the defect without being maintained in a sac of peritoneum, whereas in omphalocele, the defect is usually much larger and the abdominal contents are maintained in a sac of visceral peritoneum outside of the abdomen, protruding through the umbilicus. Both conditions occur infrequently (1 in 5000 births) but are often detected antenatally by ultrasound.⁵

Acquired hernias are usually iatrogenic, arising in a previous surgical incision. Other acquired causes of abdominal wall defects include trauma, infection (necrotizing fasciitis) and tumour. Common predisposing factors for incisional hernias include both systemic (poor intrinsic wound healing, obesity, age, malignancy) and local factors (inadequate surgical closure, infection, ischaemia, increased intra-abdominal pressure, prolonged ventilation). In terms of technique, evidence suggests that low dehiscence rates are best achieved by taking wide tissue bites (>1 cm from the fascia margin), short stitch intervals (1 cm) and non-strangulating tension on the suture. Even in the absence of identified risk factors, incisional hernias develop in 11% of primary laparotomies and may be as high as 33% in obese patients. Recurrent incisional hernias are especially challenging and the incidence of recurrent herniation after repair increases with each subsequent hernia repair operation.

Recently, the European Hernia Society has developed a classification system of abdominal hernias to deliver conformity and allow ease of comparison in published clinical studies.⁶ This classification system distinguishes between primary and incisional ventral hernias. For primary ventral hernias, the two parameters are location and size (hernia diameter) as illustrated in Table 43.1. The location is subdivided into midline defects (epigastric, umbilical) and lateral defects (Spigelian, lumbar). For incisional hernias, there are additional criteria including whether or not the hernia is recurrent.

Assessment of the abdominal wound

Clinical

An appropriately directed history and examination are an essential facet of the initial assessment. It is important to note whether the wound is acute or chronic and previous operations including use of mesh. Local and systemic risk factors for poor wound healing should be identified. The aim of the examination is to determine the wound characteristics and identify potential flap donor sites. On inspection, the presence of abdominal scars, fistulas, sinuses, exposed mesh and infection should be actively looked for. In addition, in long-standing wounds, there is a potential for malignant transformation. The wound is examined to determine which components are missing – skin, muscle, fascia. Adjunctive measures include an assessment of respiratory function as

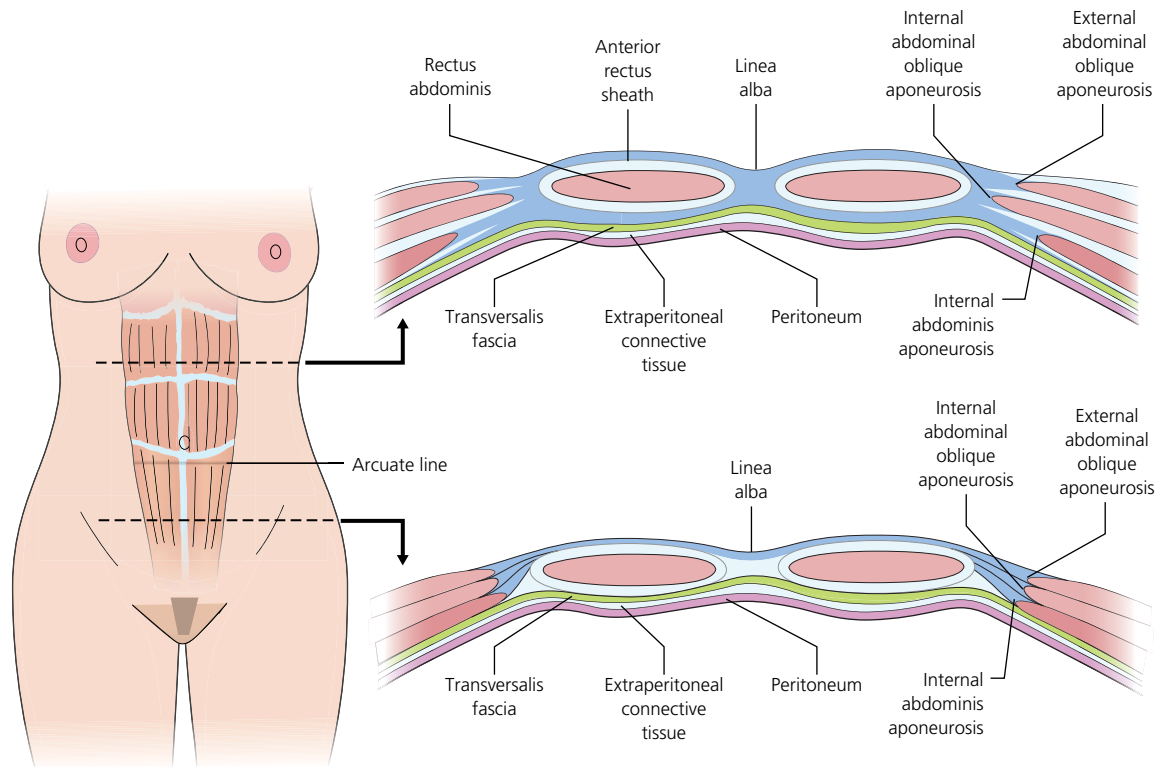


Figure 43.3 Formation of rectus sheath above and below arcuate line. Source: Anatomical considerations of body contouring, In Hunstad & Repta, *Atlas of Abdominoplasty*, Elsevier 2009. Reproduced with permission.

Table 43.1 European Hernia Society classification of primary ventral hernias

	Diameter (cm)	Small <2 cm	Medium 2–4 cm	Large ≥4 cm
Midline				
Epigastric				
Umbilical				
Lateral				
Spigelian				
Lumbar				

abdominal wall restoration can affect total lung capacity and respiratory effort.

Imaging

Preoperative imaging is very helpful in defining the pathology and in preoperative planning. Both abdominal CT and MRI can successfully identify the extent of the abdominal wall defect and the integrity of adjacent musculofascial structures. However, CT angiography (i.e. with intravenous contrast) is superior at delineating the vascular anatomy, especially when planning pedicled flaps or free tissue transfer (Figure 43.4). An abdominal and pelvic CT scan with oral contrast enables detailed understanding of the wound and intra-abdominal contents.



Figure 43.4 CT image demonstrating a musculocutaneous perforator (blue arrow) traversing through the rectus abdominis muscle from the right deep inferior epigastric artery (red arrow) at the level of the umbilicus.

Goals of reconstruction

The goal of abdominal wall reconstruction is twofold. The first goal is to restore function and integrity of the musculofascial abdominal wall to prevent visceral eventration and provide dynamic muscle support. The second is to provide skin resurfacing.

Management of abdominal wounds

Acute wounds

Acute abdominal wounds may be the result of dehiscence post laparotomy, infection, trauma or neoplasms.

Most acute abdominal wounds following laparotomy can be managed with local wound care, including bedside debridement of necrotic tissue and daily dressing changes or application of negative pressure wound therapy (NPWT). In select cases, secondary closure following wound excision may be feasible and has been shown to be superior to healing by secondary intention.⁷

Fascial dehiscence post laparotomy may occur in the acute setting without a skin defect. The typical history involves persistent drainage of serous fluid through the wound after one week. Fluid cultures are often positive. Fascial reclosure has a high chance of failure. Ideally, tension-free fascial closure is achieved and this may require use of a biologic or synthetic mesh with skin closure or NPWT.

In the trauma setting, the decision must be made between performing an immediate and a staged repair. Immediate reconstruction is preferred as it is more cost-effective and less time-consuming. However, reconstructive options are limited in unstable patients, contaminated wounds or where further procedures are planned. In these circumstances, the patient may be better served by a staged repair involving initial closure by mesh repair and skin grafting once sufficient granulation tissue has developed. The second stage involves excision of the skin graft and definitive repair of the associated ventral hernia.

There is an increasing vogue for leaving the abdomen open in severe abdominal trauma. This so-called open abdomen technique prevents abdominal compartment syndrome and allows early identification of complications such as bowel ischaemia. Managing patients with an open abdomen is also appropriate where primary fascial closure is not feasible such as from oedematous bowel in a patient with peritonitis. A recent systematic review concluded that NPWT is more successful than other methods, including a staged repair, in achieving fascial closure and reducing mortality in the open abdomen.⁸

Chronic wounds

Chronic uncomplicated wounds without fistula can be managed in a number of ways. Small wounds may be managed with simple dressings and closed directly at the time of hernia repair. Moderate to large wounds are probably best skin grafted initially to allow a temporizing wound closure. The skin graft can be excised at the time of definitive hernia repair. In cases where there is exposed mesh, the mesh should be removed if grossly contaminated. However, exposed mesh can be readily salvaged with application of NPWT and subsequent split-thickness skin grafting once sufficient granulation tissue has developed.

Small sinuses with retained foreign material but no fistula (e.g. stitch abscess) should be managed by removal of the foreign body. Healing by secondary intention usually follows.

The presence of an enterocutaneous fistula, defined as an epithelial-lined connection between the skin and internal organ, poses an immense clinical challenge. Treatment must be individualized and despite involvement of experienced multidisciplinary teams, outcomes are often poor with a high morbidity, mortality and psychosocial dysfunction.⁹

Prevention is key. Enterocutaneous fistulas most commonly develop in patients who have suffered a postoperative anastomotic leak. In wounds with exposed abdominal viscera, wet-to-dry dressings should be avoided as they can cause mechanical debridement of friable bowel mucosa and subsequent fistula development. Instead, the bowel should be covered with omentum where possible or absorbable mesh if omentum is unavailable.

Once a fistula is established, initial therapy should be directed at systemic measures to facilitate wound healing and maintain bowel function. These include aggressive treatment of sepsis, nutritional supplementation and attention to fluid and electrolyte disturbances. Patients have high caloric and metabolic needs. In addition, vitamins and trace elements such as copper and zinc can be rapidly depleted through fistula-related losses. Total parenteral nutrition (TPN) can reduce enteric losses by as much as 50% although enteral may also be used to provide trickle feeds.¹⁰ Reduction of fistula output may increase the chance of spontaneous closure.

Once systemic factors have been addressed adequately, a surgical closure should be carefully planned. Imaging is critical to determine the anatomy of the fistula. This may include CT, MRI, fistulograms and small bowel imaging. In the early stages, the wound may be skin grafted to convert the fistula into an ostomy, which can be managed more easily with a pouch system. Other techniques have been used to divert the fistula effluent from the wound bed in the early stages, such as NPWT and bridging dressings, which isolate the fistula. In either case, the periwound skin should be adequately protected with topical barrier ointments (containing zinc oxide, petrolatum, dimethicone or a combination) to prevent chemical denudation. A pouch system is used to collect the effluent and in addition may suppress odour.

The fistula can then be allowed to close spontaneously or be closed secondarily. Secondary fistula closure is best achieved by bowel resection and re-anastomosis with abdominal wall reconstruction using acellular dermal matrices or components separation.

Management of fascial defects

Mesh repair

Use of mesh has been conclusively demonstrated to reduce recurrence of both primary and incisional hernias.¹¹ Surgical mesh must provide sufficient biomechanical strength to meet

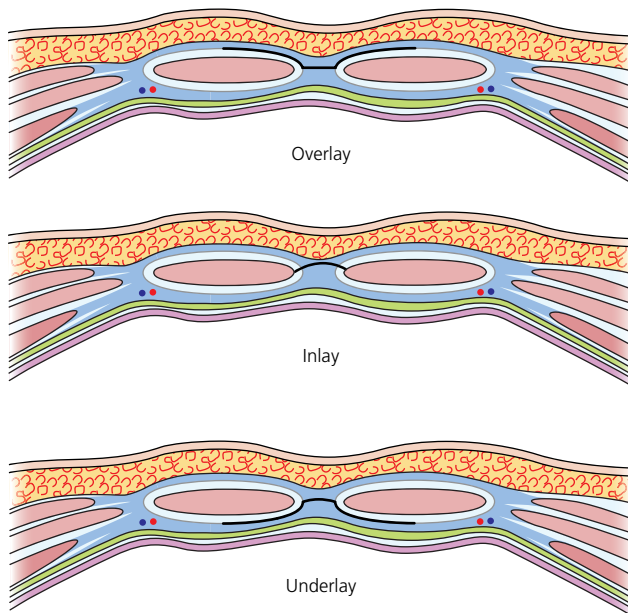


Figure 43.5 Cross-sectional image of abdominal wall above arcuate line demonstrating different positions of mesh placement – overlay, inlay, underlay.

physiological requirements in order to permanently protect the fascial defect. The tension on the abdominal wall and therefore the required tensile strength of the fascia closure is a function of the intra-abdominal pressure, which ranges from 1.55 mmHg in patients lying supine up to 150 mmHg maximum peak pressure in coughing patients.^{12,13}

There are many varieties of synthetic meshes, which are broadly similar in structure to suture materials. They can be classified according to absorbability, tensile strength, porosity and filament structure. Traditionally, non-absorbable meshes such as Prolene (polypropylene) have been favoured in view of their strength, superior handling and stability. However, concerns about mesh extrusion, pain, bowel adhesions and development of fistulas have led to the development of new absorbable meshes. The current trend is for lightweight composite meshes that have an absorbable component such as Vypro II and Ultrapro (Johnson & Johnson, India). They consist of thin filaments of Vicryl and Prolene (Johnson & Johnson, India) or Monocryl (Johnson & Johnson, India) and Prolene (Johnson & Johnson, India). Such composite meshes provide sufficient tensile to withstand high intra-abdominal pressures but reduce complications such as bowel adhesions. The results of long-term randomized controlled trials comparing non-absorbable heavyweight meshes with newer composite meshes are awaited to determine if there is a significant difference in outcome.

The general principles of a successful mesh repair technique involve wide coverage with adequate overlap and secure suture fixation to the fascia. The mesh may be inserted above the fascia

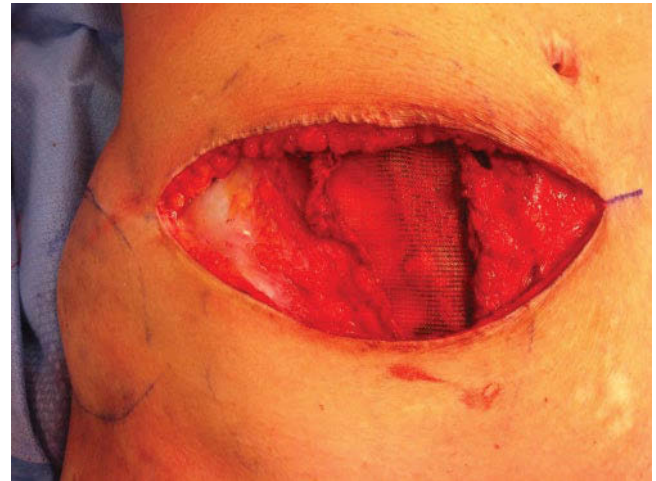


Figure 43.6 Illustration of inlay mesh technique for repair of DIEP donor site hernia.

(onlay or overlay), underneath the fascia (inlay) or deep to the rectus (underlay, Figure 43.5).

Mesh can be used to reinforce a fascial repair or as an interpositional strut where a fascial defect exists. Figure 43.6 demonstrates an inlay mesh placement of a donor site hernia post DIEP flap breast reconstruction.

Both inlay and onlay mesh repair techniques are popular methods of bridging fascial defects and seem to be equally effective.¹⁴ However, with inlay mesh placement, the mesh is in direct contact with the abdominal viscera over a greater surface area than onlay placement, increasing the theoretical risk of erosion into the bowel and fistula formation. The key component is ensuring there is sufficient overlap between the margins of the fascia and the mesh.

Acellular dermal matrices

Certain fascial defects are not suitable for mesh repair, particularly in cases complicated by gross contamination, scarring, radiotherapy and absence of local tissues for reconstruction. These challenging cases have provided the impetus for development of acellular dermal matrices (ADM), which have many theoretical advantages over synthetic meshes, mostly due to their enhanced biocompatibility. It is suggested that these biologic matrices have an increased capacity for integration with surrounding tissues, while demonstrating resistance to infection, extrusion, erosion and adhesion formation.¹⁵ They are particularly advantageous in abdominal wall reconstruction, as they can be placed directly on exposed viscera and applied in contaminated fields.

Dermal matrices are classified according to the species of origin (allogenic – human, xenogenic – porcine, bovine) and the source of the collagen matrix (dermis, intestinal submucosa, pericardium). Similar to mesh techniques, they can be applied

as onlay or inlay sheets, or both as in the sandwich technique. Where fascial defects exist, they can be used as interpositional bridging grafts.

In terms of efficacy, the reported success of ADMs in reducing hernia recurrence is mixed. The incidence varies widely among published series from 0 to 80%.¹⁵ In select groups such as transplant patients, human dermal matrices have been shown to reduce hernia recurrence compared with synthetic mesh and have a lower rate of infection.¹⁶ However, other authors have reported high recurrence rates, especially when dermal matrices have been used as interpositional bridging grafts.¹⁷ There is no consensus on whether there is any difference in effectiveness between placement of the matrix in an inlay or onlay fashion. An important consideration is the cost of the dermal matrices, which has been shown to be as high as US\$5100 per patient in patients undergoing repair of abdominal wall defects.¹⁸

In experimental models, porcine-derived matrices benefit from increased tensile strength compared with human matrices but have poorer neovascularization, which may affect long-term effectiveness. In clinical practice, human dermal matrices such as Alloderm (Life Cell Corp) are more commonly used than porcine (e.g. Permacol, Covidien, Boulder, CO, USA) or bovine-derived matrices. There is an absence of clinical trials comparing human with animal-derived products.

Complications common to all ADMs include hernia recurrence, infection, seroma and skin necrosis. Although relatively common, infection rarely leads to matrix extrusion and can often be managed with targeted antibiotics. Indeed, one of the purported benefits of dermal matrices over synthetic meshes is resistance to infection postulated to be a result of early capillary ingrowth and effective delivery of antibiotics and innate host defences. Even when implanted in a contaminated field, infection has been demonstrated to be as low as 16%.¹⁹ Seromas are common but rarely affect outcome. Strategies to reduce seroma formation include avoiding wide undermining of abdominal skin over fascia, preservation of suprafascial fat, use of quilting sutures and abdominal binders.²⁰

Components separation

Ramirez and colleagues described the technique of components separation to reconstruct midline defects, although the use of external oblique fascial relaxing incisions had previously been described for epigastric hernia repairs.^{21,22} The technique involves medial advancement of innervated musculofascial flaps involving a complex of rectus abdominis, internal oblique and transversus abdominis. Preoperative CT imaging is critical in determining the presence or absence of the rectus muscle complex, particularly in massive midline defects where the rectus muscles have been displaced very laterally.

The procedure is begun by elevating the skin flaps off the underlying abdominal musculature. This can be continued to the anterior axillary line as required. Subsequently, the linea semilunaris is identified (condensation of internal and external oblique fascia into rectus sheath). A vertical incision is made

through the external oblique fascia 2 cm lateral and parallel to the linea semilunaris. It should be just medial to the myofascial junction of the external oblique muscle itself. This is continued from the level of the costal margin superiorly to the arcuate line inferiorly. The plane between external oblique and internal oblique is developed, being careful to avoid damage to the internal oblique fascia or segmental intercostal neurovascular bundles supplying rectus. The dissection is continued lateral to the level of the midaxillary line. At this point, the mobility of the rectus abdominis–internal oblique–transversus musculofascial complex is assessed. If further advancement is required, the dissection can be continued to the posterior axillary line.

Each hemicomplex can deliver 4 cm of advancement in the upper abdomen, 8 cm in the mid-abdomen and about 3 cm in the lower abdomen (Figure 43.7). Release of the posterior rectus sheath from the undersurface of the muscle can increase medial translocation further by 2 cm at each point, making the total movement 6 cm, 10 cm and 5 cm, respectively. Therefore, components separation can reconstruct defects of 20 cm or more in the midline. The fascia is closed and the skin is advanced to the midline and closed in a layered fashion over suction drains.

Components separation has allowed the reconstruction of large midline and paramedian abdominal wall defects with well-vascularized innervated tissue and acceptable donor site morbidity compared with other autologous techniques. In addition, it frequently obviates the need for mesh and its associated complications such as infection, extrusion and enterocutaneous fistula development.

The hernia recurrence rates in published series ranges from 0% to 32%, suggesting that it can be a very effective technique.²⁴ Recurrence rates are reduced further by additional use of onlay synthetic meshes. In most cases, the strength of the abdominal wall is increased but does not return to premorbid levels. Although commonly used in adults, the components separation technique has also been applied successfully to closure of congenital abdominal wall defects in neonates, including gastroschisis and omphaloceles.²⁵

One of the potential pitfalls of the components separation technique is ischaemia of the medial skin edges and ensuing necrosis, infection or dehiscence. Compromise of the medial margin occurs due to the extensive undermining of the skin flaps required to allow sufficient exposure for division of the external oblique muscles. Modifications to the original procedure that reduce midline wound-healing complications include endoscopic-assisted components separation and techniques that preserve periumbilical perforators.^{26–28}

Soft tissue reconstruction in abdominal wall reconstruction

Rohrich and colleagues developed an algorithm for planning closure of abdominal wall defects.²⁹ They consider closure of the skin and musculofascial components independently and have a separate

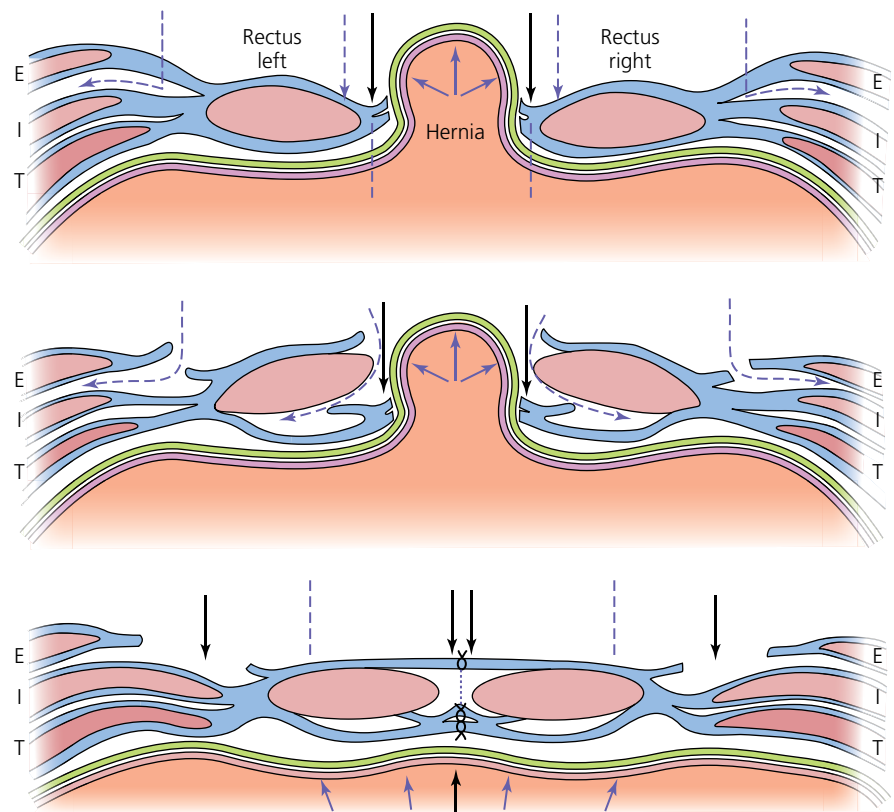


Figure 43.7 Cross-sectional view illustrating components separation technique. E, external oblique muscle; I, internal oblique muscle; T, transversus abdominis muscle. Source: Dragu *et al.*, 2009.²³ Reproduced with permission of Springer.

scheme for partial and complete defects. Although this algorithm is helpful in providing some structure for framing decisions, in reality it is too complex to be a readily useful tool. Treatment needs to be individualized and the clinician should be aware of all options. In addition to closure of fascial defects by synthetic mesh, ADMs and components separation as discussed in the previous section, there is also a place for pedicled or free flaps as discussed below. There are a variety of means of achieving skin closure.

Direct closure

Due to the laxity of the abdominal wall skin envelope, direct closure is often feasible. If required, wide undermining in the prefascial plane can be performed to deliver additional advancement of the skin flap edges such as in an abdominoplasty or components separation.

Skin grafts

Split-thickness skin grafts are often used as a temporizing measure to close a wound that is not fit for immediate reconstruction. The graft can then be excised at the time of definitive closure.

Negative pressure wound therapy

NPWT has revolutionized the management of wounds over the past decade. In abdominal wall reconstruction, it has been used to accelerate healing by secondary intention and wound

preparation prior to reconstruction with grafts. It is frequently used as a bridge between initial wound care and final-stage, definitive wound closure.

In acute abdominal wall defects, NPWT is commonly used over a mesh repair to allow development of a sufficient bed of granulation tissue to accept a split-thickness skin graft. After several months when the scar has matured and the number and density of adhesions has rescinded, definitive abdominal wall reconstruction can be performed via components separation.³⁰ As discussed previously, NPWT also has a place in management of the open abdomen.

Flap reconstruction

Soft tissue flaps can be used to reconstruct both skin and fascial components of abdominal wall defects, delivering robust, well-vascularized tissue to aid primary wound healing and maintain abdominal wall integrity. Flaps are often used to repair fascial defects in contaminated wounds where synthetic mesh is best avoided. Another common indication for flaps is to provide soft tissue cover of mesh or ADMs in clean wounds following tumour extirpation. Flaps provide an alternative autologous reconstruction in situations where components separation is not feasible.

The *pedicled rectus abdominis* flap is the workhorse flap in abdominal wall reconstruction (Figure 43.8). It can be taken



Figure 43.8 Photograph showing vertical rectus abdominis muscle reconstruction of a full-thickness right upper quadrant abdominal wall defect following excision of a liposarcoma in a 59-year-old man.

with or without a skin paddle and a variety of orientations of the skin paddle are possible, providing flexibility in design. Based superiorly, the flap is perfused by the superior epigastric artery or inferiorly on the deep inferior epigastric system.

The *anterolateral thigh* (ALT) flap can be used to reconstruct large composite abdominal wall defects, providing both fascial and skin components. A very large area of fascia lata can be harvested from the thigh with the flap to reinforce or reconstruct large fascial defects. As a pedicled flap, it can easily reach above the umbilicus. Once elevated on the lateral circumflex femoral artery, the flap is passed deep to the rectus femoris and sartorius muscles to be delivered to the abdomen. The fascia lata is sutured to the fascial margins of the defect and the skin is closed in a layered fashion over suction drains. In upper abdominal wall defects and in cases where the flap does not reach the defect adequately, a pedicled ALT can be converted into a free ALT with similar outcomes.³¹ Where a free ALT is performed, the deep inferior epigastric vessels are first-choice recipient vessels due to their accessibility and size. If unavailable, the gastroepiploic vessels provide a reliable alternative.

The *tensor fascia latae* (TFL) flap can also be used to reconstruct composite defects of the lower abdominal wall but is inferior to the ALT flap, which has a larger, more reliable skin territory and longer vascular pedicle. Figure 43.9 demonstrates an example of

a TFL flap. In particular, tip necrosis is common in both free and pedicled TFL flaps, being reported to occur in up to 43% of flaps used in abdominal wall reconstruction.³²

The pedicled *rectus femoris* muscle has also been described for use in reconstruction of lower abdominal wall defects. In contrast to TFL flaps, it provides considerable muscle bulk, which is useful for dead-space filling. It has the same pedicle as the ALT flap (descending branch of the lateral circumflex artery) although the pedicle length is shorter than the ALT flap as the rectus branch typically comes off higher up than the ALT perforators.

Other flaps used in abdominal wall reconstruction include vastus lateralis, gracilis, internal oblique, external oblique and, in cases where large skin defects exist, free latissimus dorsi flaps.

Abdominal wall transplantation

Abdominal wall transplantation is an evolving technique used to facilitate abdominal wall closure in patients undergoing intestinal or multivisceral transplantation.³³ In this group of patients, abdominal closure can be very difficult due to loss of domain and swelling after organ transplantation. In addition, they have a high mortality and morbidity. The abdominal wall graft is a composite tissue allograft composed of skin, subcutaneous tissue and rectus myofascial complex. The flap is vascularized through anastomosis of donor and recipient inferior epigastric vessels or from an iliac vessel anastomosis in continuity with the femoral vessels. The muscle is not innervated and although muscle hypotrophy develops, significant hernia formation does not seem to be a problem.³⁴

Management of the postoperative bulge

Abdominal wall complications after primary abdominal surgery or flap harvest can manifest as either an abdominal wall hernia or myofascial laxity (bulge). It can be difficult to distinguish clinically between a hernia and a bulge. A bulge is a contour abnormality of the abdominal wall with underlying myofascial continuity that is the result of global weakness. In contrast, a hernia is associated with a discrete myofascial defect. Bowel entrapment and obstruction are therefore a less common sequelae of bulges compared with hernias.

Predisposing factors to development of bulges include muscle denervation (abdominal flap harvest), inadequate mesh tension, native myofascial laxity and increased intra-abdominal pressure. In a recent systematic review, the risk of bulging associated with DIEP flap harvest was one third of muscle-sparing transverse rectus abdominis myocutaneous (TRAM) flaps.³⁵ Most frequently, a bulge presents as a contour abnormality that changes with posture and activity and can be associated with a dull ache. In severe cases, it can significantly interfere with daily activities and cause progressive skin attenuation with

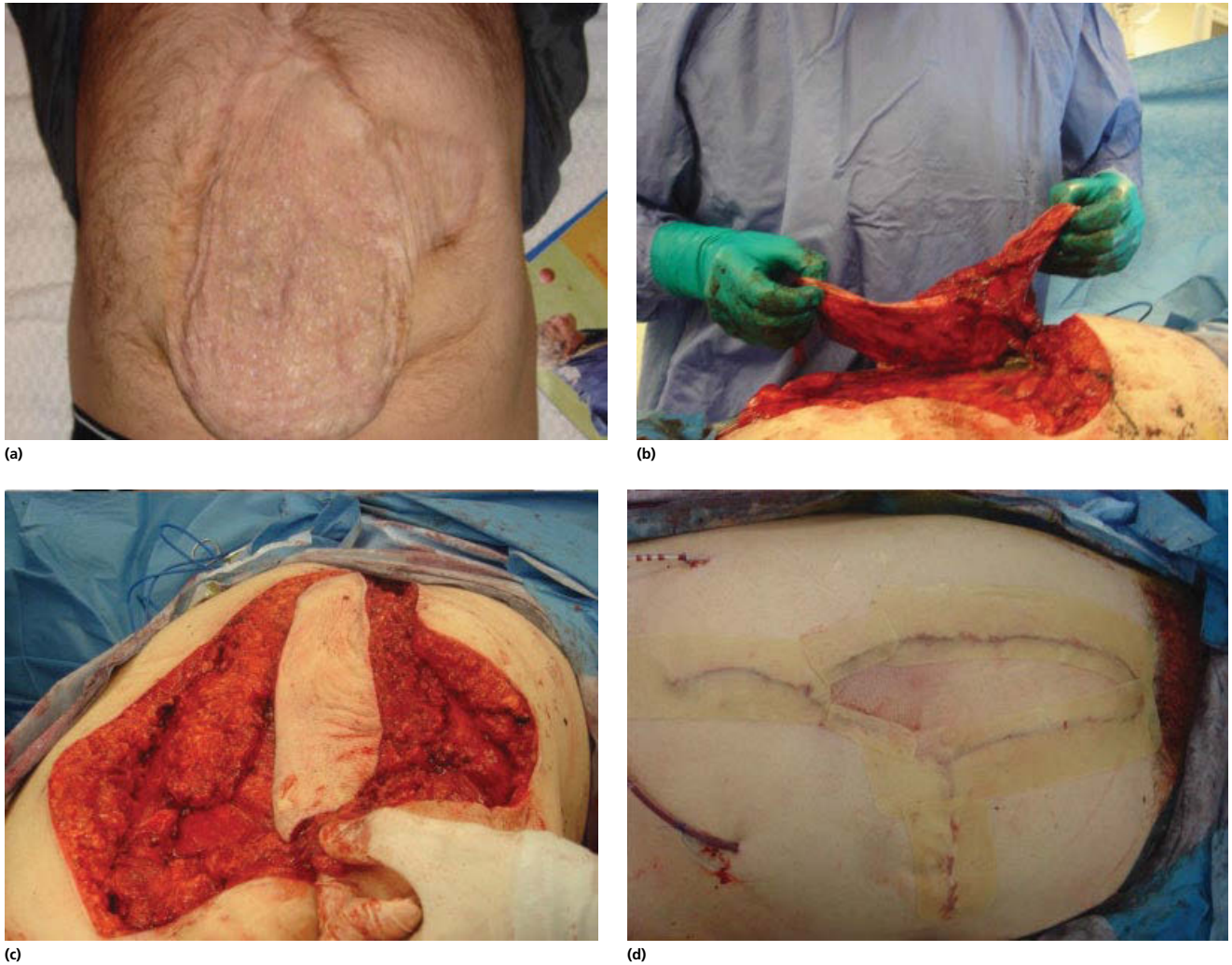


Figure 43.9 Elderly man with ventral hernia following previous laparostomy wound treated definitively by extended tensor fascia latae (TFL) flap composite reconstruction. (a) Skin graft on laparostomy wound. (b) TFL flap raised with fascial extension. (c, d) Flap inset. Source: Professor Wayne Morrison and Mr Ross Farhadieh. Reproduced with permission.

ulceration and infection. Diagnosis can be made by clinical examination and imaging, including contrast-enhanced CT scan and dynamic ultrasound assessment, which looks at the effect of posture and intra-abdominal pressure on abdominal wall integrity.

Indications for operative repair include massive bulges with loss of function due to truncal instability, progressive skin changes or significant aesthetic concerns. Pain relief is not a guaranteed outcome following repair. The principles of surgical correction include: (a) resect or plicate the area of myofascial laxity, (b) reapproximate intact dynamic fascial tissue under physiologic tension, and (c) reinforce the repair with adjunctive procedures such as mesh or components separation.³⁶ It is important to appreciate that bulges are associated with a global central or lateral fascial attenuation, often due to muscle denervation. Therefore, simple patching of the defect often results in recurrent bulges in adjacent

areas. This is best achieved by use of inlay mesh across the entire central or lateral bulge with fixation of mesh to stable points beyond the bordering oblique muscle complexes, ideally to innervated myofascia or bone. Postoperatively, patients can ambulate on day 1 but should avoid heavy physical activity and sports for six weeks.

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CHAPTER 44

Genitourinary and perineal reconstruction

PERINEAL RECONSTRUCTION

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Introduction

The perineal region has two important organs – an excretory and a sexual. Malfunction or dysfunction of either of these can affect the whole body. The perineum has very rich blood supply, fed by both the femoral and the iliac arteries; branches from these vessels anastomose with each other around the urogenital and anal orifices and this enables reconstructing surgeons to use skin flaps based on the perforators from these vessels.

The soft tissue reconstruction is challenging in terms of achieving the best outcome with respect to maintaining the normal function and cosmetic appearance. Local flaps based on perforators are the best as they replace like tissues and should be considered for small to moderate defect. However, the management of advanced pelvic malignancy requires a multidisciplinary approach. The potential method of resection and reconstruction for total perineal defects should be discussed by a multidisciplinary team, which should consist of a colorectal surgeon, urologist, gynaecologist, oncologist, radiologist, plastic surgeon, specialist nurse practitioner, physiotherapist and psychologist. Factors that dictate the choice of method are the patient's general condition and habits (age, sex, diabetes, atherosclerosis, obesity, smoking and nutritional state) and the defect created (skin, pelvic viscera, pelvic muscles). Some of the patients will have already undergone surgery and radiotherapy. In these patients, the local option may have either already been used or is not possible because of radiotherapy. The patient should be fully informed of the surgical treatment, the outcome and possible major life adjustment postoperatively (e.g. colostomy and urinary conduit). An anaesthetist who is regularly involved in this type of procedure should assess the patient.

Anatomy of the blood supply of the perineum

In 1889 Carl Manchot¹ divided the cutaneous area of the perineum into anterior and posterior regions. He also described the blood supply of the perineum in great detail. Branches of femoral and internal iliac arteries form the rich vascular network of the perineum (Figure 44.1). The anterior region is supplied by the superficial external pudendal artery and deep external pudendal artery. The obturator artery, internal pudendal artery and branches of the inferior gluteal artery supply the posterior half of the perineum.

The internal pudendal artery gives out two terminal branches: the penile/clitoral branch and the perineal artery. The perineal artery supplies the labial/scrotal part of the perineum via its terminal branches, which anastomose with their contralateral counterpart. The perineal artery also gives off medial and the lateral branches. The medial branch supplies the perianal region and the lateral branch supplies an area of posterior surface of the upper thigh. All these branches anastomose with their counterpart from the opposite side and hence form a rich circle of vascular anastomoses around the orifice. This vascular network provides the basis of the perforator flaps.

Reconstruction of partial or moderate defect of the perineum

The ideal flap for any perineal defect should use reliable and robust like tissue, not be too bulky and should have protective sensation to maintain normal function and appearance. There should also be minimal donor site morbidity. For practical purposes the perineum can be divided into right, left and central area. A line drawn between the ischial tuberosities divides these areas into anterior and posterior halves (Figure 44.2). The central area contains the urogenital orifice in the anterior half and the anal orifice in the posterior half. In the majority of the cases the defect to be reconstructed is created in the central area, for example due to vulvar/vaginal or anal neoplasia. Other situations where flap reconstruction is

used are congenital malformations and severe debilitating infections (such as hidradenitis suppurativa). Traumatic defects requiring local flaps are uncommon. For defects in the upper quadrant, donor skin flaps could come from the groin and/or mons pubis area, whereas for the lower quadrant the donor site may be from the gluteal fold and/or gluteal area. The mid thigh can also provide a flap for these situations. For the central area of the perineum the donor could be selected from any of the above sites. The selection depends on the site and size of the defect.

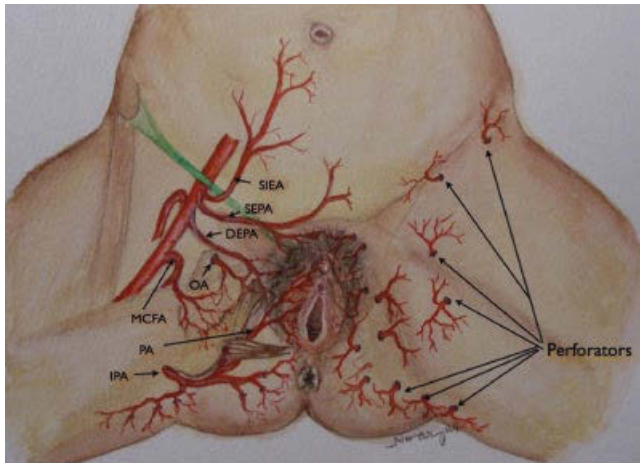


Figure 44.1 Blood supply of the perineum. SIEA, superficial inferior epigastric artery; SEPA, superficial external pudendal artery; DEPA, deep external pudendal artery; OA, obturator artery; MCFA, medial circumflex femoral artery; PA, perineal artery; IPA, internal pudendal artery.

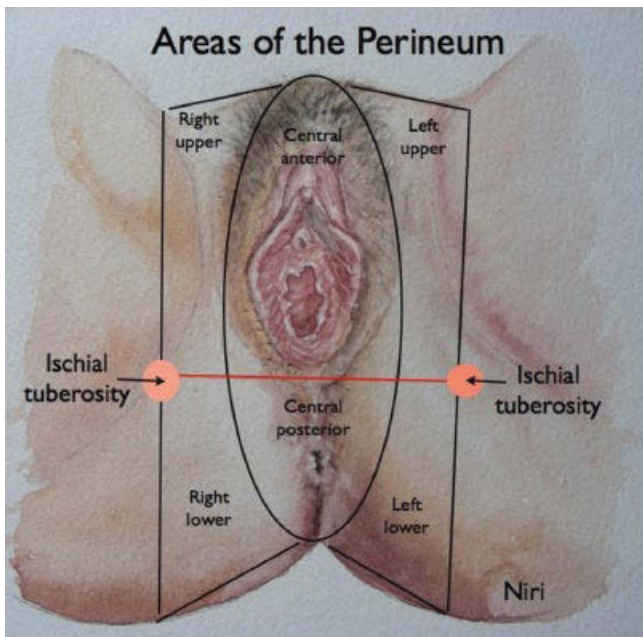


Figure 44.2 Schematic division of the perineum.

There are three ways of moving local perforator flaps, namely rotation (lotus petal flaps (Figure 44.3), V-Y advancement flaps (Figure 44.4) and transposition (pudendal, mons pubis flaps) (Figure 44.5).

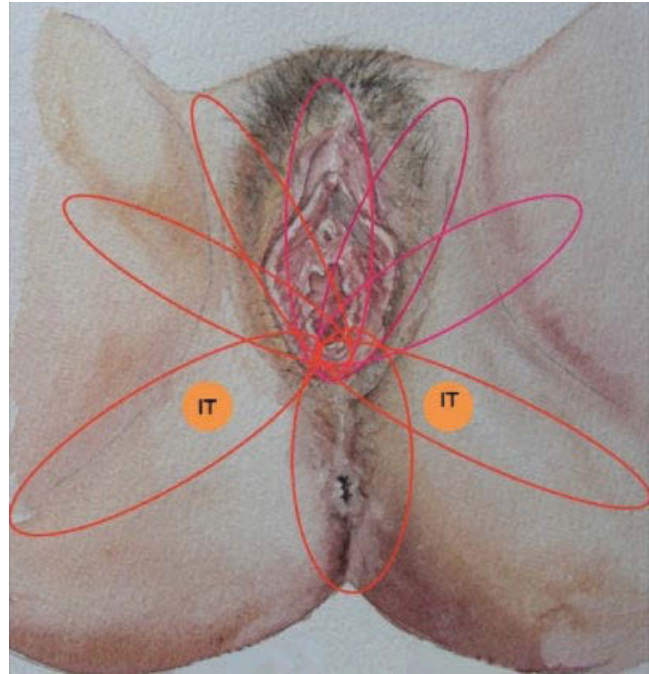


Figure 44.3 Lotus flap schema based on local perforators.

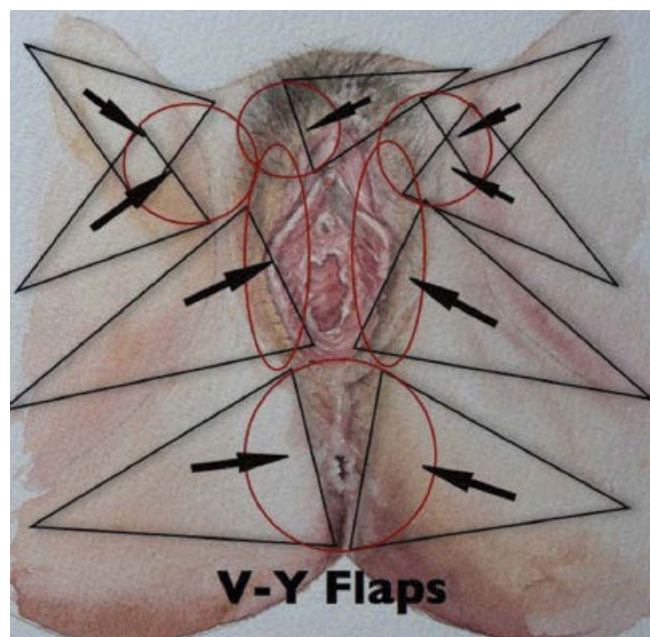


Figure 44.4 V-Y advancement flap.

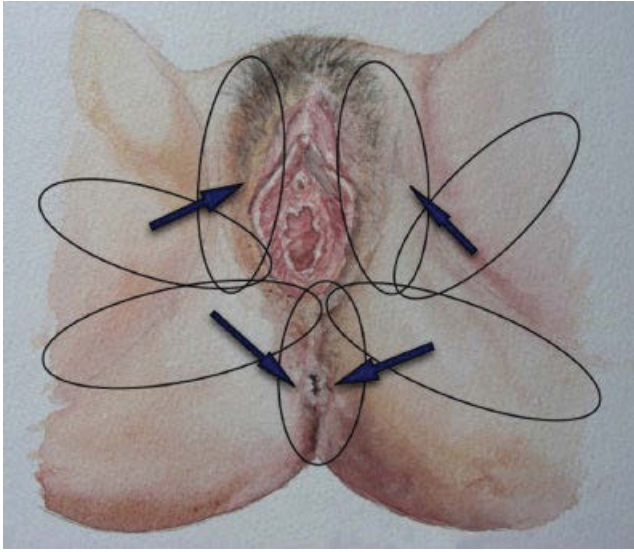


Figure 44.5 Transposition of pudendal and mons pubis flaps.

In 1973, Horton *et al.*² reported local random pattern flaps designed around the genital area. Other types of skin flaps used during the 1970s and 1980s were mostly myocutaneous flaps. Among these are the gracilis,³ tensor fascia lata^{4,5} and rectus-abdominus.⁶ These flaps were obviously bulky and accompanied by considerable donor site morbidity. The fasciocutaneous flap emerged in the early 1980s and in 1987, Wang *et al.*⁷ advocated medial thigh fasciocutaneous flaps, whereas Wee and Joseph in 1989⁸ described a pudendal thigh flap with an intact nerve supply for vaginal reconstruction. Woods *et al.* modified the same flap in 1992.⁹ The lotus petal flaps^{10–12} are perforator based and have become a standard method of reconstruction.

Operative technique

Surgery may be performed under either general or regional anaesthesia. The patient is placed in the Lloyd-Davies position. A urinary catheter is inserted. Prophylactic antibiotics should be given at induction and for 48 h post operatively. The defect is created during the resection for either cancer or infection or the debridement of traumatized tissue. Once the dimension of the defect is studied and assessed, the required flap is planned over the donor area. The flap is planned in a reverse manner by using a swab or a pattern of the defect turned towards the selected donor site in reverse.

The success of perforator flap reconstruction depends on the selection of the perforator vessel at or around the base of the selected flap. Before the operation, perforator vessels are mapped using a hand-held Doppler ultrasound probe at the base of the skin flap. An exploring incision is made along one edge of this flap down to the muscle. The flap is elevated with the deep fascia and the perforating vessel is reassessed. In rotation or transposition flaps, perforators should be selected

closer to the defect. However, for V-Y advancement flaps, perforators can be anywhere in and around the mid-axis of the V-Y flap.

Reconstruction of total defect of the perineum

Total perineal reconstruction, including skin and pelvic viscera, may be indicated for advanced cancer and post-irradiated cancer recurrence, total perineal skin loss in extensive debilitating infection or following full-thickness burn or trauma.

The 'clock-face' assessment of the defect is a very useful method of considering anatomically sound options available. There are numerous perforator-based or myocutaneous flaps available beyond the clock-face territory of the lotus petal flaps and extended lotus petal flaps, which serve this purpose.¹³ These flaps are away from disease and irradiated areas of the perineum and hence will provide well-vascularized tissue for primary healing. The aim is to achieve wound closure with primary healing. Sexual function has to be sacrificed and excretory function is diverted.

Regional flaps

Deep inferior epigastric artery-based flaps

The vertical rectus abdominis myocutaneous (VRAM) flap^{14–18} should be considered as first choice for patients undergoing abdomino-perineal resection. This is an ideal flap in total perineal resection (i.e. pelvic exenteration) (Figures 44.6, 44.7 and 44.8). The perforators of the deep inferior epigastric artery are marked over the abdomen using ultrasound Doppler and/or CT angiogram. The required skin following completion of resection, the perineal defect is reassessed in terms of the size of skin defect and the volume of pelvic cavity. The deep inferior epigastric vessels can be inspected through the abdominal wound and the required skin paddle is re-marked, working out the required length of the pedicle.

The midline incision is extended superiorly and the lateral incision of the skin paddle is incised down to the rectus sheath. The rectus sheath is incised just laterally to the lateral row of perforators and a strip of the fascia is kept intact all along the rectus muscle. The flap, consisting of muscle, a strip of fascia and an island of skin, is elevated. The deep inferior vessel is identified and carefully dissected right down to its origin. The lower attachment of the muscle is also divided, which helps in orienting the flap in the pelvic cavity and in delivering the skin paddle through the perineal wound.

The muscle is sutured to the pelvic floor. The skin paddle is usually larger than the defect, hence it is de-epithelialized, leaving the exact amount of skin required. The skin paddle is

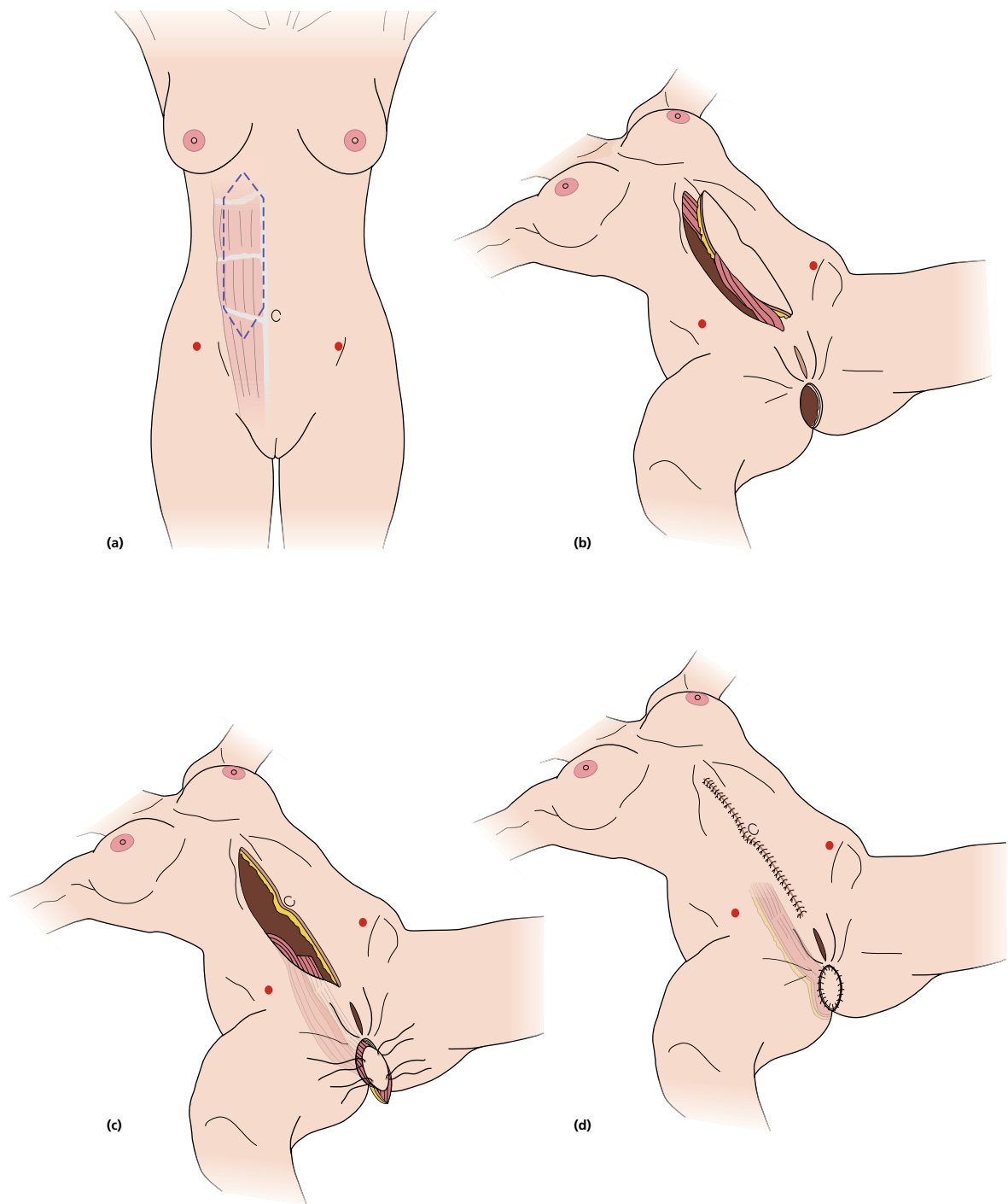
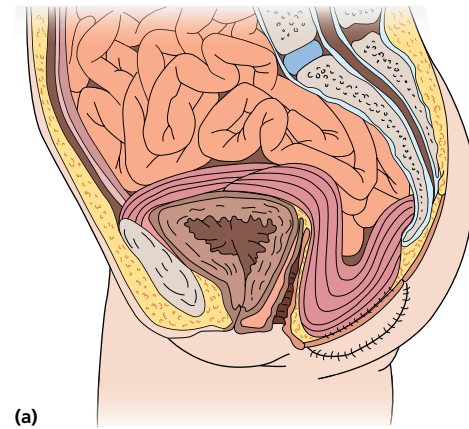
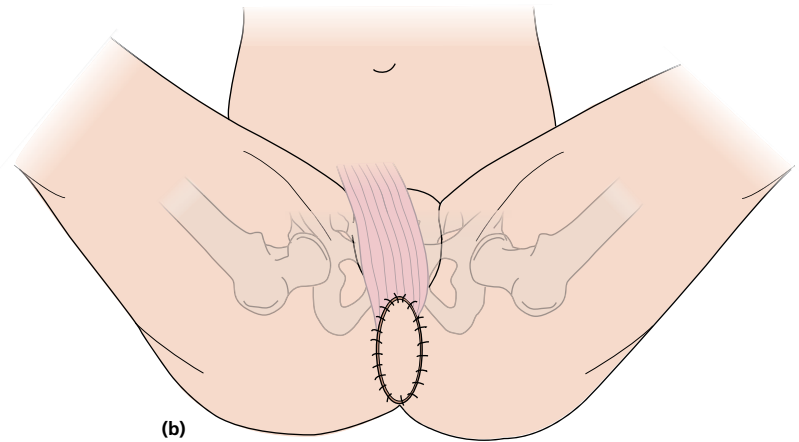


Figure 44.6 Vertical rectus abdominis myocutaneous (VRAM) flap. (a) Skin paddles, (b) VRAM flap, (c) VRAM to perineal defect, (d) wound closed.

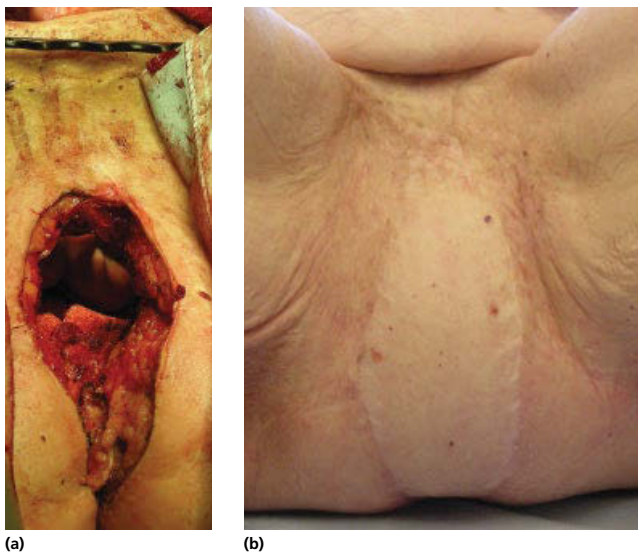


(a)



(b)

Figure 44.7 (a) Rectus muscle sutured to pelvic floor.
(b) Inset VRAM flap.



(a)

(b)

Figure 44.8 VRAM flap. (a) Defect following total perineal resection.
(b) Five year follow-up. Source: *Perforator flap; anatomy technique and clinical application*, Philip Blondeel, 2013. Reproduced with permission of Taylor & Francis.

inset, keeping two drains, and the abdominal wound is also closed in layers. Postoperatively the patient is mobilized as soon as possible.

Lateral circumflex femoral artery-based flaps

The anterolateral thigh (ALT) flap, first described by Song *et al.* in 1984¹⁹ as a septocutaneous flap, is now a workhorse in reconstructive surgery. The major advantages of this flap are that it provides a large amount of skin for harvest and that the pedicle is close enough to reach the perineum, but far enough to be away from diseased and irradiated areas of the perineum.

The blood supply of the ALT flap is dependent upon the perforators of the descending branch of the lateral circumflex femoral artery. In general, a large skin flap can be raised but a width of up to 8 cm can be closed directly and any wider skin flap requires a skin graft. The perforators are assessed for a visible pulsation and for venae comitantes. Where septocutaneous type, the vessels traverse the septum between rectus femoris and vastus lateralis muscles. If the perforators are of a muscular type, which usually

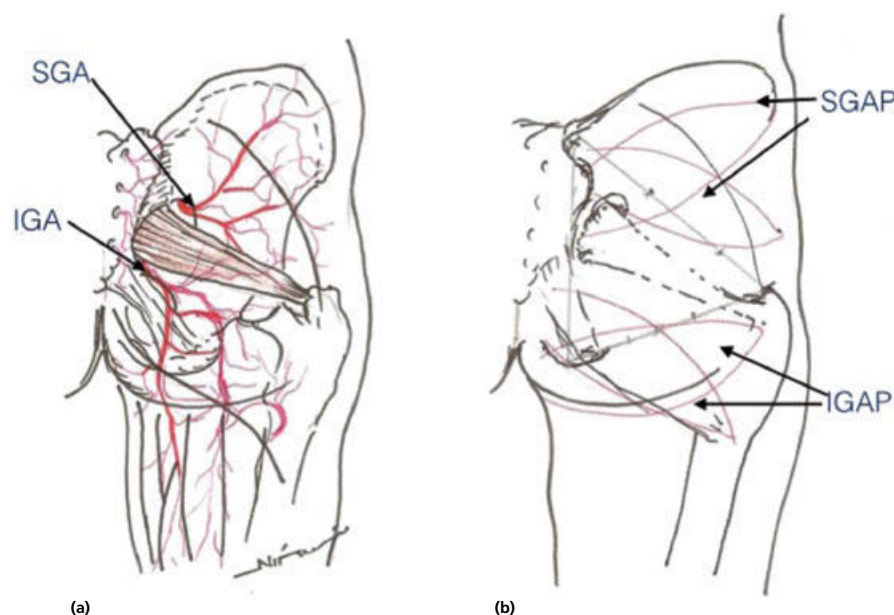


Figure 44.9 Superior and inferior gluteal artery perforator-based flaps. (a) Anatomy. (b) Skin paddle of superior gluteal artery (SGA) and inferior gluteal artery (IGA).

comes from the vastus lateralis, that part of the muscle may also be included to add bulk to the flap. The flap is passed under the rectus femoris muscle and through the subcutaneous tunnel to reach the perineum, making sure that there is no tension on the pedicle. The perineal wound is closed in layers with a drain and the donor area is either closed directly, or with a skin graft.^{20,21}

Postoperatively, the patient may be nursed in a slightly flexion and also abduction of the thighs to prevent any compression on the pedicle, although this is rarely necessary. The patient is mobilized after 48 h.

Gluteal artery-based flaps^{22–24}

The gluteus maximus muscle is a large muscle of the buttock area. It has two dominant blood supplies as a type III muscle flap. The superior and inferior gluteal arteries arise from the internal iliac artery and they emerge on either side of the pyriform muscles (Figure 44.9). The superior gluteal artery supplies the upper part of the gluteus maximus and the inferior gluteal artery supplies the lower half. The inferior gluteal artery is a direct continuation of the internal iliac artery emerging below the pyriform muscle, accompanying the sciatic nerve, inferior gluteal nerve, the internal pudendal vessel and posterior femoral cutaneous nerves. The cutaneous perforators of the inferior gluteal artery (about 8 ± 4) are concentrated in the middle third of the gluteal region, parallel to the gluteal crease.²³ The superior gluteal artery perforator or myocutaneous flaps would be suitable for the sacral part of the defect, whereas the inferior gluteal artery perforator or myocutaneous flaps would be suitable for a large part of the perineum and also in pelvic exenteration (Figures 44.10 and 44.11).

These flap are considered when VRAM or ALT flaps are not possible for reconstruction. When chosen for abdomino-perineal reconstruction, the surgery requires two different

positions of the patient. Initially, the resecting surgeon will carry out the abdominal part of the operation, including colostomy and urinary conduit and the abdominal wound is closed. The patient is then positioned in the prone, jack-knife position. Using ultrasound Doppler, the lateral groups of perforators of the inferior gluteal artery are selected in the ellipse of the skin so that the pedicle is long enough to reach without tension.²⁴ The skin flap is elevated from the lateral aspect and a small portion of the gluteus maximus muscle may be included, which not only adds to the bulk but also includes a number of perforators for the flap. Once the flap is islanded, the pedicle is dissected, ligating all other branches of the inferior gluteal artery. When the flap is transposed over the perineum, the deep layer is sutured with the pelvic floor and the skin is sutured in two layers of the perineum, with two drains.

Posterior thigh flap

The blood supply of this flap arises from the descending branch of the inferior gluteal artery. This flap contains the posterior cutaneous nerve of the thigh, which makes it a sensate flap. The course of the artery and the perforators can be mapped out by using ultrasound Doppler or by CT angiography. This helps in planning the required flap. A line is drawn from the ischial tuberosity to the lateral epicondyle of the femur. The skin paddle is centred at the midpoint of this line, with the width of the skin paddle being either the amount that can be closed directly or more if skin grafting is required. It is also possible to take two flaps or to use this in combination with other flaps. The flap is elevated by incising its lateral border and extending it towards the gluteal crease to expose the pedicle and following it up to its origin. Extra length can be obtained by dissecting the inferior gluteal artery. The flap may then be tunnelled under the skin to the perineal defect and inset.



Figure 44.10 Inferior gluteal artery flap, island flap V-Y advancement. (a) Post-perineal reconstruction wound showing intact gluteal fold flap. (b) Immediate post-op result. (c) Three months post-op result.

The postoperative regime is as described before. The donor scar of the inferior gluteal artery perforator flap will cause some discomfort for the patient during sitting for about 4–6 weeks post operatively.

Medial circumflex femoral artery-based flaps

Orticochea in 1972²⁵ and McCraw *et al.* in 1977²⁶ described the gracilis myocutaneous flap. The gracilis myocutaneous flap is a type II muscle flap, the blood supply of which is from the ascending branch of the medial circumflex femoral artery. The skin flap paddle orientation is usually in the line of the gracilis muscle, but in recent years, following the paper from Yousif *et al.* in 1992,²⁷ a transverse orientation of the cutaneous paddle has been shown to be more reliable. This is now called a transverse myocutaneous gracilis (TMG) or transverse upper gracilis (TUG) flap.^{28,29} The muscular artery for the gracilis enters the muscle about 8–10 cm from the pubic tubercle. The

gracilis muscle can be used as a myocutaneous flap, or a muscle flap in combination with other skin flaps (Figure 44.12).

The gracilis myocutaneous flap is indicated for abdomino-perineal resection with surgery carried out in the Lloyd-Davies position. A skin paddle is drawn about 1–2 cm below and parallel to the groin crease and the inferior line is drawn depending on the amount of skin available for direct closure. This gives an ellipse measuring 18–20 cm in the anterior posterior direction.

The flap is elevated from anterior to posterior in the subfascial plane until the intramuscular septum between adductor longus and gracilis is encountered. The great saphenous vein is protected and preserved (i.e. not included in the flap). The adductor longus muscle can be identified easily and the fascia over the adductor longus muscle is included with the flap. The gracilis muscle is below adductor longus and the septum between the two can be easily separated by finger dissection,

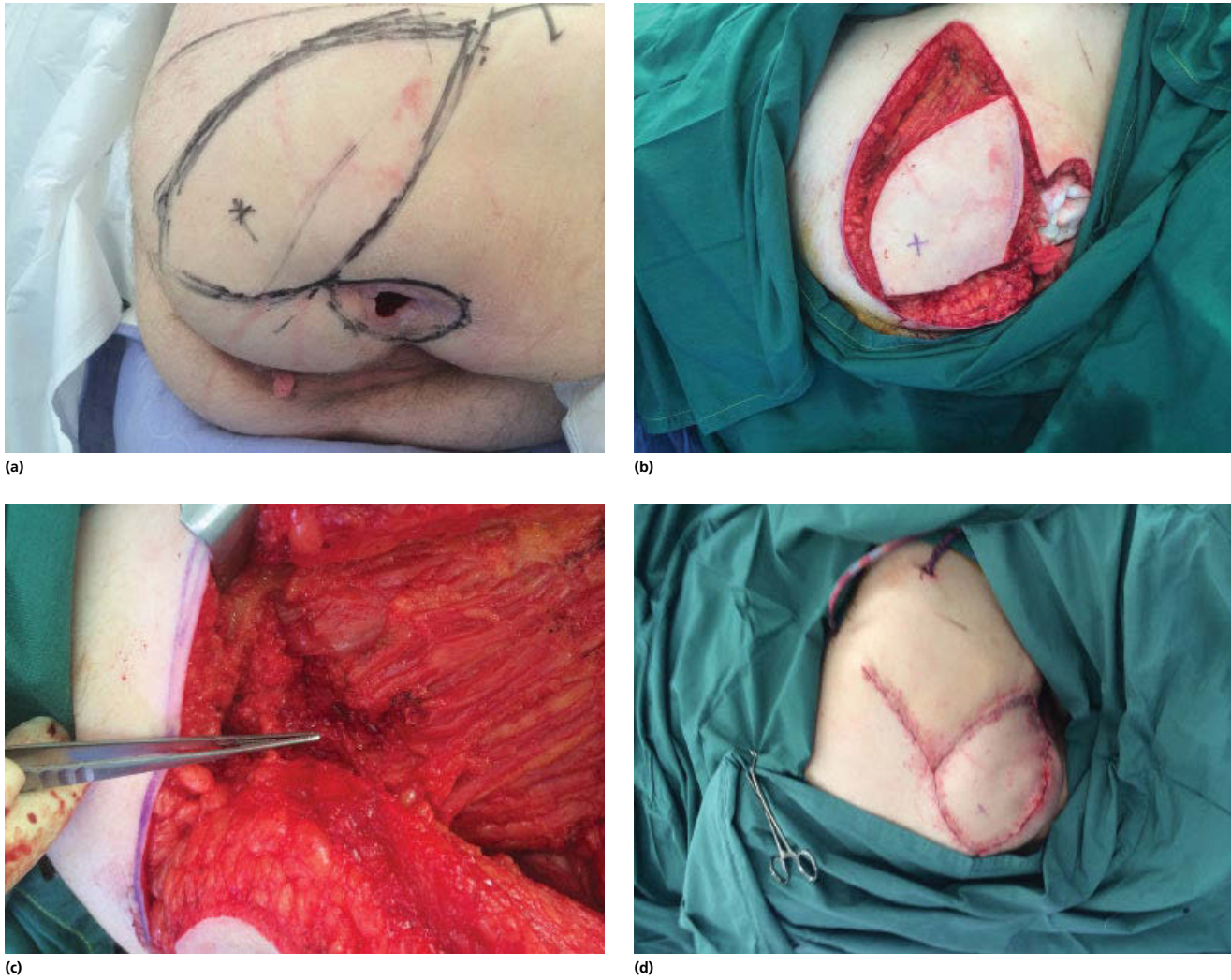


Figure 44.11 Superior gluteal artery flap islanded transposition flap. (a) Reverse flap design around the perforator. (b) Islanded flap. (c) Perforator isolation and dissection through the muscle towards the pelvis. (d) Transposition, inset and closure of donor site. Source: Photos courtesy of Mr Ross Farhadieh Reproduced with permission.

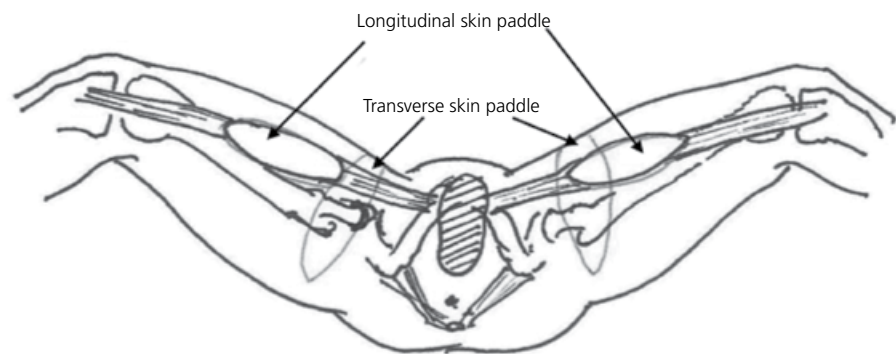


Figure 44.12 Bilateral transverse myocutaneous gracilis (TMG) design.

exposing the distal part of gracilis. The pedicle of gracilis, ascending branch of the medial circumflex femoris artery and the vein can easily be seen under the fascia.

Postoperatively, the patient is kept in a supine position with the legs slightly abducted, with a pillow under the knees to keep them flexed. After 24–48 h, the drains are removed and the patient is mobilized.

The gracilis muscle may also be considered for functional transposition for perineal reconstruction by preserving the nerve to the muscle.

Discussion

Perineal reconstruction is complex and involves a multidisciplinary approach. The ablative surgery often leaves the patients with significant and sometimes difficult lifestyle changes. Adherence to basic reconstructive surgery principles with provisions of a water-tight and reliable reconstruction offer the patients the best chance of meaningful recovery and quality of life. The lotus petal flap and the V-Y perforator fasciocutaneous flaps have become workhorses in perineal reconstruction. The perineum is basically divided into anterior and posterior halves by a line joining the ischial tuberosities. When one half of the perineum requires reconstruction, any one of the petals of lotus petal flap would be sufficient. When both halves or the whole of the perineum require reconstruction, an extended lotus petal is selected according to the required size and volume. In general, one needs three structures to reconstruct when the whole of the perineum is resected: pelvic floor, deadspace and skin cover. The first can be reconstructed either by acellular dermal matrix or de-epithelialized skin flaps. The second needs bulky but vascularized muscle or a de-epithelialized myocutaneous or fasciocutaneous flap and the last by a skin flap.

GENITAL RECONSTRUCTION

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Introduction

Congenital anomalies of the genitalia result from a variety of genetic and prenatal causes. Abnormal genitalia are seen in patients with 46,XX chromosomes, 46,XY chromosomes and mixed sex chromosomes. The expressions of these chromosomal abnormalities are the results of complex biological interplay. The manifestations range broadly and can affect multiple areas of human anatomy and physiology. In males, testosterone promotes development of the epididymis, vas deferens and seminal vesicle; and Müllerian-inhibiting substance (MIS) prevents the maturation of the uterus,

cervix and upper vagina. In females, the absence of MIS allows maturation of the uterus, cervix and upper vagina. At approximately the 9th week of gestation, normal males have produced enough testosterone to allow synthesis of DHT (dihydrotestosterone). DHT then induces fusion of the genital folds and promotes growth of the genital tubercle.³⁰

A defect in any of the genes or environmental milieu that affect this developmental process can result in abnormal genitalia. Certain developmental problems, such as bladder exstrophy, can simply disrupt normally developing anatomy. Other developmental problems, specifically those disrupting gender-specific development, result in anatomy that lies on a spectrum between normal female and male external genitalia. The Prader Scale, originally described by Andrea Prader in 1954, is often used to describe this anatomy. It is a 5-level scale where 1 represents normal female external genitalia with clitoromegaly and 5 represents normal male external genitalia.³¹

The type of reconstruction required for each patient varies depending on the cause of ambiguous genitalia and the resulting appearance. The most common causes of anomalous genitalia in those patients undergoing feminization procedures are congenital adrenal hyperplasia and mixed gonadal dysgenesis.^{32,33} Gender assignment in those with anomalous genitalia is crucial, although the timing of surgery is a controversial issue. Some have suggested that the birth of ambiguous genitalia is a neonatal emergency and requires immediate intervention. Others have advocated delaying surgery until the patient can give informed consent. Most physicians, however, suggest performing surgery prior to the age of 3. Performing surgery during this earlier time increases compliance with dilations and decreases parental concern about their child's gender.³⁴ The goals in vaginal reconstruction of ambiguous genitalia include preserving fertility, adequate sexual function and eliminating the risk of malignancy in the intra-abdominal gonads.³⁴

Vaginal reconstruction

Acquired vaginal defects are commonly due to oncologic resections, although other causes include trauma and infection. Rectal adenocarcinoma is frequently cited as the most common indication for vaginal resections, accounting for approximately half of all reconstructions. Other causes include anal squamous cell cancer, cervical carcinoma, bladder or urethral transitional cell, bladder or urethral squamous cell cancer, vaginal or vulvar squamous cell cancer, endometrial adenocarcinoma and trauma.^{35,36} According to Crosby *et al.*,³⁶ the most common surgical procedures resulting in vaginal defects are abdomino-perineal resections and pelvic exenterations.

In 2002, Cordeiro and colleagues published a classification system to define full-thickness vaginal defects.³⁵ Type I defects include those involving the anterior, lateral or posterior walls of the vagina. Type IA defects involve the anterior or lateral vaginal wall, whereas type IB defects involve the posterior wall. Type II

refers to circumferential vaginal defects. Type IIA defects encompass up to two thirds of the upper portion of the vagina, whereas type IIB defects are the result of circumferential total vaginectomies. The most common vaginal defect is a Cordeiro type IB or full-thickness posterior wall defect.^{35,36}

Vaginal reconstruction has positive effects on the psyche and has been shown to improve wound healing. Placing healthy, well-vascularized tissue in a previously radiated area prevents enteroperineal fistulas and limits deadspace, thereby reducing pelvic fluid collections.³⁷

Non-surgical options

In cases of vaginal agenesis, where a dimple of vaginal mucosa is present, often non-surgical techniques are preferred to surgical creation of a neovagina.³⁸ The Frank procedure involves the use of serial vaginal dilators. Using the dilator, pressure is placed on the vaginal dimple for 30 min to 2 h per day. Over time the width and length of the dilator are increased to allow for a neovagina that is appropriately sized for sexual activity. Some discomfort is expected during dilations, but after dilations are complete, only intermittent dilation is required if sexual activity is frequent enough to maintain patency.³⁹ Edmonds *et al.*⁴⁰ document a 94.9% success rate with dilation treatment in patients with Mayer–Rokitansky–Küster–Hauser syndrome. Ingram⁴¹ described a modification of the Frank procedure in which the serial dilators are attached to a bicycle seat upon which the patient sits. The modified positioning allows for improved patient comfort during serial dilations.

Surgical options

The reconstructive surgeon must consider multiple factors when planning the surgical creation of a neovagina. The location and size of the defect is the most important factor. However, it is also valuable to consider the patient's position on the operating room table. The oncologic surgeon may have completed the procedure with the patient in the prone or supine position. This may influence the options that are available without repositioning the patient. The type of incision, particularly whether a laparotomy incision was performed, and whether the initial incision remains open or has been closed can alter the operative plan. The desired neovagina length and width should be based on the patient's postoperative goals for sexual activity. The need for a connection between the neovagina and the uterus, when present, must be considered in congenital reconstruction.

Small defects of the vagina are amenable to *primary closure* if they do not significantly alter the length or width of the vagina. For larger defects, which are superficial or partial thickness, a *skin graft* can be considered. A vaginal conformer is often used to help promote take of the skin graft, which provides compression and immobilization of the graft to the underlying wound bed. In the paediatric gynaecology literature, the preferred method for surgical creation of a neovagina in patients with vaginal agenesis is the Abbé–McIndoe procedure.³⁸ First documented in 1938, this involves creating a space between with rectum and bladder

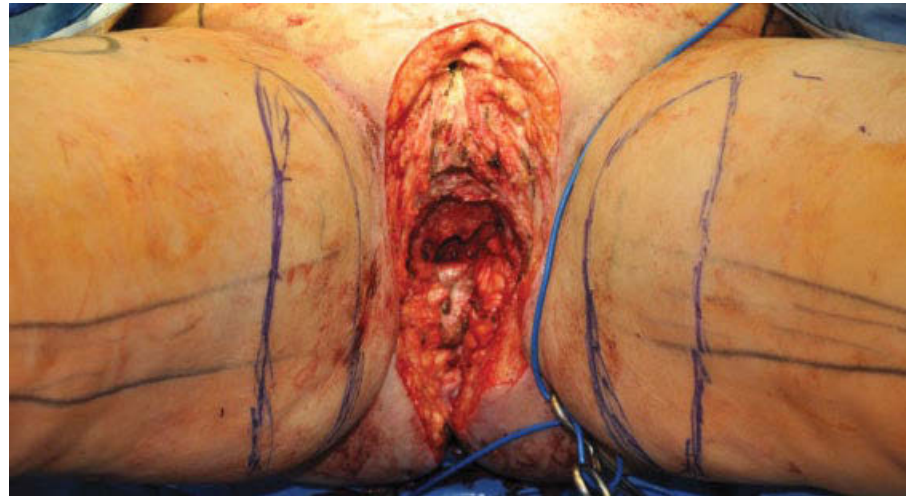
and inserting a skin graft-lined mould to create the neovagina.⁴² This procedure requires the use of a mould or vaginal conformer for 6–12 months post operatively and dilations to maintain a vagina that can be used for sexual activity. However, case series document success with this technique.⁴³ Fussey *et al.*⁴⁴ have described the use of continuous negative pressure therapy to aid in skin graft take during vaginoplasty.

The *pedicled rectus abdominis myocutaneous flap* is the work-horse of vaginal reconstruction. Cordeiro described the use of the rectus flap for both partial posterior vaginal defects and circumferential upper two thirds defects.³⁵ The myocutaneous flap can be designed and harvested with a vertical or transverse skin paddle. The senior author prefers the VRAM flap as it is aligned with the midline incision that is most often used for a laparotomy incision and, therefore, provides the greatest length of flap and greatest reach for the vaginal and perineal defect. The flap is based upon the deep inferior epigastric artery and can be passed directly into the pelvis/vaginal defect. Authors have recently described the harvest of the flap as a perforator flap for vaginal reconstructions, noting decreased donor site morbidity.⁴⁵ However, incorporation of the rectus muscle provides additional bulk and blood supply, which can be helpful in eliminating pelvic deadspace and facilitating healing.

The *pedicled gracilis myocutaneous flap*, based on the medial femoral circumflex vessels, is another versatile option for vaginal and perineal reconstruction. The flap can be harvested with a transverse or longitudinal skin paddle. Cordeiro favoured the longitudinal skin paddle for total circumferential vaginal defects, whereas the transverse skin paddle can be used for labial reconstruction (Figure 44.13). It is important to note past reports of necrosis of the gracilis skin paddle when designed distally on the muscle down the thigh. Surgeons should note that the gracilis muscle tapers distally and becomes more tendinous, and that the blood supply of an overlying skin paddle at this level becomes less reliable. Hence, a skin paddle that incorporates the proximal major skin perforator, which enters at approximately 8–10 cm below the pubic ramus, is important for maintaining maximal perfusion.

The *puddendal thigh flap* was first published by Wee and Joseph in 1989,⁸ and is often referred to as the 'Singapore flap'. The flap is an innervated fasciocutaneous flap based off perforators from the internal pudendal artery, which travel from posterior to anterior. The skin flaps are designed over the proximal medial thigh and harvested deep to the fascia of the adductor muscles. Proponents of this flap cite the benefit of an inconspicuous donor site and more reliable blood supply than the gracilis myocutaneous flap. This technique has been used in the reconstruction of both congenital and acquired defects.⁴⁶ This flap is of limited bulk and should be considered for defects that do not require a large amount of deadspace obliteration.

Intestinal conduits, including the colon and jejunum, have also been utilized for vaginal reconstruction. This type of reconstruction is more widely documented for congenital defects. Erman Akar *et al.*⁴⁷ described their experience with vaginal reconstruction



(a)



(b)

Figure 44.13 Transverse upper gracilis (TUG) flap for total vaginal reconstruction. (a) Defect after oncologic resection and flap design. (b) Final flap inset with donor site closed.

for congenital defects (33 of 34 patients) using a free vascularized jejunal flap. At a mean follow-up of 50 months, the authors noted satisfactory vaginal depth and diameter. However, six patients noted jejunal hypersecretion post reconstruction and ten patients required two lengths of jejunum opened along its antimesenteric border in order to increase luminal diameter. In contrast, the benefit of the colon in vaginal reconstruction is its larger diameter in comparison to the jejunum. Lima *et al.*⁴⁸ reviewed their 34-year experience treating 46 cases of vaginal agenesis with sigmoid colon interposition. Although they noted no cases of excessive mucus production, eight patients had stenosis, six of who required operative intervention. Of note, 34% of the group was sexually active at a mean follow-up of 12 years. Anecdotally, some patients may report a foul odour with the use of intestinal conduits as a result of mucus secretions.

Omental flaps, which have been used to treat rectovaginal and vesicovaginal fistulas as well as to close off the pelvic inlet after

pelvic exenteration, can be modified for vaginal reconstruction. Authors have documented the use of a cylindricized omental flap based off the left gastroepiploic artery with a skin graft for neovaginal creation.⁴⁹ The minimal donor site morbidity and ease of flap harvest are among the benefits of this flap. However, like other skin graft-based reconstructions, stenting is necessary for up to six months post operatively to maintain adequate vaginal depth and to prevent strictures and stenoses.

The *ALT flap* has emerged as another option for vaginal and perineal reconstruction.^{50,51} Based on the descending branch of the lateral femoral circumflex artery, the ALT flap is a versatile flap for reconstruction. The flap can be harvested as a fasciocutaneous, an adipofascial or a musculocutaneous flap. This versatility is beneficial for reconstruction in the perineum where deadspace obliteration may be required or thinner pliable tissue may be ideal. The flap is tunnelled beneath the rectus femoris and sartorius to improve pedicle length.

Complications

The most common complication after vaginal reconstruction is delayed wound healing or dehiscence. Other more devastating complications include: flap necrosis, fistula formation and pelvic abscesses. Strictures may develop which require dilations or revision surgery. Donor site infections may also be seen, as vaginal reconstruction is not a clean procedure. It is advised to use a separate clean set of instruments for flap harvest outside of the abdominal cavity if practical. After vaginal reconstruction with a rectus abdominis myocutaneous flap or an intestinal conduit, neovagina prolapse can occur which becomes bothersome to the patient. Huffman *et al.*⁵² have published a case series of correction of rectus abdominis myocutaneous flap prolapse utilizing sacral suspension.

Penile reconstruction

Acquired penile defects may be secondary to trauma, infection or oncologic resections. Penile trauma is rather uncommon since the penis has a relatively protected position between the thighs and pubic bone, but can occur, particularly in military injuries where the trunk is often protected but the lower extremities and pelvis are not and vulnerable to explosive devices. Penile skin loss occurs from necrotizing infections, burns, constrictive bands, degloving injuries as well as dog and human bites. Avulsions are most often caused by power tool injuries or motor vehicle accidents. They have also occurred from self-inflicted causes such as insertion of the penis into vacuum cleaners or other suction devices. Penetrating penile trauma is usually secondary to stab or gunshot wounds.⁵³

The most common cause of penile carcinoma requiring major resection is squamous cell cancer. Approximately 1500 new cases are diagnosed each year in the United States, accounting for less than 1% of all malignancies in men.⁵⁴ The tumour is age-related, with a peak incidence in the fifth and sixth decades.⁵³ In addition to squamous cell carcinoma, almost all tumours that involve the skin, subcutaneous tissue and smooth muscle have been found in the penis.⁵⁵ Resection can include circumcision for foreskin lesions, Mohs micrographic surgical resections, glansctomy, wide local excision, partial penectomy and total penectomy.⁵⁶

Surgical options

Preoperatively, the surgeon should discuss with the patient his sexual history as well as his goals for reconstruction. The need for multiple procedures in order to fully restore sexual function should be discussed in detail with the patient. If the defect is self-inflicted, a psychiatric evaluation is necessary prior to reconstruction. Akin to vaginal reconstruction, the reconstructive surgeon must consider the size and location of the defect.

Subtotal reconstruction

Subtotal injuries to the penis may involve the shaft and/or glans. For injuries involving the skin only, such as those due to avulsion or infection, a skin graft is the best option

for reconstruction. A full-thickness skin graft is preferred for shaft resurfacing due to decreased contracture during the healing process. Injuries to the glans only can be reconstructed using a split-thickness skin graft or a urethral flap. A urethral flap involves dissection of the penile urethra from the corpora cavernosa. The distal end of the urethra is then spatulated and folded ventral to create a pseudoglans.⁵⁷

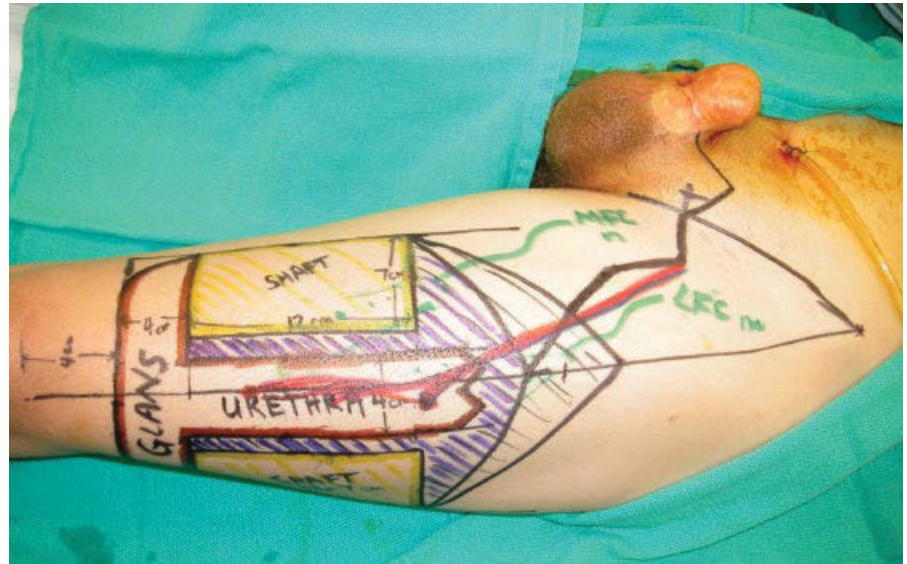
Total reconstruction

Total penile reconstruction may be based on the tube-within-a-tube concept to create the shaft and glans as well as the penile urethra. Gillies was among the first to describe a technique for total penile reconstruction.⁵⁸ Because of the unique anatomy of the erectile tissue of the penis, urologists recommend maintaining penile length whenever possible and then utilizing secondary procedures, such as excision of the suprapubic fat pad or division of the suspensory ligament, to increase penis length. However, if the trauma, tumour or infection is so severe that total penile reconstruction is required, local/regional flaps as well as free tissue transfer techniques are available.

Local and regional flaps available for phallus reconstruction include the pedicled rectus abdominis, groin and tensor fascia lata flaps. Recently, the ALT flap has been described for total penile reconstruction.^{59,60} The available width and length of skin and soft tissue which can be harvested and tailored as well as the favourable donor site make the ALT flap the favoured option for many reconstructive surgeons⁶¹ (Figure 44.14) A neurotomy between the lateral femoral cutaneous nerve harvested with the ALT flap and the dorsal pudendal nerve gives sensation to the neophallus. Insertion of a prosthetic stiffener can allow the patient to have an erect reconstruction.

Free tissue transfer techniques have allowed the use of the radial forearm free flap (RFFF), based on the radial artery, and the free fibula, based on the peroneal vessels, for penile reconstruction. The use of bone within the flap avoids a second stage surgery for implantation of penile prosthesis, which is required in soft tissue only reconstructions if erectile function is desired.⁶² However, this creates a permanently erect penile reconstruction, which can potentially be problematic. With regard to the RFFF, the conspicuous donor site involving the majority of the skin on the forearm can leave an unsightly scar.

As advances in transplant immunology continue and the immunosuppression needed to accept transplanted composite tissue decreases, penile transplants may be considered in the future. Transplantation would allow replacement of the unique erectile tissue of the penis and eliminate donor site morbidity. However, the current risks of immunosuppression remain too great for this to be a viable option in the near future. Psychological considerations and consequences associated with penile transplantation must also be factored.



(a)



(b)

Figure 44.14 Anterolateral thigh flap for penis reconstruction. (a) Flap design prior to reconstruction. (b) Completed reconstruction one month postoperatively.

Gender reassignment surgery

Gender reassignment surgery can be performed in patients with gender identity disorder to help alleviate gender dysphoria. Gender identity disorder is defined as the disturbance from a strong and persistent cross-gender identification and it cannot be concurrent with a physical intersex condition.⁶³ A variety of treatments exist, ranging from medical and hormonal therapy to gender reassignment surgery. Similar to gender assignment surgery for ambiguous genitalia, gender reassignment surgery

aims to create an outcome that optimizes appearance, urinary function and sexual function.

The timing of gender reassignment surgery is important for developing a lasting benefit. Studies of prepubertal children who were referred to clinics for gender dysphoria have suggested that fewer than 30% of those patients had persistence of this dysphoria into adulthood.⁶⁴ The percentage of lasting gender dysphoria increases with increasing age of initial presentation.

Upon embarking on gender reassignment, a number of steps should be taken prior to surgical reassignment. These include

social changes and reversible hormonal treatments.⁶⁴ The World Professional Association for Transgender Health recommends only carrying out genital surgery when the patient has reached a legal age to give informed consent and after the patient has lived for at least 12 continuous months in the gender role that is congruent with his or her gender identity.⁶⁴ A multidisciplinary approach with psychiatrists, endocrinologists, speech and language therapists, breast surgeons, plastic surgeons, gynaecologists or urologists is often employed.⁶⁵

Conclusion

Successful genital reconstruction requires open communication between the surgeon, the patient, and other medical personnel. The reconstructive surgeon must have a comprehensive picture of the patient's postoperative goals. In cases of tumour resection, the oncologic and reconstructive surgeons should develop a team approach. For congenital reconstructions or gender dysphoria cases, a psychologist or psychiatrist should be involved to help the patient navigate the complex and diverse psychosocial issues involved in gender assignment and reassignment. The reconstructive surgeon has a variety of options available to achieve functional and satisfying results in vaginal and penile reconstruction.

HYPOSPADIAS

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Introduction

Hypospadias is the commonest congenital abnormality of the penis. It is characterized by the association of three anatomical abnormalities that are present in a variable manner:

- a proximally located urethral meatus on the ventral aspect of the penis;
- a ventral curvature of the shaft of the penis (chordee); and
- a dorsally placed hooded prepuce that is deficient and incomplete ventrally.

Hypospadias results from a deficiency of the tissues forming the ventral aspect of the penis beyond the division of the corpus spongiosum, most likely due to incomplete virilization of the genital tubercle. As a consequence, there is a ventral triangular defect with the apex located at the division of the corpus spongiosum, the sides of the triangle being formed by the two pillars of the atretic spongiosum and the base by the glans. The typical hypospadiac penis has the glans open ventrally, with an absent segment of urethra of variable length. In place of the normal tubular urethra is the urethral plate which extends from the ectopic meatus up onto the glans. The urethral plate is often hypoplastic or dysplastic, and may be partially responsible for the chordee. For this reason the fibrotic tissue that forms a part of the urethral plate is referred to as the 'chordee tissue'. The

corpus spongiosum, which would normally encircle the urethra, is either absent or displaced laterally in the region of the urethral plate. More proximal to the ectopic urethral meatus, the structures forming the ventral aspect of the penis are mostly normal.

The diagnosis of hypospadias is usually made at birth but occasionally when the child is brought for circumcision. In both instances, the parents should be advised not to have the child circumcised, as the foreskin may be required for surgical reconstruction.

Classification

Hypospadias is most commonly classified anatomically according to the position of the urethral meatus (Figure 44.15), with 70% of cases being located at the glanular, coronal and sub-coronal level. The remaining 30% are described as midshaft, penoscrotal, midscrotal and perineal. Mouriquand *et al.*⁶⁶ have proposed that a more accurate means of classification would be according to the level of division of the corpus spongiosum, as the urethra can be hypoplastic and adherent to the ventral skin distal to the division of the spongiosum.

Incidence and aetiology

Hypospadias occurs in around 1 in every 250 male births, with a degree of geographical variation. In Western countries the incidence of hypospadias has been noted to increase over the

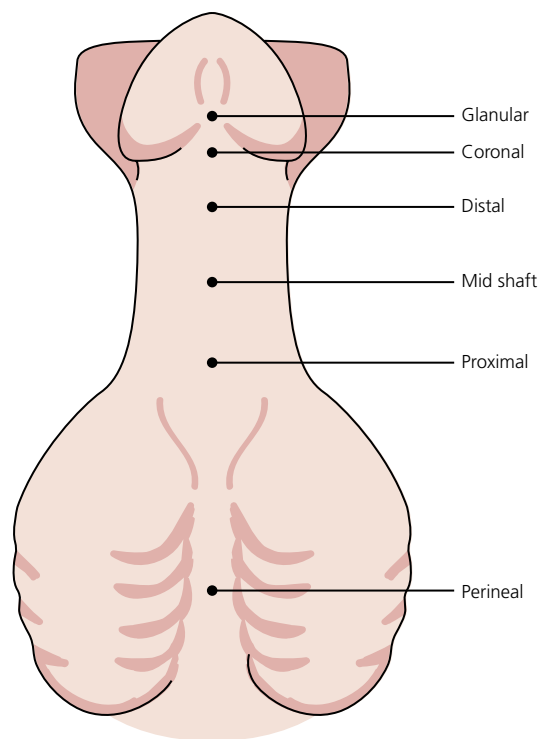


Figure 44.15 Classification of hypospadias according to the position of the urethral meatus.

past 15 years.⁶⁷ Some of the differences in prevalence reported between countries and over time are artefacts of study methodology. However, there appears to be a genuine increase in the incidence of related abnormalities such as cryptorchidism and testicular cancer, as well as a fall in male fertility. The precise cause is unknown but likely to be multifactorial. The main aetiological factor is believed to be exposure to endocrine-disrupting chemicals. These are exogenous substances that interfere with endocrine pathways. Examples would include dietary phyto-oestrogens, pesticides (e.g. dichlorodiphenyltrichloroethane (DDT)) and other toxins that are known to disrupt reproduction. There is also genetic component as well, as the incidence is higher if there is a positive family history (1 in 100 to 1 in 80 male births). For example, brothers of affected individuals have a 6–17% chance of also being affected. In a series of 205 hypospadias patients compared to 205 controls, the risk of having another child with hypospadias was increased 17-fold. In a Swedish series of 2500 patients, 7% reported one additional family member with hypospadias.⁶⁸

Associated anomalies

Although hypospadias is mostly an isolated anomaly, 10–15% of patients have additional congenital malformations, primarily affecting the urogenital system. Some of these will have a pattern of malformations suggestive of recognizable chromosomal abnormalities, as summarized in Table 44.1. Boys with hypospadias and an undescended testis should be investigated for the possibility of an underlying disorder of sex development. Karyotyping should therefore be performed in children who have hypospadias in association with other anomalies.

Surgical principles

The main goals of surgery are to straighten the penis, to reconstruct the urethral defect, to create a slit-like meatus in a near terminal position on the glans and finally to reconstruct the penile shaft skin, either with or without circumcision. A successful surgical reconstruction should allow the individual to micturate standing up with a single straight urinary stream and to allow for sexual intercourse with straight erections. At the same time, the cosmetic result must be acceptable to the parents

and the individual. This is a formidable challenge, which is perhaps why more than 300 different operations have been described for hypospadias repair.

Correction of chordee

The first step in hypospadias surgery is the correction of chordee. This begins with fully degloving the penis all the way down to the base of the penis, and sometimes beyond the penoscrotal junction. In the majority of cases (87%) this manoeuvre alone will straighten the penis as the chordee commonly results from an abnormal attachment of the ventral skin to the penile shaft. If the chordee persists after degloving then some surgeons would advocate mobilizing the urethral plate off the ventral surface of the corpora cavernosa. This can jeopardize the blood supply to the urethral plate and is thus not a popular technique.

Most surgeons in Europe would perform a dorsal plication in the corpora cavernosa to correct the residual chordee. The two most common techniques for this are the Nesbit procedure and the Baskin modification. The Nesbit procedure was initially described in 1965 and modified in 1994. It is performed by plicating the tunica albuginea by suturing two parallel incisions with a 6-0 monofilament suture. The Baskin modification⁶⁹ is performed in the midline to avoid injury to the neurovascular bundles. The correction of the chordee can be confirmed by means of manual pressure on the bulbospongiosus, without the need for a formal artificial erection test. The potential disadvantage of a dorsal plication is that it may cause shortening of the penis. For this reason some surgeons, particularly those in North America, would favour ventral lengthening. There are essentially two ways to achieve ventral lengthening. The first involves multiple superficial transverse incisions along the corpora cavernosa and the second involves a single incision through the tunica albuginea at the point of maximal curvature and the placement a graft into the resulting defect, using dermis,⁷⁰ tunica vaginalis⁷¹ or small intestinal mucosa.⁷²

Reconstruction of the urethra

When the urethral defect is relatively short and the urethral plate is wide, the urethra can be reconstructed by simple tubularization of the lateral edges (Thiersch–Duplay technique). If the urethral plate is narrow then a midline longitudinal incision may provide further width to allow tubularization (Snodgrass technique). The other option would be to suture a pedicled rectangular flap of penile or preputial skin to the lateral edges (Onlay island flap procedure).

With a lengthier urethral defect, there are essentially two options. The first would be to perform the surgery in a staged manner, with the first stage involving placing a free or pedicled graft (inner prepuce, penile skin, postauricular skin or buccal mucosa) on the ventral aspect of the corpora and glans. Once the graft is mature (after six months) this can be used to fashion the urethra by tubularization in the second stage. This two-stage technique has gained significant popularity worldwide. The alternative would be to substitute the urethra with a tubularized

Table 44.1 Syndromic gene mutations associated with hypospadias

Gene	Disorder
<i>SF1</i>	No adrenal glands
<i>WT 1</i>	Wilms' tumour
	Denys–Drash syndrome (Wilms' tumour and renal failure)
	Frasier syndrome
<i>SOX9</i>	
<i>HOXA13</i>	Hand–foot–genital syndrome

flap of pedicized inner prepuce (Duckett procedure) or buccal mucosa. The Duckett procedure, once very popular in the 1980s, has lost favour due to high reoperation rates.

Reconstruction of the penile shaft skin

Once reconstruction of the urethra has been completed, it is customary to cover the urethroplasty with a 'waterproofing' layer which is harvested from the adjacent dartos tissue or a dorsal pedicle of dartos tissue which is brought ventrally to cover the urethra. Finally the penile skin is reconstructed with or without a circumcision.

Surgical techniques

The main challenge of hypospadias surgery is whether the desired outcome can be achieved in a single operation or will require a staged procedure. In some cases this cannot be determined until during the actual surgery.

Most surgeons focus on two or three preferred techniques and presently the Snodgrass technique or tubularized incised plate (TIP) repair is the favoured technique for distal hypospadias. For the more proximal repairs, most surgeons use a two-stage repair ('Bracka technique'). The remainder of this chapter will focus on these two most common techniques for hypospadias repair.

The surgery is ideally performed between 6 and 18 months age. If a two-stage procedure is anticipated, then the aim should be to complete both stages by the age of 2 years. This is based on emotional, cognitive and psychosexual factors as well as improved tissue healing in the younger patient. Some surgeons prefer to do the surgery around the age of 5 years due to anaesthetic limitations. Another advantage of performing surgery at an older age is that the tissues may be more robust, making the repair somewhat easier.

There is considerable debate regarding preoperative penile hormonal stimulation when the penile size is very small. It can be administered systemically as beta-human chorionic gonadotrophin (beta-HCG) or testosterone or topically in the form of dihydrotestosterone gel. There is emerging evidence, however, that their use can lead to delays in healing.⁷³

Tubularised incised plate (Snodgrass) repair

Warren Snodgrass described the technique in 1994.⁷⁴ It is versatile, easily performed, has fewer complications and results in a cosmetically acceptable slit-like meatus. Relative contraindications include proximal hypospadias, a distal hypospadias with significant chordee or a poor glans groove, which would be too narrow for tubularization.

Operative technique

Degloving the penis and chordee correction

A polypropylene traction suture is placed in the glans just beyond the anticipated dorsal lip of the neomeatus. The urethra and overlying skin are assessed with a urethral sound or the tip

of the forceps. The distal portion of the urethra may be quite thin and adherent to the skin. Usually, a circumscising skin incision is made 1–2 mm proximal to the meatus and the shaft skin is degloved to the penoscrotal junction. If the urethra is excessively thin, a U-shaped incision is made extending to more healthy tissues. At this stage the penis is assessed for any chordee and this can be corrected (as described above) (Figure 44.16).

Urethroplasty

The urethral plate is separated from the glans wings by parallel longitudinal incisions. The glans wings are mobilized laterally, taking care to avoid damage to the margins of the urethral plate. In most cases the urethral plate is not sufficiently wide at this point to tubularize into a functional urethra. Therefore a relaxing incision is made in the midline from within the meatus to the end of the urethral plate. This incision should extend through the epithelium to near the underlying corpora cavernosa. An 8Fr stent is passed into the bladder. The urethral plate is then tubularized over this stent using a fine monofilament suture. The neomeatus should be generously sized. Adjacent dartos layer is then used to cover this suture line; alternatively a dartos pedicle is developed from the dorsal shaft skin and transposed to the ventral surface. Following this the glans wings are mobilized and then closed in two layers using a fine monofilament suture.

Skin closure and dressing

The skin is then reconfigured, ensuring that there is enough skin to cover the deficiency of skin on the ventral surface. The excess dorsal skin is excised to complete the circumcision. The stent is secured in place using the traction suture and an expanding foam dressing (Cavicare®) is applied and this is then covered with Elastoplast®. This remains *in situ* for 7 days with a one week course of antibiotics and anticholinergics to prevent bladder spasms.

Outcomes

With careful patient selection, the results of the TIP repair are very favourable and reproducible. Snodgrass reported a multi-centre experience using the TIP urethroplasty.⁷⁵ The study included 148 boys operated on by six paediatric urologists. All patients had a vertically orientated and normally placed meatus. The cosmetic appearance was considered superior to that obtained by standard flap techniques, with a 7% complication rate. Five boys developed a fistula, two of these closed spontaneously, three boys developed meatal stenosis and three had a partial glans dehiscence.

Two-stage repair

The two-stage technique described by Bracka⁷⁶ is indicated when the meatus is located at the midshaft or more proximally, in the presence of severe chordee and in those with an inadequate urethral plate. This technique is also excellent for failed hypospadias repairs.⁷⁷ The main aim of the first stage is to lay the foundation for the construction of a straight, aesthetic and

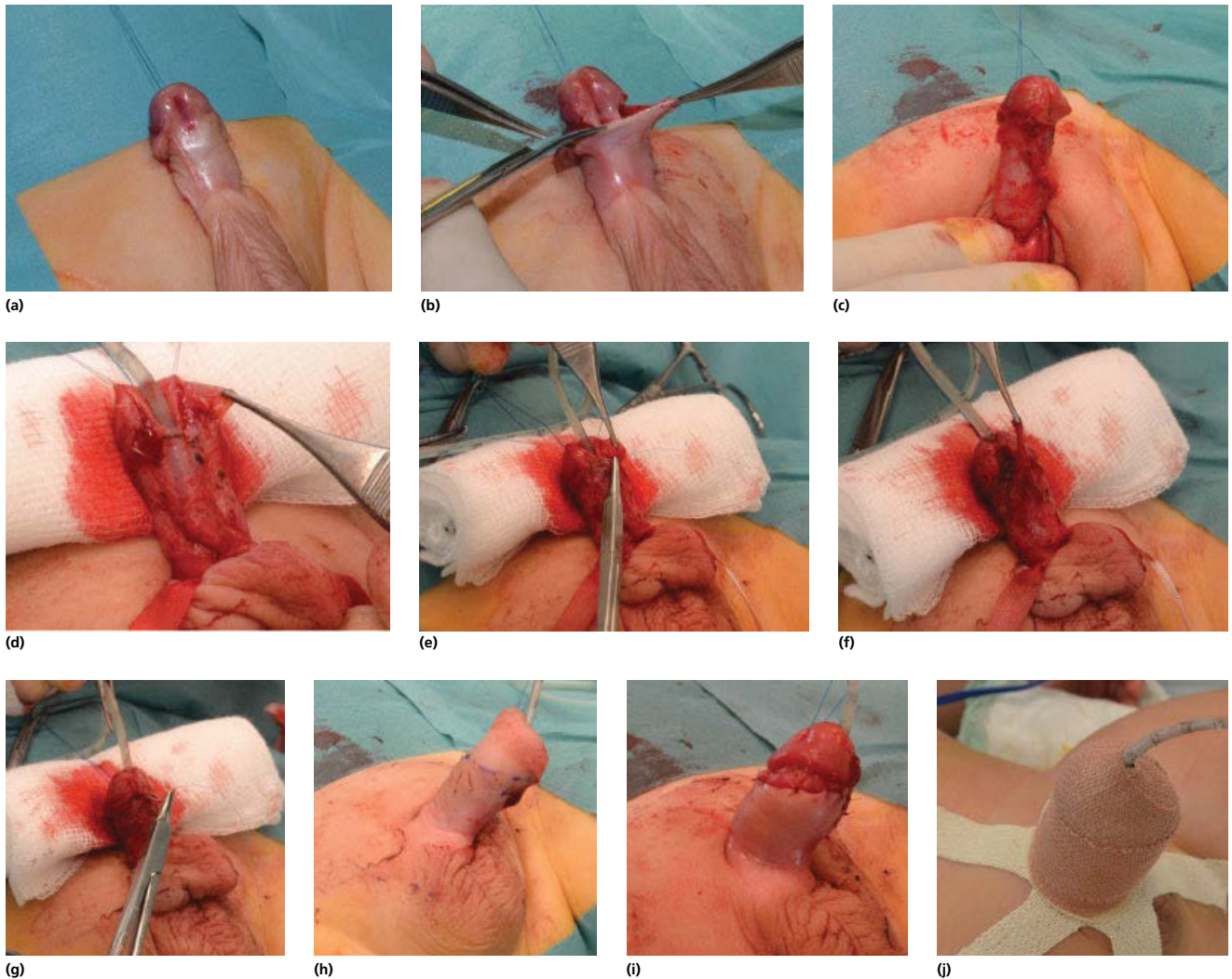


Figure 44.16 (a) Placement of the glans stitch. (b) Degloving of the penis. (c) Assessment of chordee once fully degloved. (d) Glans wings are formed. (e) The urethroplasty is performed. (f) Dartos waterproofing layer is secured over the completed urethroplasty. (g) The glans wings are closed in 1 or 2 layers. (h) The excess dorsal skin is excised. (i) The excess skin is reconfigured to complete circumcision. (j) Cavicare and Elastoplast dressing are applied.

normally functioning penis in the second stage. The first stage comprises correction of the chordee, circumcision and grafting the ventrum of the penile shaft and glans.

The ideal material for the graft should be non-hair bearing, robust and supple. It should be able to be harvested without leaving a cosmetic defect. The inner preputial skin is most commonly used for the graft, as it is readily available. In its absence posterior auricular skin is a good alternative as it is relatively thin, pliable and essentially non-hair bearing. It also leaves an inconspicuous scar. Some surgeons would prefer to use buccal mucosa, as it is abundant and particularly useful when balanitis xerotica obliterans is a complication of previous repairs with skin grafts. Buccal mucosa can be harvested from the inner cheek or a combination of inner cheek and inner lip. The parotid duct located near the upper second molar should be identified and carefully preserved.

First stage operative technique

Degloving and chordee correction

The penis is fully degloved down to the penoscrotal junction following a circumscribing incision. Dorsally the dissection continues superficial to Buck's fascia and on the ventral aspect the urethral plate, Buck's fascia and the corpus spongiosum are mobilized from the corpus cavernosa and excised. The meatus moves to a more proximal position and this should be opened ventrally to ensure it is of adequate size. The glans is incised in the midline, deep enough to clearly visualize the corpora cavernosa. The dorsal limit of this incision will correspond to the dorsal margin of the future external urethral meatus. At this stage if there is still residual chordee then one of the techniques described previously can be utilized (Figure 44.17).



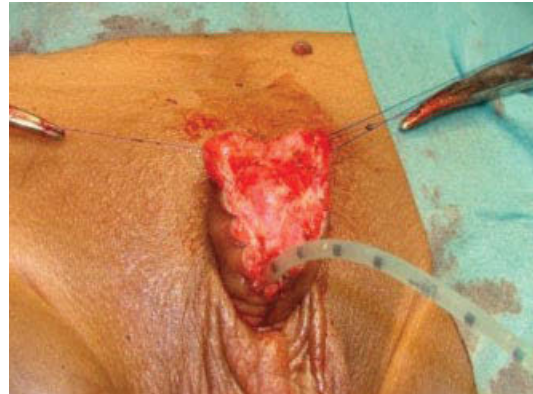
(a)



(b)



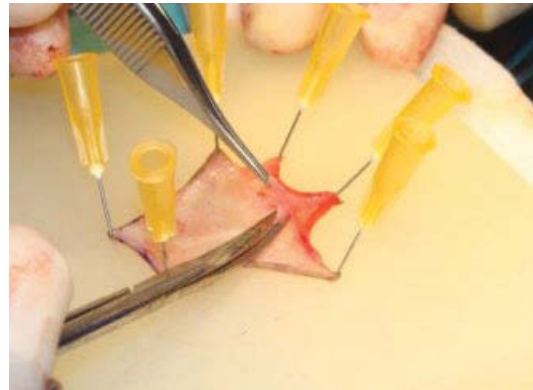
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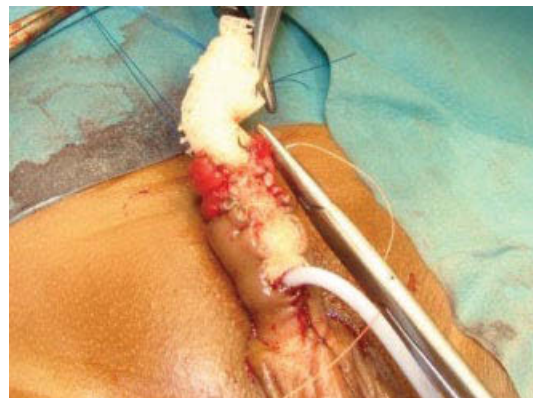
(e)



(f)



(g)



(h)

Figure 44.17 (a) Midshaft hypospadias suitable for a two-stage procedure. (b) Penis is fully degloved and any chordee is corrected. (c) The glans is split in the midline. (d) The penis is split in the midline to allow placement of the graft. (e) The graft is harvested from the dorsal prepuce. (f) The graft is prepared on a silicone block. (g) The graft is secured in place. (h) The vaseline gauze dressing is secured in place.

Harvesting the graft

The size of the graft needs to be adequate to fully cover the ventral defect without tension. The graft is usually harvested from the inner and/or outer aspect of the prepuce, or alternatively the posterior auricular skin or buccal mucosa. On occasions composite grafts may be required if the defect is large. The graft is tacked down to a silicone board to facilitate removal of the surplus subcutaneous fat and alveolar tissue. This process should result in a translucent membrane.

Securing and immobilizing the graft

The graft is placed onto the defect, trimmed to size and secured with fine monofilament sutures. Fenestrations may be made in the graft to allow any haematoma to escape. A few midline quilting sutures are recommended to secure the graft in place.

A roll of Vaseline gauze dressing is secured onto the graft with sutures. This will minimize shearing forces on the graft and is supplemented with a foam dressing (Cavicare) and Elastoplast. The dressing is left *in situ* with a dripping stent for 7 days.

Post op care

The dressing should be removed under general anaesthetic, at which stage the graft can be inspected. The parents should be instructed to apply an antibiotic ointment (chloramphenicol) three times daily to the graft for a period of two weeks. After a further four weeks the graft should be moisturized daily with a simple emollient cream.

Second stage operative technique

The second stage is usually performed after a minimum period of six months. The graft should be smooth, well vascularized and the glans cleft sharply delineated. A U-shaped incision on the grafted skin is marked from the ventral limit of the neourethral meatus with the lower limits skirting closely around the margin of the proximal urethral meatus (Figure 44.18). The marked ventral strip is incised and mobilized to allow tubularization of the neourethra over an 8Fr stent. A layer of adventitia should be used to cover the urethroplasty. The glans wings are mobilized and closed in one or two layers, although this may not be possible in all cases. The skin is approximated and the stent is secured using the glans traction suture. A Cavicare foam dressing is applied and secured with Elastoplast. The dressing and stent are then removed after 7 days in the outpatient setting.

Outcomes

A study on the outcome of the two-stage repair in 62 boys was published from the authors' institution.⁷⁸ Indications for a two-stage repair included proximal meatus (midshaft, penoscrotal or perineal), poor glans groove and lichen sclerosis with a median age at surgery of 27.6 months. Inner preputial skin was the graft of choice and was successful in all the patients. One patient developed a haematoma. Overall cosmetic results were

subjectively excellent. Complications included partial glans dehiscence (3 patients), residual mild curvature (3) and meatal stenosis (3).

Complications

Complications include bleeding, infection, meatal stenosis, urethrocutaneous fistula and urethral stricture.

Bleeding

The most common early complication is bleeding but it is rarely significant. Bipolar diathermy is used during the surgery for precise pinpoint cauterization. Excessive cauterization should be avoided to avoid tissue ischaemia. The risk of bleeding is minimized by using the supportive foam dressing and applying the compressive Elastoplast. Early haematoma formation can lead to infection, fistula formation and breakdown of the repair. It is rare for a patient to return to theatre for bleeding, and if required the haematoma should be evacuated, the bleeding controlled and the dressing reapplied.

Infection

Skin infections following hypospadias repair are uncommon as the patients are maintained on a 7-day course of oral antibiotics. If following dressing removal an infection is evident, the antibiotic treatment should be broadened and continued for a further 7–10 days.

Meatal stenosis

Ensuring a wide distal anastomosis while preserving good blood supply can minimize the incidence of meatal stenosis. It may result from ischaemia of the glans wings or an overzealous attempt to terminalize the urethral meatus in the setting of an inadequate glans. In such cases it may be pertinent to leave the meatus in a coronal location. The child may present with straining to void, a fine urinary stream, spraying or urinary tract infections due to incomplete emptying of the bladder. When meatal stenosis is suspected, the child should be evaluated with an assessment of the urinary flow rate and a careful external inspection. If confirmed, the patient will require a meatal calibration and meatoplasty. Meatal stenosis can occur many years following the hypospadias repair and in these delayed cases, balanitis xerotica obliterans should be suspected.

Urethrocutaneous fistula

Fistulas are the most common significant complication following hypospadias repair. A fistula may be seen at the time of dressing removal and some of these may heal spontaneously. The patient may present with a history of passing urine with two streams or dripping from the ventral surface of the penis. Usually a dimple can be seen on the ventral surface proximal to the neomeatus. It is always important to exclude a distal obstruction at the time of fistula repair.

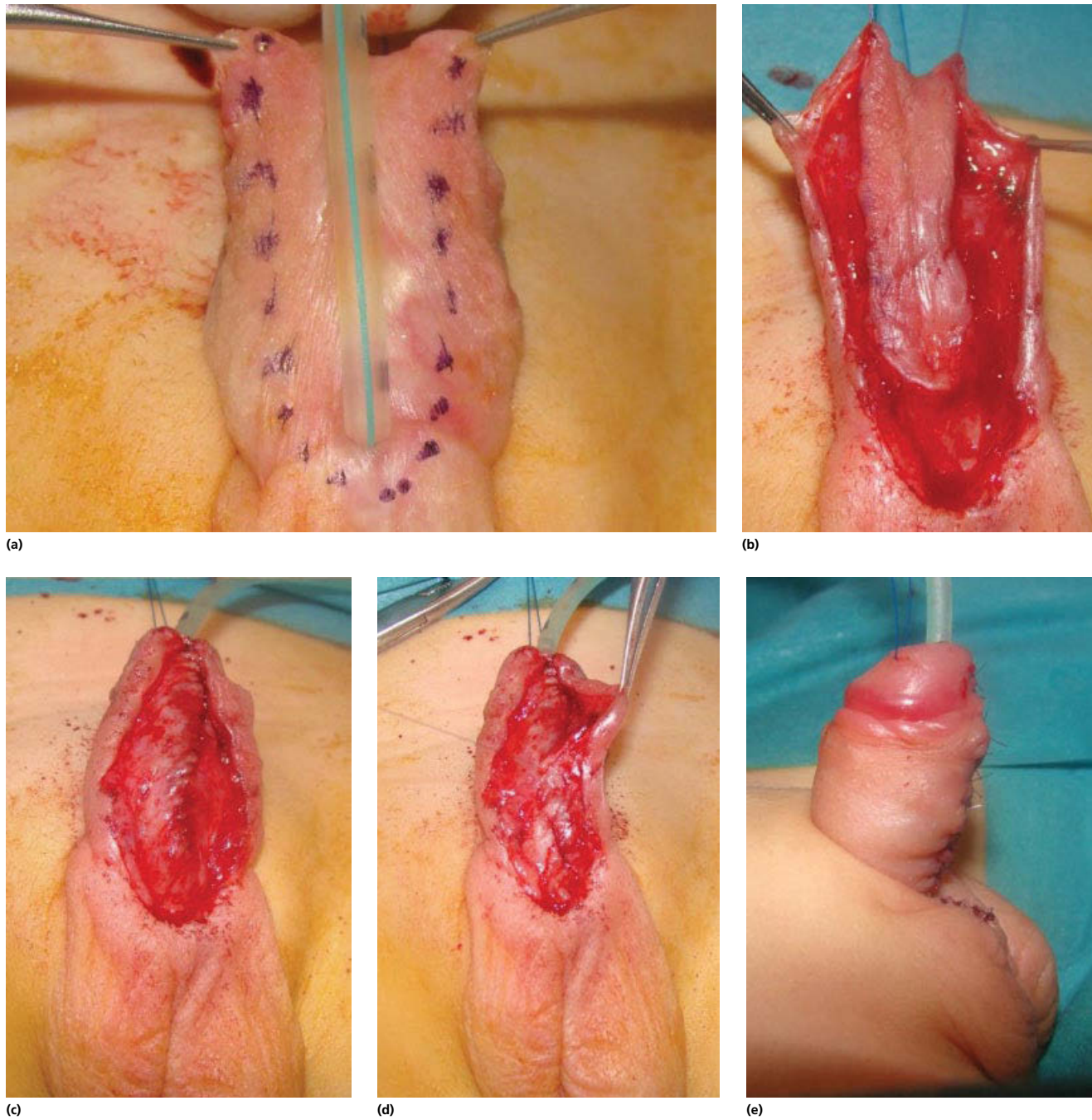


Figure 44.18 (a) The location of the incision is marked. (b) The incision is made with a scalpel and/or monopolar diathermy. (c) Urethroplasty is performed in one or two layers. (d) Waterproofing layer is secured over the urethroplasty. (e) Skin closure.

The reported fistula rate for a single stage repair is 10–15%. This may be lower than the actual incidence as small fistulas may not become apparent until years after the procedure. Success rates for fistula repair are in the region of 90%.⁷⁹

Urethral stricture

Urethral strictures are less common with modern techniques. They may respond to urethral dilatation but more often require a two-stage redo urethroplasty.

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CHAPTER 45

Pressure sores

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Introduction

Pressure sores – also known as pressure injuries, pressure ulcers, decubitus ulcers and bedsores – are a common problem among certain patient populations. A pressure sore comprises a 'localised injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction'.¹ The development of a pressure sore has implications for patients and healthcare providers, increasing length of admission and requiring increased utilization of healthcare resources. Australian studies have shown that developing a pressure sore increases length of hospital stay by a median 4.31 days, resulting in AU\$285 million in additional healthcare costs.^{2,3} With an ageing population and more widespread predisposing comorbidities, the overall prevalence of pressure sores and their associated costs is likely to increase. In this setting there is great need for the development of specialist multidisciplinary teams for the care of patients with pressure sores, including reconstructive surgeons for the management of advanced, complex, or refractory pressure sores.

Epidemiology

Pressure sore incidence and prevalence are determined by the characteristics of the population studied. The normal response to an injurious stimulus, such as the prolonged application of pressure, is withdrawal from the noxious source. However, in some individuals this does not occur, either because the pain stimulus is not present, as in sensory impairment, or because of an inability to reposition the body in order to redistribute pressure. Such patients include the elderly, the acutely unwell, patients who are immobilized as a result of spinal cord injury or following surgery, and patients with sensory impairment due to diabetes or chronic neurological conditions. Other risk factors include a past history of pressure sores, obesity, smoking, and peripheral vascular disease, among others (see Box 45.1).

The prevalence of pressure sores in the general population is low. A study of the population of a county in Denmark found a prevalence of 0.04%,⁵ and an American study of elderly patients seen in general practice found a prevalence of 0.31–0.7%.⁶ However, the epidemiology of pressure sores among hospitalized patients is of greater interest, partly because hospitalization is a significant risk factor for pressure sore development, and partly because it is increasingly used as an indicator of health-care quality and safety. A number of studies have examined the epidemiology of pressure sores among hospital inpatients, resulting in prevalence ranging from 10.2 to 19.7%, and incidence from 0 to 7.3%.^{7–12} Such wide variation partly results from differences in study design. However, there are two further major difficulties that prohibit study comparison.

The first is the variable nature of the population studied. As noted above, it has long been recognized that pressure sores are more common among certain patient populations, such as the elderly, among patients with spinal cord injuries, and among patients with sensory impairment.⁵ As such, the prevalence of pressure sores in a given hospital or institution is significantly influenced by the characteristics of the patient population serviced by that facility than any difference in quality of care or pressure sore prevention strategies. As an example, a centre that specializes in the care of patients with spinal cord injuries is likely to have a much higher incidence of pressure sores than other institutions which may be identical in every other regard. The second difficulty is how the study defines a pressure sore. Though most studies now use the National Pressure Ulcer Advisory Panel/European Pressure Ulcer Advisory Panel (NPUAP/EPUAP) classification system, they may differ on whether or not the milder forms are included, such as a grade 1 pressure injury which comprises only non-blanching erythema (see below, under Assessment).

The EPUAP collected detailed data in 2001 and 2002 and found an overall prevalence of pressure sores in five European hospitals of 18.1%.¹³ However, the majority of these consisted of only a grade 1 pressure injury, with a minority (2.5% of the

Box 45.1 Risk factors for development of pressure sores⁴

- Past history of pressure sores
- Immobility (spinal cord injury, post-surgery/injury, critical illness)
- Neuropathy (spinal cord injury, diabetic neuropathy, other neurological causes)
- Abnormal posture (musculoskeletal abnormalities, spastic contractures)
- Anaemia
- Poor nutrition
- Diabetes
- Peripheral vascular disease
- Obesity
- Poor skin quality (age, endocrine disorders)
- Smoking

total) suffering the most severe, a grade 4 pressure injury. For these reasons it is important to carefully review the methodology when reviewing studies of the epidemiology of pressure sores, and they should be interpreted warily if used as indicators of healthcare quality and safety.

Pathophysiology

Pathologically, pressure sores are areas of localized tissue necrosis resulting from external forces.¹⁴ As the name suggests, external pressure is the key causative element in their pathogenesis. In experimental studies of the effect of pressure on limb vascular volume using human volunteers in the 1940s, Landis established a threshold pressure of 35 mmHg, noting that external pressures above this figure resulted in reduced vascular volumes, likely reflecting capillary occlusion.¹⁵ This process, if repeated or sustained, ultimately results in ischaemic necrosis of skin, subcutaneous tissue and deeper tissues. Further studies by Kosiak demonstrated irreversible changes in muscle of rats subjected to 70 mmHg of constant pressure for a period of 2 h or more. Such changes were also seen in rats exposed to pressure which was applied and removed at 5 min intervals, but only at much higher pressures (155 mmHg). Importantly, it was also established that denervated muscle required the same magnitude and duration of pressure in order to demonstrate signs of injury and that normal and denervated muscle had the same susceptibility to pressure injury. This was contrary to prior thinking that supposed that the absence of neurotrophic factors secreted by functional nerves was causative in pressure sore pathogenesis.¹⁶

More recent studies in an animal model have demonstrated pressure sore formation and progression resulting from repeated and sustained application of pressure, the extent of injury varying according to these two factors.¹⁷ In combination with external pressure, shear stress and the presence of moisture may contribute to the extent of injury.¹⁸ Shear stress is a mechanical force acting parallel to the plane of compressive pressure. In the

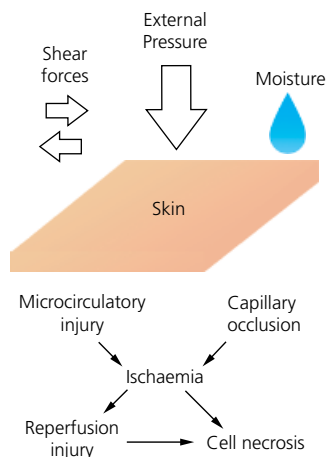


Figure 45.1 Pressure sore pathogenesis.

presence of a significant degree of shear stress, the magnitude of external pressure required to cause a pressure injury is significantly reduced.¹⁹ This is hypothesized to occur due to the deforming effect of shear stress on tissues deep to the skin resulting in direct injury through sustained deformation of cells and worsening of ischaemia by microcirculatory injury.^{20,21} Recent research has also identified reperfusion injury as an important factor in the pathogenesis of pressure sores, with greater tissue necrosis and reduction in functional capillary density following repeated compression and release of tissues by comparison with compression alone.²² The interaction of these biomechanical factors in the pathogenesis of a pressure sore is complex, and research into their relative contributions is ongoing. However, it is clear that the key pathological insult in the formation of a pressure sore is repeated compression of tissues by external pressure over a prolonged period, which may be exacerbated by contributory factors including shear forces, moisture, and reperfusion injury (Figure 45.1).

Pressure sores have been described as occurring in many locations, though the vast majority occur over bony prominences in the lower limb. The aforementioned epidemiological study by the EPUAP found the following to be the commonest sites of pressure injury of any severity, in descending order (percentages stated according to data collected in the United Kingdom): sacrum (37.5%), heel (26.2%), ischium (13.7%), elbow (10.3%), ankle (6.4%) and hip (5.8%).¹³ The reason for pressure injuries occurring most frequently over bony prominences may seem intuitively obvious; nevertheless the physiological explanation follows. Sangeorzan *et al.* compared the effects of applied external pressure to skin overlying the subcutaneous surface of the tibia with that over tibialis anterior. The researchers established that much greater external pressure was required to reduce capillary perfusion in skin overlying muscle than in skin directly overlying bone.²³ Crucial to this is the realization that the subcutaneous (interstitial) tissue pressure is of greater physiological significance than the external

pressure. The interstitial pressure resulting from a given external pressure is determined by the nature of those tissues and whether they overlie bone. Over bony prominences where there is a thinner layer of soft tissue between skin and bone, a relatively minor external pressure may result in significant, ischaemia-inducing interstitial pressure.

Prevention

Effective pressure sore prevention requires ongoing assessment of patients for risk factors, together with appropriate interventions to minimize risk to vulnerable patients. Pressure sore risk factors are common in the hospitalized population (see Epidemiology, above).

Assessment of risk

Pressure sore risk assessment should be performed for all patients on admission and repeated daily in order to identify at-risk patients and track response to interventions. A number of structured risk assessment tools have been developed to identify patients at risk of developing a pressure sore. The Norton scale was developed in 1962, from which were developed the Waterlow and Braden scales in 1985 and 1987, respectively. The latter two are in widespread use today and have been demonstrated to have a high sensitivity in predicting patients likely to develop a pressure sore.^{24,25} There is, however, no good evidence to suggest that the use of these tools results in a decreased incidence of pressure sores.²⁶ Regardless of whether or not a scale is used, assessment of patients for pressure sore risk allows identification of patients likely to benefit from prevention strategies.

Prevention strategies

Interventions for the prevention of pressure sores follow two main strategies. First, the extrinsic causes of pressure sores (see Pathophysiology, above) should be minimized. The effect of external pressure may be ameliorated using pressure redistribution techniques, such as manual repositioning and the use of appropriate lifting devices for patient transfers. There is no evidence to recommend any particular manual positioning regimen over any other.²⁷ Use of pressure-relieving support surfaces such as gel-filled overlays during surgery and air mattresses or cushions on the ward are also beneficial, though again there is nothing to suggest the superiority of any particular surface type, except over basic mattresses.²⁸ Medical-grade sheepskins are also of benefit in reducing pressure sore risk, which may be particularly relevant in the nursing home environment.²⁹ However, if achievable, early mobilization is the best strategy for external pressure reduction. Daily skin care and prompt cleaning and changing following episodes of incontinence reduces the effect of moisture and may allow the identification of early-stage pressure injuries.

Second, modifiable risk factors should be managed. The management of any medical comorbidities should be reviewed and the patient's nutritional state assessed and managed if

Box 45.2 Institute for Healthcare Improvement recommendations for pressure sore prevention

- Conduct a pressure sore admission assessment for all patients
- Reassess risk for all patients daily
- Inspect skin daily
- Manage moisture
- Optimize nutrition and hydration
- Minimize pressure

necessary. Patients should be discouraged from smoking, and obese patients should be counselled regarding the benefits of weight reduction. These strategies are summarized in the Institute for Healthcare Improvement (IHI) recommendations for pressure sore prevention (Box 45.2).

Assessment

The aims of assessment of the patient with a pressure sore are: to establish the severity of the pressure sore; to evaluate treatment options and their appropriateness and acceptability for the individual patient; and to assess risk of complications and recurrence of pressure sores.

General assessment

If the patient has a past history of pressure sores, a complete history of their prior management should be taken. An evaluation of the patient's general health status should be made, including reviewing their complete medical history, to identify comorbidities such as diabetes mellitus, peripheral vascular disease and sensory impairment, which may increase their risk of complications such as recurrence or the development of further pressure sores. In particular, diabetic control should be assessed through testing of HbA1c levels, and referral to a specialist endocrinologist considered if control is poor. Furthermore, a complete functional assessment should be completed in order to determine the patient's cognitive state and capacity for ambulation. A full nutritional assessment should be performed, including testing of serum albumin. Where there are concerns regarding the patient's mood or mental state, formal psychological assessment is recommended. The patient's social situation and support network, including the availability and appropriateness of caregivers should be examined, and the goals of care should be discussed with the patient and their family, especially in cases where overall prognosis is poor. In palliative patients, or if the patient's medical or nutritional status indicate that a poor outcome following surgery would be likely, conservative management of the pressure sore is indicated.

Wound assessment

Features of the pressure sore should be ascertained by taking a history from the patient regarding symptoms, including pain, associated features to indicate infection such as fever

Box 45.3 Pressure sore features

- Size of pressure sore
- Location of pressure sore
- Stage of pressure sore
- Presence of eschar or exudate
- Signs of local infection and collections
- Evidence of osteomyelitis

Table 45.1 Classification (International NPUAP-EPUAP Pressure Ulcer Classification System)³¹

Stage	Features
Stage I	Non-blanchable redness of intact skin. Area may also be painful, firm, soft, warmer, or cooler by comparison to adjacent tissue
Stage II	Partial-thickness skin loss involving epidermis and/or dermis presenting as a shallow open ulcer with a pink wound bed without slough/eschar. May also present as open/ruptured fluid-filled blister
Stage III	Full-thickness skin loss with subcutaneous fat visible but without exposure of deeper structures such as bone, tendon, or muscle
Stage IV	Full-thickness tissue loss with extensive tissue necrosis and exposure and/or involvement of deeper structures such as bone, tendon, or muscle. Slough or eschar may be present
Suspected deep tissue injury	Area of discoloured intact skin or blood-filled blister. Skin integrity maintained with suspected damage to deeper tissues
Unstageable	Full-thickness tissue loss in which actual depth of ulcer is obscured by slough and/or eschar in the wound bed

and localized swelling. Relevant characteristics to note are summarized in Box 45.3. The familiar four-stage grading system for pressure sores was devised by Shea in 1975.³⁰ This system was revised by the NPUAP in 2007 to include two new categories: suspected deep tissue injury and unstageable (see Table 45.1). The addition represents the recognition that pressure sores may develop from bottom to top rather than progressing through the classic four stages, resulting in rapid development of an advanced pressure sore. Comprehensive assessment and staging of the pressure sore is important in order to avoid underestimating severity and to determine what treatment may be required. This may necessitate debridement to ascertain depth of tissue loss. It is important to note that in the special situation of a pressure sore on the heel, the eschar layer is an important part of the wound-healing process and it is recommended that it not be removed.^{32,33} If osteomyelitis is suspected, appropriate imaging with scintigraphy or MRI is indicated.

Management

Multidisciplinary team

Management of the patient with a pressure sore should involve a multidisciplinary team to allow collaboration between reconstructive surgeons, rehabilitation medicine physicians, specialist wound care nurses, and members of allied health disciplines including occupational therapists, physiotherapists, dieticians and social workers if required.

Wound care

Cleansing the wound with an antiseptic solution and dressing with a non-adherent dressing (such as hydrocolloids, hydrogels, hydrofibres, foams, films, alginates and soft silicones) provide an optimum wound healing environment. There is little evidence to recommend a particular type of dressing or antiseptic over any other, though some trials have found a benefit for hydrocolloid dressings over saline gauze.^{34,35} Negative pressure wound therapy (NPWT) is frequently used in management of chronic sores including pressure sores. It has advantages in reducing frequency of dressing changes and may have a positive effect on wound healing.⁴ Constant assessment for signs of infection allows early diagnosis and treatment. Tissue obtained during debridement and any swabs taken of fluid exudate should be sent for microbiological examination to allow tailoring of antimicrobial agents to the pathogenic organism and its sensitivities.

Pain management

Patients with pressure sores frequently have numerous sources of pain. The pressure sore itself may directly cause pain, there may be chronic pain issues related to musculoskeletal abnormalities or neuropathy, and healthcare interventions may also cause pain. Wound debridement is a particular cause of breakthrough pain that may require additional short-acting analgesia. Strategies to reduce pain include regular repositioning, regular and sufficient analgesia, coordination of interventions such as dressing changes, debridement, and wound examination with analgesia administration, prescription of additional analgesia as required, use of non-adherent dressings (not gauze), and adjuvant analgesia for neuropathic pain.

Nutrition

It is important to ensure the patient with a pressure sore is adequately nourished and to give nutritional supplementation for wound healing. Enteral feeding by nasogastric tube may be necessary in patients unable to tolerate oral feeding. Mixed nutritional supplementation with protein, arginine, zinc and vitamin C has been demonstrated to be effective in some clinical trials.³⁶ Suggested daily intakes for patients with pressure sores are: energy: 30–35 kcal/kg, protein: 1.2–1.5 g/kg³⁷ as part of a high-protein supplement containing arginine, zinc and vitamin C.

Box 45.4 Initial surgical management

- Drainage of all collections
- Adequate debridement of necrotic, infected, and scarred tissue
- Excision of pseudobursa
- Removal of infected bone
- Haemostasis, drainage

Adjunctive treatments

Many experimental treatments have been trialled in the treatment of pressure sores. These include therapies such as electrical stimulation, pulsed electromagnetic field treatment, ultraviolet light C, ultrasound and hyperbaric oxygen. Currently there is little evidence to recommend their routine use in the management of pressure sores.³¹

Initial surgical management

Prior to attempting reconstruction, there are several surgical measures which may be employed to assist pressure sore healing (Box 45.4). Debridement of necrotic and infected tissue is an essential part of the care of any wound. Devitalized tissue provides a focus for infection, prevents healing by stimulating a persistent inflammatory response, and prevents wound contraction.³³ Pressure sores are commonly associated with collections of fluid surrounded by a wall of scar tissue, which are called pseudobursae. These and any other serous or infected collections should be excised or drained. Any osteomyelitic bone should be resected, though bone excision should be minimized if possible, as removal of bone results in alteration of pressure distribution patterns and the potential for development of new pressure sores in previously uninvolved areas. The wound may then either be closed if possible to do so without tension, with drain tubes left *in situ*, left open to heal by secondary intention or in preparation for a reconstructive procedure.

Reconstruction**Preoperative management**

Prior to embarking on surgical reconstruction of a defect resulting from a pressure sore, all of the above strategies should be instituted to minimize the need for surgery and to improve the chance of success of any reconstructive procedure. The physiological stress resulting from a prolonged operation such as is generally required for the reconstruction of a pressure sore would have a dire effect on any patient who is acutely unwell. As such the management of any medical conditions should be optimized and any nutritional deficiencies remedied preoperatively. Furthermore, careful patient selection is vital in order to ensure that surgical reconstruction is the appropriate course for the individual patient. Factors that may increase the risk of failure of a surgical reconstruction or recurrence of a pressure

Box 45.5 Indications and contraindications for reconstructive management of pressure sores**Indications**

- Stage 3/4 pressure sore
- Unlikely to/has not responded to more conservative management

Contraindications

- Poor overall prognosis
- Poor compliance
- Acutely unwell
- Malnourished/cachectic

sore postoperatively include any of the risk factors listed above (see Box 45.1) and poor compliance with previous treatment.

Patients in whom these risk factors are present should only be considered for reconstructive surgery if there is a clear plan in place to ameliorate or reduce the effect of the risk factor, as a poor outcome is otherwise highly likely. In addition, patients with a poor overall prognosis are generally unsuitable for reconstructive surgery for pressure sores (Box 45.5). The preoperative period is also a good time in which to educate patients and caregivers regarding the operation and strategies for prevention of further pressure sores.

Reconstructive ladder

The reconstructive ladder is a concept every plastic surgeon should be familiar with. It arranges options for surgical reconstruction in order of increasing complexity, from basic healing by secondary intention to the technically and physically demanding free vascularized tissue transfer. It is important to note that this does not necessarily mean that the simplest reconstructive option is the most appropriate for the individual patient. There are often situations where a more advanced procedure will have benefits over a simpler procedure. This is particularly true in the management of the patient with a pressure sore, in whom reconstructive options that do not provide tissue bulk, such as healing by secondary intention, primary closure and skin grafting, are likely to result in recurrence. For this reason myocutaneous or fasciocutaneous flaps are usually the treatment of choice, as they provide vascularized tissue bulk to fill dead space and allow wider distribution of pressure over bony prominences. Local or regional flaps are most commonly used, though free flaps may be a viable alternative where all other options have been exhausted. In general, procedures should be planned so as to preserve other options, as pressure sore recurrence is common and another flap may be required in future.

Reconstructive options by pressure sore site**Sacral**

The area overlying the sacrum and coccyx is very commonly affected by pressure sores, usually occurring in bedridden patients who remain in the supine position for prolonged

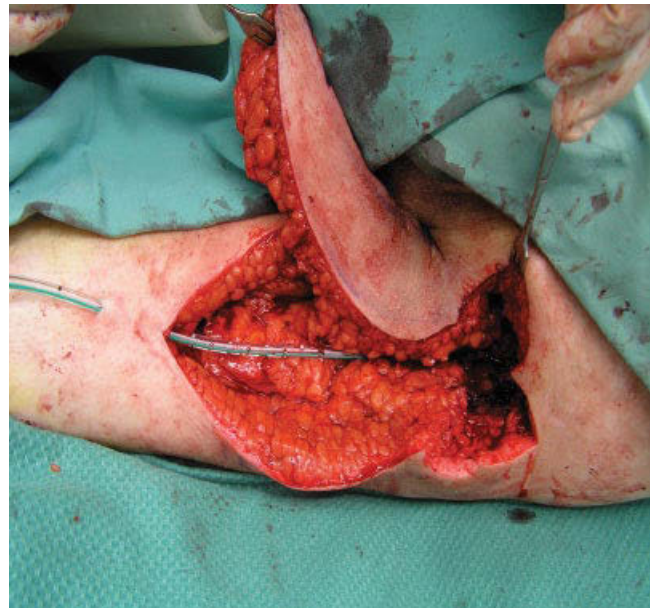
periods. Local myocutaneous flaps based on the gluteus maximus muscle, or fasciocutaneous flaps based on the overlying fascia, are versatile and effective methods for reconstructing these defects.^{38,39} Techniques include V-Y advancement flaps, rotation flaps and split flaps. Note that myocutaneous flaps based on the gluteus maximus muscle are not generally recommended for ambulatory patients due to the resultant functional deficit. Other techniques include the superior gluteal artery perforator flap and the transverse back flap. In particular, the perforator-sparing rotation technique allows re-rotation in patients likely to experience sore recurrence.⁴⁰

Ischial

Pressure sores overlying the ischial tuberosities are common in wheelchair-bound patients, such as patients with spinal cord injuries. Similarly techniques to those used for sacral pressure sores may be effective in the reconstruction of defects caused by ischial pressure sores, such as myocutaneous flaps based on the gluteus maximus muscle, or fasciocutaneous flaps based on inferior gluteal perforators. The posterior thigh flap (Figure 45.2) is another useful option, its advantages including a reliable blood supply, good long-term outcomes, no functional deficit, and the preservation of other options for repeat flap repair in cases of



(a)



(b)



(c)

Figure 45.2 Posterior thigh flap for reconstruction of an ischial pressure sore. (a) Preoperative markings. (b) Raising of flap based on medial perforator. (c) Flap sutured into defect, donor site closed primarily.

recurrence.^{41,42} It is a fasciocutaneous flap composed of posterior thigh skin and subcutaneous tissue, based on perforators from either the first and second perforating arteries of the profunda femoris or the inferior gluteal artery.⁴³ Other options include flaps based on the hamstring muscles, such as the V-Y advancement biceps femoris myocutaneous flap, or the gracilis flap.

Trochanteric

Patients spending a prolonged period in the lateral recumbent position are at risk of developing pressure sores overlying the greater trochanter of the femur. Common options for reconstruction of these defects are flaps based on the tensor fascia lata (TFL) muscle.⁴⁴ This flap is based on a branch of the lateral circumflex femoral artery and may be composed of muscle and skin or muscle alone.⁴⁵ It may also be used for reconstruction of ischial pressure sores, and is particularly useful in situations where both trochanteric and ischial sores are present. Other options include the anterolateral thigh flap, which may provide

additional tissue bulk where the size of the TFL may be insufficient. Another option is the groin flap, based on the superficial circumflex iliac artery, which may be useful if the TFL has already been used as a local or free flap for reconstruction of pressure sores elsewhere.

Heel and lateral malleolus

Pressure sores on the posterior aspect of the heel and overlying the lateral malleolus are common in neuropathic or insensate patients. They pose a challenge to the reconstructive surgeon due to the scarcity of local tissue for coverage. Options for reconstruction of a pressure sore on the heel or elsewhere in the foot include the lateral calcaneal and medial plantar artery flaps.^{46,47} The lateral supramalleolar flap (Figure 45.3), based on a distal perforator of the peroneal artery,⁴⁸ is a rotation and/or propeller flap which provides coverage of a lateral malleolar defect, while the donor region can be safely covered with a split-thickness skin graft.⁴⁹ For recurrent or refractory pressure sores,



(a)



(b)



(c)

Figure 45.3 Lateral supramalleolar perforator flap reconstruction of a lateral malleolar pressure sore. (a) Preoperative markings. (b) Flap raised on peroneal artery perforator. (c) Flap sutured into defect, donor site closed with split-thickness skin graft.

the distally based superficial sural artery flap is a versatile option that may be rotated to cover either of the malleoli or the heel.⁵⁰

Last resort

If the above strategies fail, or if they have been used in prior reconstructions, then the situation is a difficult one. The management of a patient suffering from recurrent pressure sores is complex, and requires the involvement of all members of the multidisciplinary team to evaluate the causes of recurrence. If all potential causes of recurrence have been addressed, then further reconstruction may be considered. Further procedures should be tailored to the patient, and may require some creativity. Free flaps from distant sites, or tubed pedicle flaps from the contralateral side are options, and choice of one over another is reserved for the surgeon's clinical judgement.

Postoperative care

Excellent postoperative care and follow-up following surgical reconstruction of a pressure sore is essential if complications and a poor outcome are to be avoided. Potential complications to be vigilant for include: sepsis, wound dehiscence (superficial and deep), haematoma, seroma, partial or total flap loss, and pressure sore recurrence.^{51,52} Good operative technique with low-tension skin closure and use of drains reduce the risks of haematoma, seroma and wound dehiscence. Rates of partial or total flap loss may be reduced by attentive nursing care, and by having a low threshold for returning to theatre to revise flaps that appear to be failing. Recurrence or the development of a new pressure sore is unfortunately very common. Following reconstruction of a pressure sore, it is crucial to continue prevention strategies for pressure sores. As stated above, in patients with recurrent or refractory pressure sores, the causes for recurrence should be addressed. Pressure sores provide a focus of necrotic tissue which acts as a nidus for bacterial growth, increasing the risk of postoperative wound infection. For this reason broad-spectrum antibiotics are frequently continued for extended periods. Other potential complications following pressure sore reconstruction are corollaries of immobility. Loss of muscle and bone mass result from prolonged bed rest, and immobilization of joints may result in the development of contractures. It is also important to be aware of the risk of deep venous thrombosis and pulmonary embolism, and staff should be especially vigilant for the signs of these conditions in the postoperative period.

A general protocol followed by the senior author is for surgical sutures and drains to be left *in situ* for 2–3 weeks to minimize the risk of complications such as seroma, haematoma and wound dehiscence, all of which may compromise flap viability. Appropriate broad-spectrum antibiotics, administered orally or intravenously, should be continued for up to six weeks, with a low threshold for referral to specialist infectious diseases physicians if there is uncertainty regarding choice of agent, especially if there is suspicion of osteomyelitis or if multiresistant

organisms are present. In the absence of major bleeding, prophylactic doses of anticoagulation should be administered from the first postoperative day for prophylaxis of deep venous thrombosis and embolic events.

The strategies outlined in the prevention and general management sections above should be continued to prevent recurrence or the development of a new pressure sore, including repositioning and use of pressure-relieving support surfaces. Minimizing shear and pressure on the operative site is especially important in the postoperative period. Protection of the flap pedicle should be a priority, and intensive monitoring of flap viability using clinical examination including bedside Doppler ultrasound by resident medical staff and specialist nurses is essential.

Once the immediate postoperative period is successfully navigated, the patient is likely to require a period of rehabilitation with graduated increase in pressure on the operative site. During this period any required devices, such as wheelchairs, should be evaluated and customized for the patient.

Conclusion

Pressure sores are a common problem in the inpatient population, particularly among patients with sensory impairment or reduced mobility. The development of a pressure sore has implications for patient length of stay and increased use of healthcare resources. Through a sound knowledge of the pathophysiology of pressure sores, and using the prevention strategies outlined in this chapter, occurrence of pressure sores and their attendant costs may be reduced. The patient with a pressure sore is best managed with a multidisciplinary team approach, incorporating physicians and allied health practitioners. Their general medical condition and nutrition should be optimized and any management of modifiable risk factors reviewed. Following initial surgical debridement and treatment of any infection, further reconstructive procedures may be considered if indicated. Careful patient selection is necessary prior to embarking on any advanced reconstructive procedure to avoid a poor outcome. Ideally, a local fascio- or myocutaneous flap should be chosen to provide well-vascularized tissue bulk. Following the procedure, attentive postoperative care will reduce the risk of complications and pressure sore recurrence.

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CHAPTER 46

Lower limb reconstruction

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Introduction

Open tibial fractures are severe limb-threatening injuries associated with considerable morbidity. They largely affect young men of working age and often occur as a result of motor vehicle accidents. Typically, high-energy transfer results in fragmentation and periosteal stripping at the fracture site. Recovery from these injuries is prolonged and the average time to fracture union is between 41 and 43 weeks.^{1–3} The rate of fracture non-union or delayed union following high-energy trauma is estimated to be 10–31%.^{4,5} Moreover, as the soft tissue envelope is breached, all open fractures are contaminated by microbes and susceptible to infection. Indeed, 8–13% of Gustilo IIIB fractures are complicated by deep infection, which may result in delayed amputation.⁶ A multicentre study conducted by the Lower Extremity Assessment Project group found that 8.6% of salvaged limbs were complicated by osteomyelitis and 31% developed non-union.⁵ Furthermore, between 20% and 50% of patients with open lower limb fractures were permanently disabled⁷ and almost 40% had not returned to work by 3.4 years.² One retrospective study found that despite a successful limb salvage rate of 93% in patients with open tibial fractures, only 28% returned to long-term employment, and no patient returned to work after 2 years of unemployment.⁸ Hence, these injuries place a significant health, social and economic burden on the individual and wider society.

Classification of open fractures

Injury characteristics, including the level of energy, fracture configuration, soft tissue disruption and wound contamination determine the final clinical outcome.⁹ Various classification systems of open fractures have been described. The most widely used is the Gustilo and Anderson classification,¹⁰ which is relatively simple but has poor interobserver reliability¹¹ and is best applied after wound debridement (excision) (Table 46.1). Other more comprehensive systems such as the AO classification

are more difficult to apply and tend to be used for research purposes. Extremity injury scoring systems such as the Mangled Extremity Severity Score (MESS)¹² were developed to identify patients who would benefit from primary amputation. The score is specific, but lacks sensitivity.¹³ The decision to amputate remains challenging and is best taken on detailed assessment of each patient in a multidisciplinary setting.

The combined orthoplastic approach

Trauma care pathways adopting a multidisciplinary and integrated orthoplastic approach have been shown to improve outcomes.^{14–18} Critical aspects include meticulous surgical planning and execution, microbiological management and rehabilitation tailored to the individual. Coordinated treatment carried out in a timely manner can reduce the mean union time for tibial diaphyseal fractures to 26 weeks.¹⁹

The development of major trauma centres with associated trauma networks in the USA and UK has facilitated the primary transfer of patients with these complex injuries to specialist centres. Hospitals that lack a team with requisite expertise should arrange for immediate referral to the nearest specialist centre.

Early assessment and initial management

The initial assessment and management of the vital systems follows Advanced Trauma and Life Support (ATLS) principles before attention is focused on the injured lower limb. Direct manual pressure or a tourniquet is used to control limb haemorrhage. A thorough vascular assessment is crucial to identify a devascularized limb or compartment syndrome. Absent pulses may be restored by realignment of the limb. Examination and documentation of the neurological status is essential. Although the instability of the fracture and pain often preclude assessment of movement and power, it is important to assess sensation, including in the territory of the deep peroneal nerve (web space

Table 46.1 Gustilo and Anderson classification of open fractures¹⁰

Type	Description
I	Low energy fracture with clean wound less than 1 cm
II	Wound more than 1 cm but without extensive soft tissue damage or major bone comminution
III	Open fracture with extensive soft tissue damage and high-energy fracture pattern
IIIA	Type III with adequate periosteal cover at fracture site and flap cover not required
IIIB	Type III with periosteal stripping at fracture site, flap cover required
IIIC	Type III with arterial injury requiring repair, irrespective of soft tissue injury

between the first and second digits). The wound is then examined, photographed and covered with sterile gauze moistened in saline and a semipermeable film to prevent dessication. No attempt should be made to lavage the wound as this simply ‘washes in’ the debris. The dressing should then be left undisturbed until the patient is examined in the operating room. Antibiotics are administered intravenously and tetanus prophylaxis should be given if necessary.

The neurovascular status must be reassessed after attempts at fracture reduction and splintage. Plain radiographs, showing orthogonal views of the fractured bones and including the joints above and below the fracture, should be obtained.

Antibiotic prophylaxis

Early antibiotic prophylaxis reduces infection in open fractures and should ideally be given within 3 h.²⁰ Although open fractures are contaminated during injury, the majority of deep infections are due to nosocomial organisms. Most patients are exposed to hospital organisms prior to definitive soft tissue coverage.²¹ The commonly cultured organisms from infected fractures are staphylococci (including methicillin-resistant strains), coliforms and pseudomonads.^{22,23} These findings led to the development of a two-phase antibiotic prophylaxis protocol,⁶ the first to cover bacteria encountered at the time of injury and the second for prophylaxis against hospital-acquired organisms.

Gustilo grade I open fractures should not receive antibiotics beyond 24–48 h. For more severe injuries, prophylaxis should be continued until definitive soft tissue closure or for a maximum of 72 h, depending on the timing of debridement. There is no evidence for prophylactic antibiotic use beyond definitive soft tissue closure.

Antibiotics should be tailored to the local microbiological profiles (Box 46.1). Coverage against gram-negative bacteria and *Clostridium* spp. in addition to gram-positive bacteria remains controversial and depends on the nature of the injury.²⁴ Agricultural injuries are often contaminated by staphylococci, enterococci and aerobic gram-negative bacilli,²⁵ whereas multi-drug-resistant acinetobacter has been found in military injuries.²⁶

Box 46.1 Antibiotic guidelines^a

- 1 Co-amoxiclav 1.2 g 8-hourly IV or a cephalosporin (e.g. cefuroxime) as soon as after the injury as possible and continued until debridement.
 - 2 Co-amoxiclav/cephalosporin and gentamicin 1.5 mg/kg at the time of debridement and co-amoxiclav/cephalosporin continued until definitive soft tissue closure, or for a maximum of 72 h, whichever is sooner.
 - 3 Gentamicin 1.5 mg/kg/h and either vancomycin 1 g or teicoplanin 800 mg on induction of anaesthesia.

^aPatients with penicillin allergy should receive clindamycin.

Wound debridement

Meticulous debridement of the traumatic wound is essential to prevent infection after open lower limb fractures but immediate surgical exploration is not indicated unless there is agricultural or aquatic contamination of the wound, compartment syndrome, a devascularized limb or a multiply injured patient. In the absence of these conditions, debridement should be performed as a combined orthoplastic procedure by experienced surgeons on the next scheduled operating list during normal working hours and 18 h of injury. There is a lack of evidence to support the oft-quoted ‘six hour debridement rule’.¹

The limb is initially washed with a soapy solution and a tourniquet applied but not inflated. The skin is prepared with an alcoholic chlorhexidine solution, avoiding contact with the open wound and pooling under the tourniquet.

We recommend wound extensions along accurately mapped fasciotomy lines used for compartment decompression (Figure 46.1). These extensions allow inspection of perforators for local fasciocutaneous flaps and provide access to the posterior tibial vessels. First, the subcutaneous borders of the tibia are marked. The posteromedial incision is marked 1.5 cm medial to the medial subcutaneous border of the tibia. The anterior incision is marked 2 cm lateral to the lateral subcutaneous border of the tibia. Care must be taken as a simple error can lead to inadvertent division of a perforator or an incision that is too posterior. Either would preclude the use of local fasciocutaneous flaps. To avoid undermining, the subcutaneous tissue and deep fascia are divided in the precise line of the cutaneous incision.

Once the zone of injury has been exposed by wound extensions, debridement of devitalized tissues can be undertaken while preserving local perforator vessels and neurovascular bundles. Soft tissue debridement under tourniquet control allows identification of neurovascular bundles, which may be displaced and permits accurate examination of tissues by avoiding blood-staining. The tissues are assessed systematically from superficial to deep (skin, fat, muscle, bone) and from the periphery to the centre of the wound. Non-viable skin, fat, muscle and bone are excised. Bone fragments that are not viable

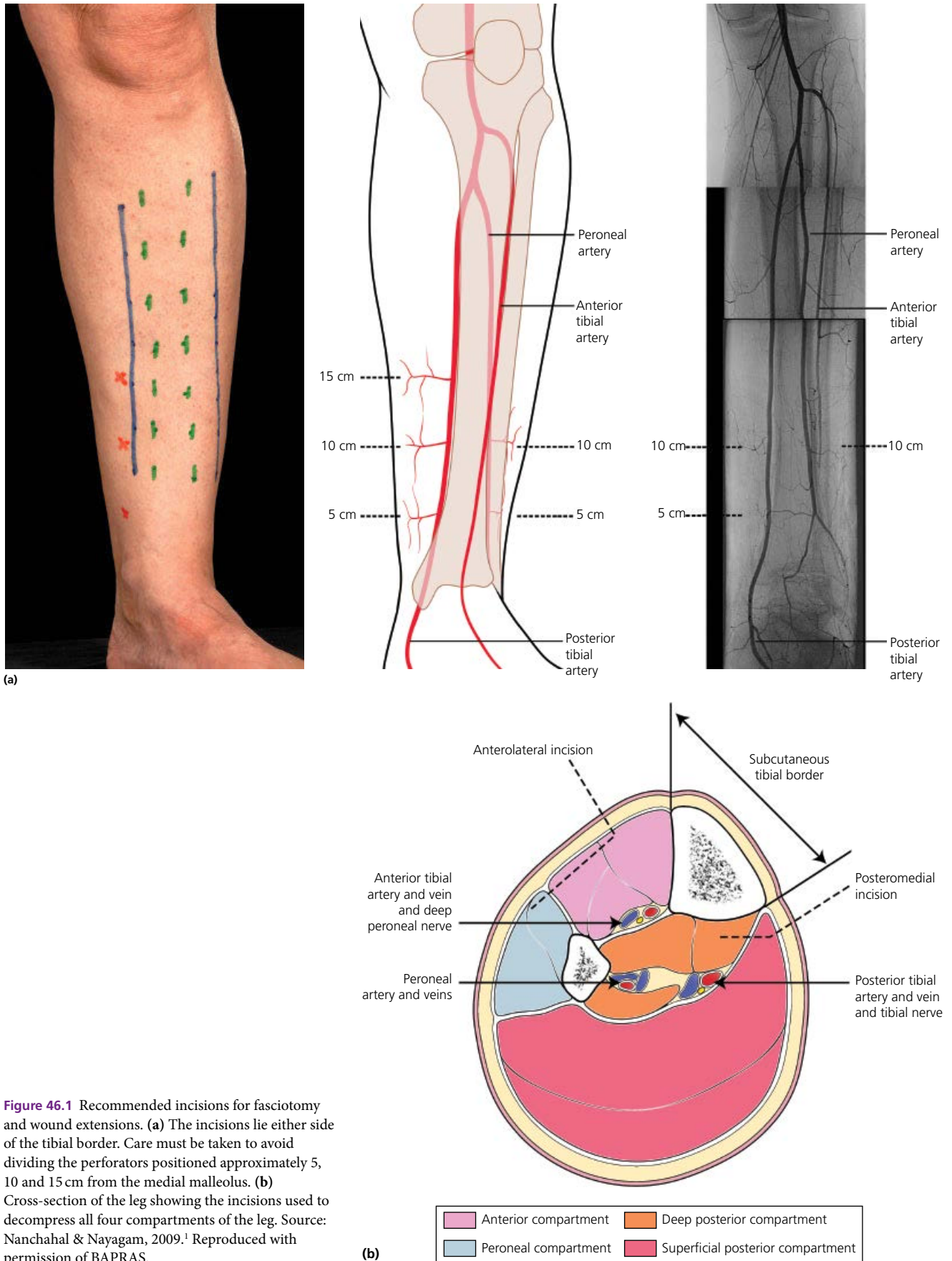


Figure 46.1 Recommended incisions for fasciotomy and wound extensions. **(a)** The incisions lie either side of the tibial border. Care must be taken to avoid dividing the perforators positioned approximately 5, 10 and 15 cm from the medial malleolus. **(b)** Cross-section of the leg showing the incisions used to decompress all four compartments of the leg. Source: Nanchahal & Nayagam, 2009.¹ Reproduced with permission of BAPRAS.

and fail the 'tug' test are removed. Careful attention is paid to the medullary cavity and the soft tissues posterior to the tibia, which often harbour devitalized bone fragments.

Microbiological tissue sampling after debridement in patients with a delayed presentation or after transfer from another centre is recommended. Deep bone and muscle specimens are most relevant (3–5 samples collected with sterile instruments), ideally after antibiotics have been discontinued for 24 h. Tissue sampling in patients presenting following the primary injury has little merit as positive cultures do not predict subsequent deep infection.⁶ As mentioned above, most deep-tissue infections post fracture are due to nosocomial organisms.^{22,23}

The wound is then lavaged with saline, but high pressure must be avoided as this drives devitalized tissue and bacteria deeper into the tissues²⁷ and predisposes to non-union.²⁸ After debridement the injury is classified and the combined ortho-plastic team plans definitive reconstruction.

Degloving

Degloving injuries are caused by shearing forces, which separate the skin and subcutaneous tissues from the underlying deep fascia. Four patterns of injury are recognized:²⁹

- localized abrasion/avulsion with limited adjacent degloving;
- non-circumferential single plane degloving;
- circumferential single plane degloving; and
- multiplanar degloving.

The full extent of degloving can be difficult to assess and subcutaneous fat necrosis is often more extensive than skin necrosis. Fixed staining of the skin or subcutaneous vein thrombosis³⁰ are indicative of necrosis. The most reliable sign of adequate debridement is bleeding from the cut skin edge. Skin that has not been directly traumatized but has lost its blood supply can be used as a source of split-thickness skin graft. Circumferentially degloved skin never survives. Multiplanar degloving is particularly challenging and requires meticulous systematic excision of all non-viable muscle, with a second look usually required 24–48 h later.

Temporary wound dressings

If single-stage skeletal and soft tissue reconstruction is not undertaken, a dressing is applied until definitive surgery is performed. Negative pressure wound therapy (NPWT) has revolutionized the management of heavily exuding wounds. It provides a sealed wound environment limiting bacterial ingress and wound desiccation and simplifying nursing care. A randomized trial of NPWT in open fractures showed an overall deep infection rate of 5% for NPWT compared to 28% for gauze alone.³¹ Simple gauze dressings fail to control the wound exudate and allow rapid microbial colonization. Soaking the gauze in anti-septic solutions, such as povidone iodine, does not offer any benefit as the latter is rapidly inactivated by serum.³²

Although antibiotic cement bead pouches have been shown to reduce deep infection rates compared to systemic antibiotics alone,^{33–36} the use of antibiotic pouches, even for segmental bone defects, has been superseded by NPWT. A combination of antibiotic cement with NPWT can be used without adversely affecting local antibiotic levels. An experimental study of antibiotic beads with or without NPWT found comparable levels of antibiotics in the periosteal tissues and the drain fluid in both groups.³⁷ The choice of wound filler and interface material is based on the wound characteristics. Gauze is useful for complex three-dimensional cavities, whereas relatively simple wounds can be managed using foam. An interface dressing reduces ingrowth of granulation tissue into the filler and facilitates dressing change. Repeated dressing changes should be avoided to reduce the risk of further bacterial contamination.

Surgeons must be aware of the limitations of NPWT in open fracture care. NPWT does not maintain a sterile environment. A retrospective comparison of NPWT with gauze found the total bacterial load was comparable between the groups. NPWT was associated with a decrease in pseudomonads accompanied by an increase in *Staphylococcus aureus*.³⁸ Experimental studies have also found a decrease in *Pseudomonas* with NPWT but no change in the *Staphylococcus aureus*.³⁹

The importance of NPWT as a temporary dressing is emphasized by the findings of deep infection rates of 0.3–12% in Gustilo IIIB fractures if NPWT was applied for less than 7 days compared to 31–57% with NPWT beyond 7 days.^{40,41} Another study reported deep infection rates of 4% with NPWT for 3 days or less compared to 21% if applied for longer than 7 days.⁴²

We do not advocate extended NPWT in order to reduce the need for flaps by encouraging the development of granulation tissue, which can be skin grafted.^{40,43–45} Careful analysis of papers supporting extended NPWT reveals high infection and non-union rates together with increased amputation rates.

Principles of skeletal fixation

Prompt reduction and stabilization of open fractures reduces further soft tissue injury by minimizing motion at the fracture site and by relieving pressure on the soft tissues from fracture fragments. Restoration of limb alignment also improves blood and lymph flow at the injured area. Fixation aids deadspace management by restoring soft tissue tension and provides a stable platform for subsequent soft tissue reconstruction. Effective fracture stability also decreases pain, enables patient mobilization and facilitates subsequent investigations (e.g. angiography).

The method of fixation remains controversial. Systematic reviews of open fracture fixation have been undermined by the inclusion of historical data relating to obsolete, unstable fixation systems which have biased the conclusions.⁴⁶ The choice of skeletal stabilization is largely determined by the injury characteristics, with internal fixation used for less severe injuries and external fixation for more complex fractures.

Planning fixation

Plastic surgeons must be intimately involved in the fixation planning process because of the potential impact of the fixation on both soft tissue reconstruction and the final outcome. Poor external fixation design may impede access, leading to early fixation revision. Early internal fixation in the presence of complex soft tissue defects, requiring staged reconstruction, may result in exposed metal work. This is a risk for biofilm formation and later deep infection.⁴⁷

Temporary external fixation

Temporary or bridging external fixation (BEF) is a powerful and flexible method for the initial stabilization of severe limb injuries and should be considered in the following situations: extensive soft tissue injury, significant bone loss, wound contamination, metaphyseal fractures and for rapid stabilization of severe injuries in a physiologically unstable patient. If there is doubt regarding tissue viability at the end of the initial debridement, a BEF frame should be applied until conditions become favourable for definitive fixation.

Careful half pin insertion and optimum frame design are the keys to successful BEF. The number and configuration of the half pins and the width and the number of connecting longitudinal bars determine frame stability.

We recommend marking all definitive reconstruction incisions and access zones on the limb before pin placement. Half pins are ideally located outside the zone of injury and within safe corridors whenever possible.⁴⁸ Pin insertion involves pre-drilling with water-cooled drills and tissue-friendly techniques. Modern external fixation pin clamps are flexible and half pins do not need to be precisely aligned to reduce the fracture. A minimum of two anterior half pins per limb segment with maximum pin spread is recommended. A simple four-pin

unilateral fixator along the anterior border of the tibia will resist anterior and posterior forces. An additional 2–4 pins placed at an angle to the anterior pins and connected by short rods will significantly increase stability (delta configuration) but may hamper access for soft tissue reconstruction. Fracture reduction is achieved by manipulation before tightening the clamps and bone-on-bone contact should be obtained whenever possible to increase the stability of the final construct.

Joint spanning is indicated in proximal and distal tibial fractures. A simple anterior BEF utilizing two half pins in the distal femur and two half pins in the shaft of the tibia can be used to maintain stability across the knee joint and the proximal tibial fractures. A triangular-type construct can be used for stabilizing distal tibial fractures (Figure 46.2a). Smaller half pins can be placed into the metatarsal or tarsal bones and connected to the tibial half pins by appropriately positioned bars and clamps. The addition of an elevation frame to a distal tibial external fixator ensures limb elevation and limits shear forces and direct pressure on fragile soft tissues⁴⁹ (Figure 46.2b).

Careful insertion techniques and adequate frame stability will limit pin site infection and loosening, which are potential problems with all external fixation. A recent Cochrane review did not reach any definitive recommendations regarding pin site care.⁵⁰ Pins placed through a muscular compartment such as the anterior compartment are at risk of pin site infection, because of the adjacent mobile soft tissues. We recommend rolling gauze soaked in alcoholic chlorhexidine around the pin between the bar and the skin to produce gentle axial compression on the local soft tissue.

Conversion of BEF to internal fixation can be considered provided it is within 72 h of the index procedure, the pin sites are clean and definitive soft tissue cover will be accomplished immediately after fixation. Studies have shown that the longer



(a)



(b)

Figure 46.2 Bridging external fixation. (a) Open distal tibial injury stabilized by cross-ankle bridging external fixation (BEF). (b) An elevation frame applied in a case of severe open foot trauma following anterolateral thigh flap reconstruction. The elevation frame prevents inadvertent pressure over the flap pedicle, protects skin grafts in the initial period and reduces extremity oedema.



(a)



(c)



(d)



(b)

Figure 46.3 A 38-year-old female patient with an isolated open mid-shaft tibial fracture, suitable for single-stage reconstruction ('fix and flap'). (a) Small, well-circumscribed medial soft tissue defect with minimal degloving. (b) The initial plain radiograph shows a non-comminuted tibial shaft fracture suitable for intramedullary nail stabilization. (c) The wound was debrided and a distally based fasciocutaneous flap marked on limb before a nail was inserted via a suprapatellar approach while the patient remained supine (d). The distally based fasciocutaneous flap was raised based on the perforator located 15 cm from the tip of the medial malleolus (e) and inset. The donor site was covered with a meshed split-thickness skin graft (f). The plain radiograph at three months confirms fracture union (g).

the external fixator is in place, the greater the risk of infection after intramedullary nailing (IMN) because of contamination of the medullary canal by external fixator pins.⁵¹ Overdrilling and lavage of all pin sites immediately before internal fixation may minimize subsequent infection. The presence of frankly infected pin sites precludes internal fixation.

Internal fixation

Definitive internal fixation and soft tissue cover may be considered at the index procedure ('fix and flap') only if the injury characteristics are favourable (Figure 46.3). Meticulous aseptic techniques similar to those used during joint replacement are paramount in order to minimize infection risk. Surgeons should



(e)



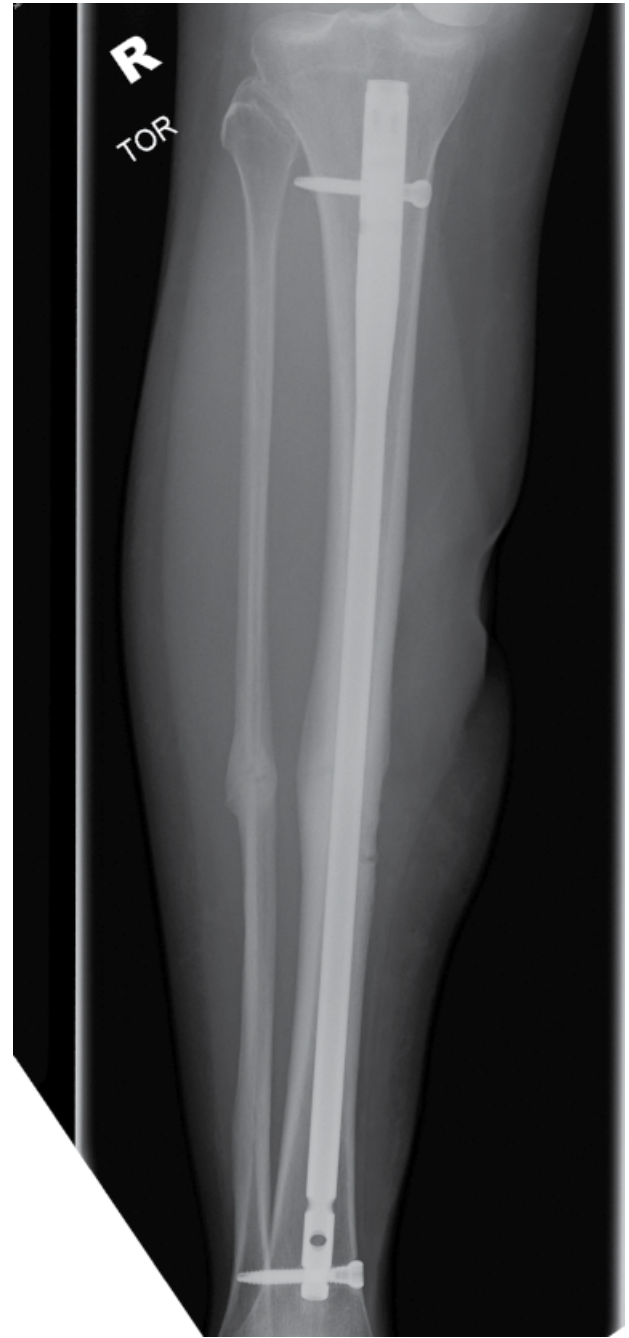
(f)

Figure 46.3 (Continued)

de-gown and re-prepare the injured limb after completing debridement and the internal fixation instrumentation and implants are only opened immediately prior to their use.

Metaphyseal fractures with intra-articular extensions may be stabilized with plates utilizing biological fixation methods, which avoid further soft tissue damage by stripping. Alternatively, circular external fixation (CEF) can be considered for metaphyseal and periarticular open fractures.

IMN is ideally suited for open shaft fractures with minimal contamination and predictable soft tissue defects.⁵² We



(g)

advocate the recently described supratibial approach for IMN of open tibial shaft fractures, which offers a number of advantages⁵³ (Figure 46.3). The patient can remain in the supine position throughout the surgical procedures and repositioning is minimized. The injured limb is securely held in a reduced and aligned position by an assistant throughout the nailing procedure allowing local flaps to be mobilized prior to fracture stabilization and held with holding sutures. The flap can then be inset at the end of the IMN procedure.

Definitive external fixation

Definitive external fixation principally involves either a monotube unilateral fixator or CEF and both are used in open fractures with satisfactory outcomes.^{19,54} The main advantages of definitive external fixation are the ability to modulate the fracture site and a reduced risk of fracture infection.

Unlike the smaller unilateral fixators used for BEF, *monotube unilateral fixators* (Orthofix®-type) have a large-diameter connecting body, which increases the rigidity of the fixator.⁵⁵

These devices attach to bone by clusters of half pins at each end of the monotube body, which limits the flexibility of pin placement but avoids the problems of soft tissue transfixation caused by wires. Monotube fixators are suitable for mid-shaft fractures and can also be used in cases with bone loss.

Circular external fixators are extremely versatile and can be used to stabilize both mid-shaft and metaphyseal fractures (Figures 46.4, 46.5 and 46.6). The frame design can be customized to the specific injury characteristics and easily modified to

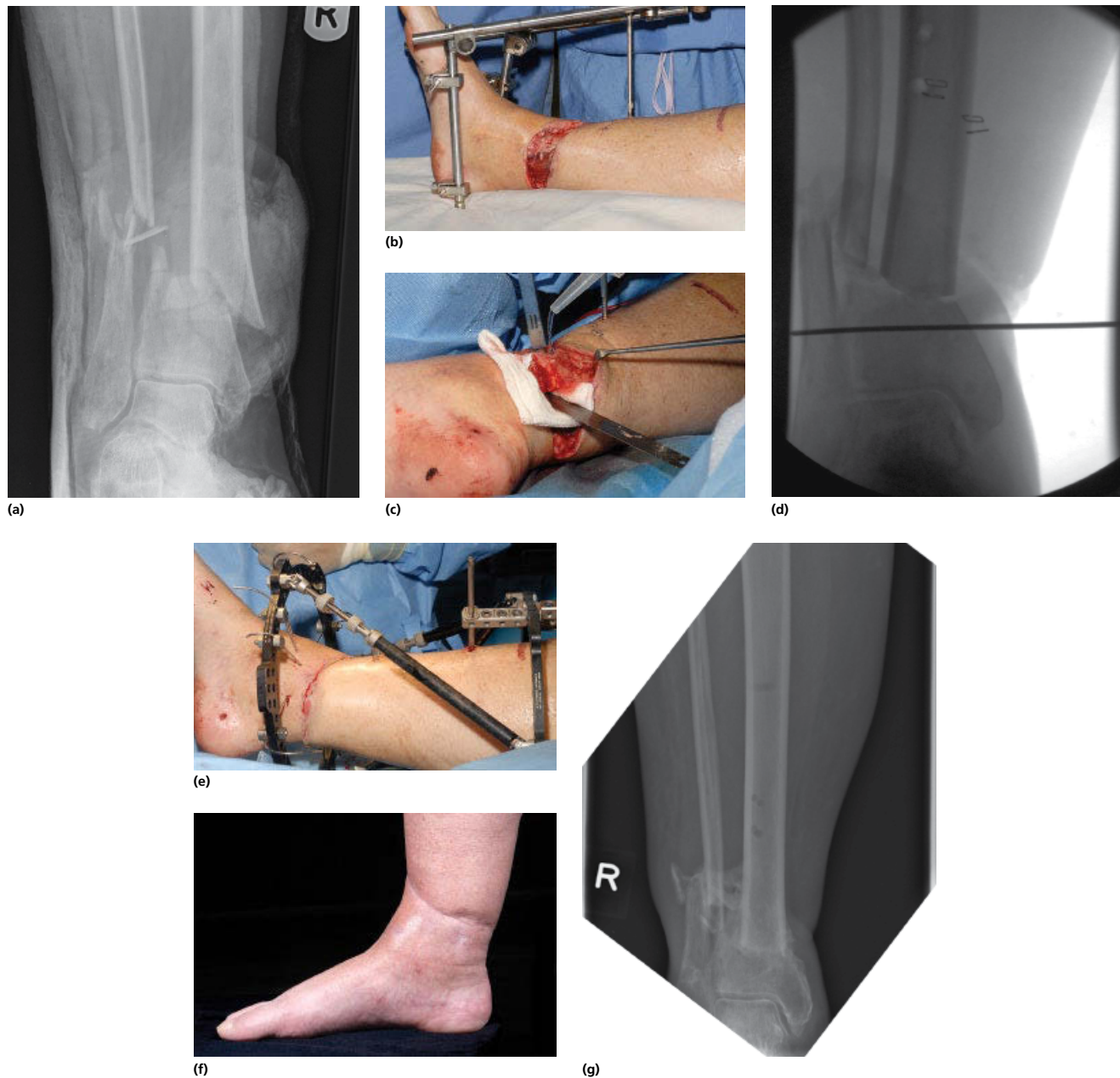


Figure 46.4 Acute shortening of distal tibial fracture in an 84-year-old female. (a) The initial radiograph showing a displaced comminuted fracture of the distal tibia and (b) the transverse wound extending posteriorly to the medial fascial perforating vessels, precluding the use of local fasciocutaneous flaps. (c) Bone resection using a water-cooled oscillating saw. (d) Intraoperative radiographs showing acute reduction with 'male-female' fashioning of fracture ends. (e) Acute tension-free wound closure after shortening. (f) The final clinical appearance and (g) final radiograph showing fracture union.

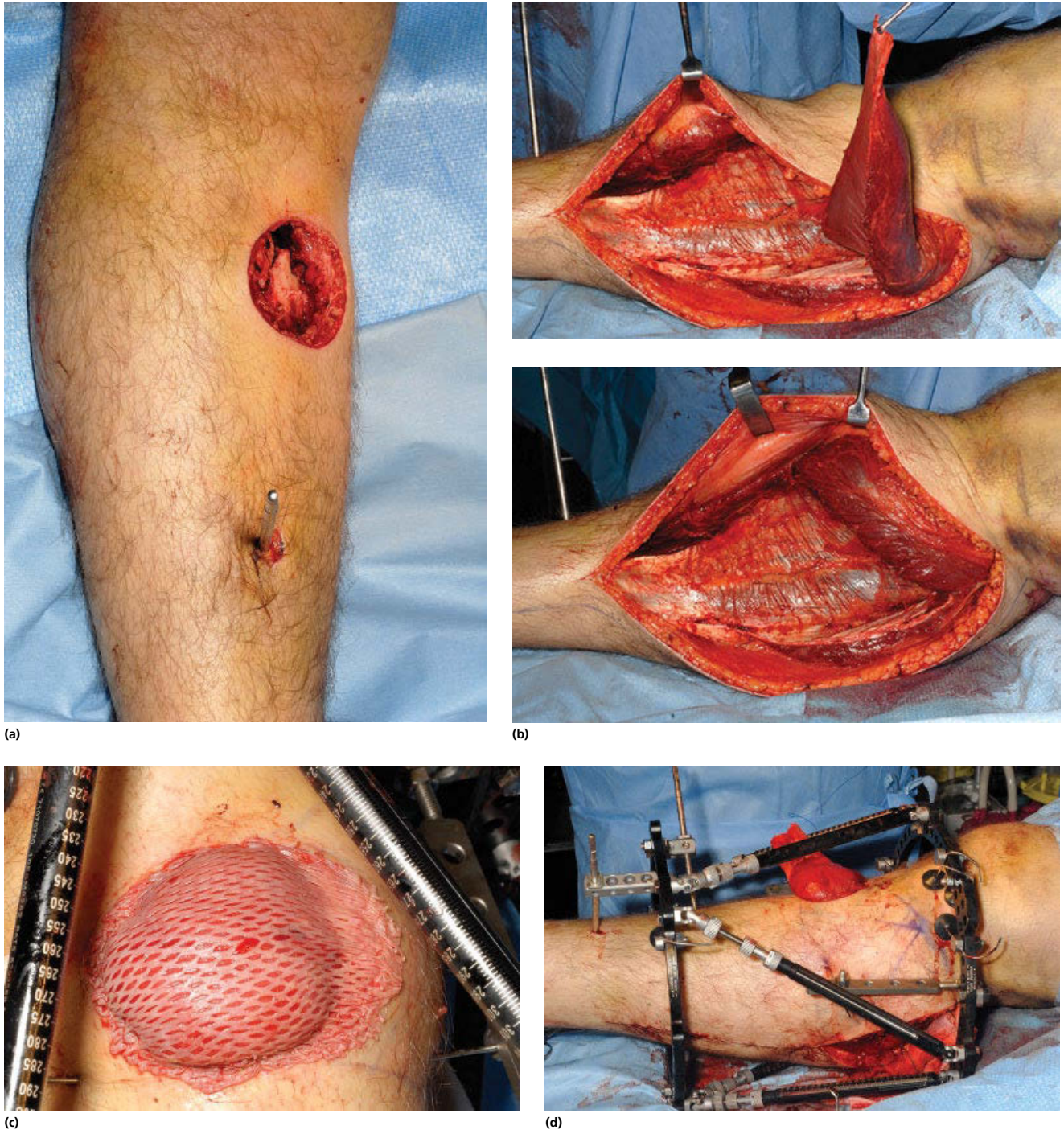


Figure 46.5 Gastrocnemius local muscle flap. (a) A proximal open tibial fracture with a small anteromedial soft tissue defect. Half pins used for the preliminary bridging external fixation (BEF) were retained for the circular external fixation (CEF). (b) The medial head of gastrocnemius, with a portion of Achilles tendon, was raised via a posterior incision. The muscle was tunneled beneath intact medial tissues and (c) covered with a split-thickness skin graft. (d) The appearance following stabilization with a circular external fixator (Taylor Spatial Frame). Case courtesy of Mr Jon Simmons FRCS(Plast).

allow soft tissue access. CEF systems comprise varying sized rings or half rings connected by longitudinal rods to form a cylinder and fixed to the skeleton by half pins (in the shaft) and tensioned smooth wires (in the metaphysis). Wires with

spherical swellings ('olives') can be used to increase the construct stability or for fixation of periarticular fragments. CEF can be used to correct limb deformity or fracture reduction in an outpatient setting. Stable CEF allows full joint and patient

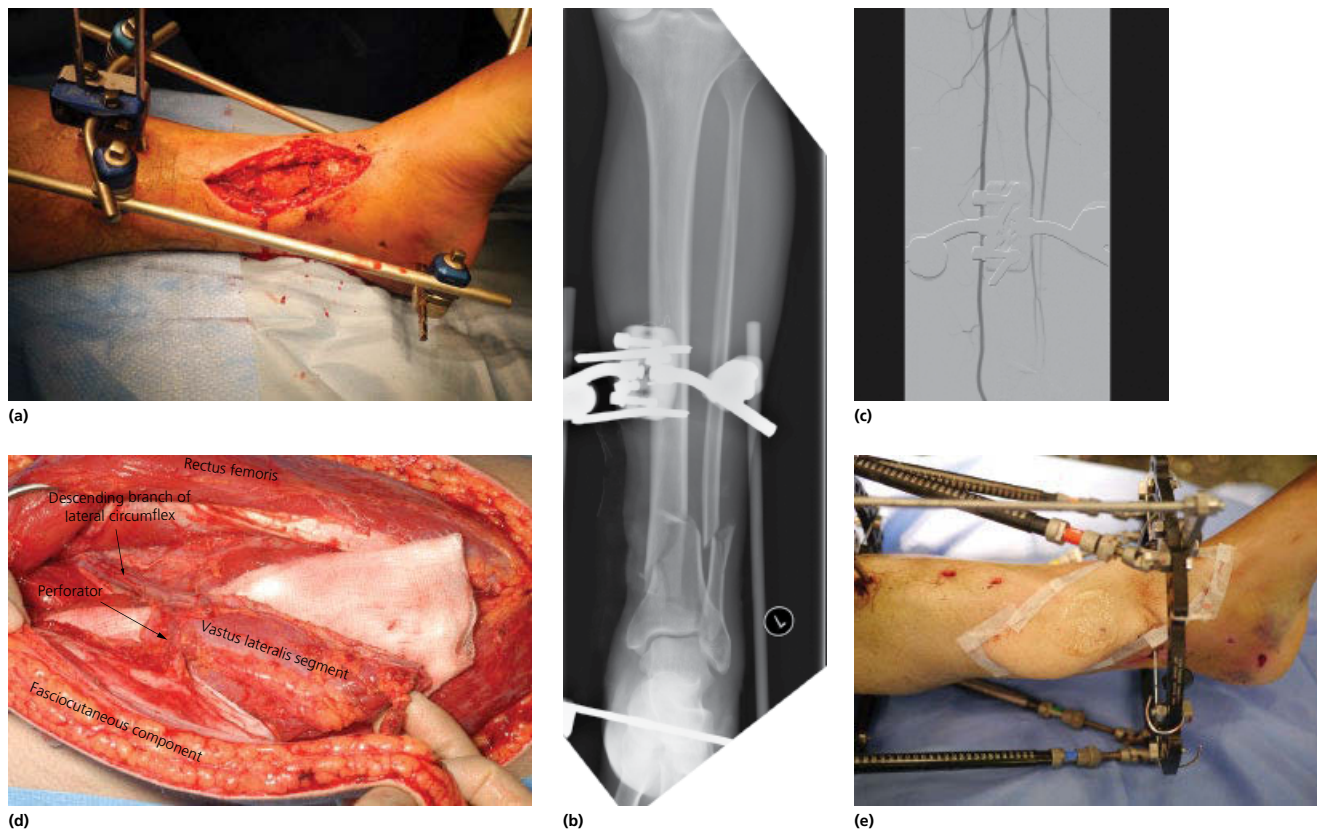


Figure 46.6 Free anterolateral thigh (ALT) fasciocutaneous flap. (a) Open distal tibial fracture with longitudinal soft tissue defect. (b) Radiograph following BEF showing high-energy fracture pattern. (c) Preoperative angiogram revealing no flow in the anterior tibial and peroneal arteries at the level of the fracture (single vessel leg). (d) Intraoperative photograph of free ALT flap that includes a segment of vastus lateralis. (e) Clinical appearance following coverage with ALT and fracture stabilization with a circular external fixator (Taylor Spatial Frame). Sources: (a, b, c, e) Mr Jon Simmons, FRCS(Plast). Reproduced with permission. (d) Chan et al., 2012.⁷¹ Reproduced with permission of Lippincott, Williams & Wilkins.

mobilization with early active weight bearing. The rings also offer protection for skin grafts during maturation.

Recent modifications of CEF include the hexapod fixator such as the Taylor Spatial Frame™ (Smith and Nephew Richards, Memphis, TN, USA), which has the advantages of ease of application and very accurate, computer-driven fracture reduction. The hexapod frames have simplified many of the demanding techniques associated with standard Ilizarov circular frames and they can be easily mounted on the half pins of the initial BEF at the time of the definitive fixation. In order to minimize the number of operations, we recommend application of an incomplete but stable CEF frame at the time of the initial debridement, which allows access for soft tissue reconstruction. The definitive CEF is then completed at the end of the soft tissue coverage.

Bone loss

The management of bone loss depends on the size and site of the defect. Partial posterior defects may spontaneously heal because of the local vascularized muscle bed. Anteriorly based partial defects or complete defects under 2.5 cm will require open

grafting at six weeks. Exchange nailing is a useful technique for healing of small defects around a nail, which involves removing the original nail, reaming the canal and inserting a larger nail. Alternatively, the fracture site can be manipulated by an external fixator to encourage healing (compression–distraction).

The management of larger bone defects remains controversial. Non-vascularized options include the Masquelet technique. The original bone defect is plugged with a bone cement spacer, which facilitates the formation of an induction membrane. The cement plug is removed at around six weeks and bone graft is packed into the membrane.

Disadvantages of bone grafting large defects include a lack of available autograft, pain from the donor sites and inconsistent bone formation. We favour a combined orthoplastic approach to bone grafting beneath a flap. The patient is placed in the lateral position to facilitate a simultaneous approach to the posterior iliac spine to harvest bone graft while the flap is being raised.

Vascularized options for large defects include free fibular flaps. However, free fibular flaps are technically demanding and associated with problems including mismatch with the tibial fracture ends, prolonged partial weight bearing and late fracture

of the fibular flap. The use of a free fibular flap in the presence of a large soft tissue defect poses additional challenges.

We prefer the Ilizarov method for the management of major bone defects.¹⁹ A low-energy fracture (corticotomy) is created in the metaphyseal bone adjacent to the fracture gap and the fracture surfaces are slowly distracted after a latency period of around a week. Reliable bone can be produced by this method (distraction osteogenesis). Bone transport describes the method whereby the moving segment is brought down to close the gap created by the original defect until the bone ends meet or dock. Additional surgery may be required to bring about union at the docking site. Alternatively, the gap at the original bone defect can be closed either acutely or gradually by shortening and limb length is gradually restored by distraction at the corticotomy site.

Acute shortening of 2–3 cm is particularly useful for open fractures with transverse soft tissue defects in elderly patients and may preclude the need for additional complex plastic surgery and hasten healing times (Figure 46.4). A shoe raise easily compensates for the resultant shortening.

The Ilizarov method is not without complications, and pin site problems and impaired rehabilitation due to pain are common. Bone formation may be delayed resulting in prolonged frame times. Later problems include analgesic addiction and fracture or deformity of the regenerate bone.

Soft tissue reconstruction

Timing

Early definitive soft tissue coverage of open fractures is associated with reduced free flap failure and deep infection rates.⁵⁶ Delays due to patient preparation, including investigations such as angiography need to be balanced against technical demands as the perivascular soft tissues become increasingly oedematous, friable and fibrotic with time. Current guidelines advocate definitive coverage ideally within 72 h^{1,42} and no later than 7 days after the injury.^{40,41}

Type of soft tissue coverage

The surgeon must consider the size and location of the defect, availability of flaps and donor site morbidity when planning soft tissue coverage. In addition, there is increasing evidence to support the potential biological role that soft tissue flaps may play in the fracture repair process.

Local flaps

In general, local flaps are best suited for small defects with a limited zone of injury. The flap chosen should lie outside the zone of injury to prevent complications such as partial or complete necrosis.

Local muscle flaps

There are limited local muscles available for coverage. The medial or lateral heads of the gastrocnemius provide versatile coverage around the knee but do not reach as far proximally as

the proximal pole of the patella (Figure 46.5). The dominant proximal blood supply with a relatively clear separation between the two heads allows for easy dissection and means that the flap is extremely robust and reliable. It is most easily harvested through a posterior paramedian incision, taking care not to inadvertently divide the sural nerve. The muscle can be mobilized proximally as far as its origin but we would recommend not detaching the origin to avoid undue tension on the pedicle. The flap is then usually tunnelled to cover anterior defects and it is important to ensure that the tunnel is sufficiently capacious to allow for postoperative muscle swelling. Harvesting a small cuff of the Achilles tendon insertion facilitates inset of the flap by avoiding cut out of sutures through the muscle fibres. Coverage of the muscle with split-thickness skin graft completes the procedure. Harvest of a single head of the gastrocnemius is associated with very little functional loss and the contour of the reconstructed site improves as the muscle atrophies through disuse.

A tibialis anterior muscle turnover flap can cover long narrow defects over the tibial crest. The anatomical arrangement of the muscle is unusual in that the blood supply splits around a central tendon, which permits unfolding of the muscle like opening a book. This flap has limited application as it only covers defects up to about a centimeter wide.

The soleus muscle flap has been advocated for coverage of middle third tibial defects. Unlike the gastrocnemius, the soleus muscle has proximal and distal pedicles as well as multiple nutrient vessels entering the muscle along its length. Mobilization of the muscle by necessity involves division of the proximal or distal primary pedicle as well as some of the secondary vessels. Together with the fact that the muscle lies close to the posterior aspect of the tibia and hence is injured when the bone fractures are driven backwards, it is in the authors' view a precarious flap prone to partial or complete loss. Furthermore, the rich venous plexus within the muscle makes it an important pump for venous return.

Local fasciocutaneous flaps

An appreciation of the lower limb angiosomes⁵⁷ combined with accurate mapping of perforating vessels⁵⁸ has enabled the reliable use of local fasciocutaneous flaps. On the medial aspect of the tibia just distal to the knee are multiple perforators, including those traversing the muscle and via intermuscular septae. Proximally based flaps extending as far distally as the perforator usually situated 15 cm from the tip of the medial malleolus can be reliably raised on these perforating vessels. The reliability of the flap is enhanced by inclusion of the saphenous vein and the accompanying artery, which runs approximately 1 cm anterior to the vein. The anterior border of the flap runs 1.5 cm posterior and medial to the medial subcutaneous border of the tibia, coinciding with the medial fasciotomy incision. The posterior margin can extend to the posterior midline, taking care to avoid inadvertent injury to the sural nerve. The deep fascia is included within the flap, thereby protecting the delicate vascular plexus

that lies just superficial to the fascia. It is essential to preserve the filmy vascularized tissue overlying the proximal part of the tendoachilles to ensure an adequately vascularized bed for the split skin graft used to reconstruct the flap donor site.

Distally based fasciocutaneous flaps are based on relatively constant septocutaneous perforators that arise from the posterior tibial artery and venae comitans 5, 10 and 15 cm from the tip of the medial malleolus (see Figure 46.3e). The anterior border of the flap is based on the medial fasciotomy incision, 1.5 cm behind the medial subcutaneous border of the tibia. The integrity of the perforator must be confirmed before raising the flap and the flap is planned in reverse, ensuring that the flap territory lies outside the zone of injury. By definition, extensive degloving precludes the use of these local flaps due to avulsion of the perforators. The 10 or 15 cm perforator is usually dominant and inspection of each allows planning to ensure flap survival. The 5 cm perforator is almost never adequate as the basis of a flap. It is important to either exclude the long saphenous vein from the anterior border of the flap or, if this is not possible, to ligate the vein at the base of the flap to prevent venous congestion of the flap as the venae comitans of the perforator struggle to cope with the venous drainage from the medial side of the foot. Again the deep fascia is included in the flap to protect the delicate pre-fascial vascular plexus. Islanding of the flaps allows turning through a greater angle and a neater inset. However, in a series of 61 patients with open fractures treated with islanded distally based flaps, there was a 20% complication rate in patients with Gustilo IIIB injuries and an overall flap failure rate of 7.6%.⁵⁹ It is our preference not to island the distally based flaps and to allow the 'dog-ear' to settle spontaneously over a few months.

Although fasciocutaneous flaps provide coverage of 'like with like' and harvest does not result in any functional deficit,⁶⁰ the donor defect can be aesthetically unsatisfactory.⁶¹ In our practice, local fasciocutaneous flaps are most commonly used in male patients with small, localized defects who undergo simultaneous definitive skeletal fixation and flap reconstruction.

The sural artery flap, based on the sural artery and venae comitans that run with the sural nerve has recently gained popularity. However, we do not recommend this flap in patients with open tibial fractures. The supplying perforator arises from the peroneal artery, which is often injured when the fibula fractures. Although the flap has been shown to be useful for chronic disorders, a review of 70 flaps⁶² found that up to 36% developed necrosis, particularly in the presence of comorbidities such as diabetes, venous insufficiency and peripheral arterial disease. This is the subgroup that is erroneously considered by some surgeons to be unsuitable for free flaps. Careful optimization of the patient and techniques⁶³ can result in free flap success rates in open fracture equal to those for elective reconstructive surgery.

Free flaps

Free tissue transfer has revolutionized the management of open fractures. Almost any defect can now be reconstructed with a variety of tissues, including fasciocutaneous, muscle,

bone and chimeric flaps. Donor site morbidity as well as defect size and configuration influence flap selection. For example, the rectus abdominus muscle has now fallen out of favour because of the subsequent anterior abdominal wall weakness. In addition, the biological contribution of the constituent tissues should also be taken into consideration during flap selection.

Muscle flaps easily fill complex defects and in our practice the latissimus dorsi is used for covering large areas, whereas the gracilis, with minimal donor site morbidity, is ideally suited to small defects. Although the muscle flap is bulky initially due to oedema, this can be controlled with a pressure garment once the overlying skin graft is stable and any external fixator has been removed. The denervated muscle soon atrophies and does not require thinning. The disadvantage of muscle flaps is that the overlying skin graft may be susceptible to minor trauma and the flap can be difficult to raise and re-inset if bone grafting is required for non-unions.

The anterolateral thigh flap is the commonest fasciocutaneous free flap for open tibial coverage (Figure 46.6).^{64,65} The pedicle is long (8–16 cm × 2 mm) and use of the contralateral thigh allows a two-team approach and the recipient vessels of the injured limb can be dissected under tourniquet control. Very large flaps can be harvested by including multiple perforators and a segment of vastus lateralis can be included in chimeric flaps (Figure 46.6d). Although the bulkiness of the flap is a disadvantage in obese individuals, this can be subsequently addressed by liposuction.⁶⁶ We prefer secondary liposuction as edge necrosis may result from primary thinning of large flaps.

When choosing the recipient vessels for anastomoses, it is important to select an area outside the zone of injury. Our preference is to site the anastomoses proximal to the defect. Recipient vessels, in particular the venae comitans, can be injured at the level of the fracture and intimal injuries may not be apparent on inspection of the external wall. The posterior tibial vessels are less likely to have been injured during the initial trauma and are more reliable.⁶⁷ The anterior tibial vessels run deep just proximal to the ankle joint and the venae comitans can be of small calibre. The posterior tibial vessels are easily identified distally as they lie just deep to the fascia covering the flexor hallucis longus. Proximally the vessels lie deep to soleus. The artery is always anastomosed end-to-side to preserve distal vascular supply. A single vessel leg is not a contraindication to it being used as a recipient. We also favour use of the venae comitans as the recipient veins as they are more reliable than the superficial veins.⁶⁸ There is often a size mismatch but this is not a problem when using end-to-side anastomoses. Proximally in the leg, frequent branching, multiple valves and deeply situated vessels add to the challenges of the venous anastomoses. There is no evidence to suggest that utilizing only one donor vein increases the flow across the anastomosis and we also do not believe there is any merit in using a combination of a deep and superficial vein as recipients.

Although deep vein thrombosis can result in late loss of the flap, the risk is best mitigated by the use of low molecular weight heparin and encouraging early ankle motion to activate the calf pump.

Choice of muscle versus fasciocutaneous flaps

There is no clinical evidence to support the use of one form of soft tissue cover over another for open tibial shaft fractures. Published evidence consists almost entirely of descriptive, retrospective, observational case series (level IV evidence) with major discrepancies in outcome measures, which precludes a meaningful meta-analysis of these data. However, there is increasing experimental evidence which clearly shows that muscle coverage of shaft fractures leads to superior fracture healing compared to fasciocutaneous tissue.^{69–71} Muscle in direct apposition with diaphyseal fractures may aid healing by supplying important mesenchymal stem cells, which can undergo osteogenic differentiation into bone-forming osteoblasts.

In order to combine the benefits of a fasciocutaneous flap with the healing potential of vascularized muscle, we advocate the use of a free anterolateral thigh flap that includes a segment of vastus lateralis⁷² (Figure 46.6d). The plasticity of muscle also helps to obliterate the dead space, thereby reducing potential complications associated with haematoma formation.⁷³

From the available data and our own experience, we suggest that fasciocutaneous flaps may be superior to muscle for coverage of rapidly uniting metaphyseal fractures, particularly around the ankle, thereby avoiding skin grafts, which might be susceptible to minor trauma.

Compartment syndrome

Compartment syndrome develops when the pressure in the unyielding osseofascial compartments rises to reduce capillary flow to the point where nerve and muscle viability is impaired. Patients with open fractures can develop compartment syndrome. The first clinical sign is impaired function of the nerves traversing the compartment, leading to paraesthesia, followed by severe pain out of proportion to the injury and aggravated by passive muscle stretch.⁷⁴ Impaired pulses and reduced cutaneous capillary refill are late features, by which stage permanent nerve and muscle damage have already occurred. Pain can be obscured in patients with regional nerve blocks and particular vigilance is required in patients with impaired consciousness and in children. Measurement of compartment pressure using dedicated devices may be helpful, particularly serial measurements. A difference of 30 mmHg or less between the diastolic blood pressure and the compartment pressure may be used as a threshold for surgical decompression, but this results in unnecessary surgery in a number of patients. Increased specificity may be achieved by combining pressure with clinical findings but at the expense of much reduced sensitivity.⁷⁵ Discretion is the better part of valour and ultimately the diagnosis of

compartment is a clinical judgement and any suspected case should have urgent fasciotomies irrespective of measured compartment pressures.

Surgical decompression of the compartments is a surgical emergency as irreversible muscle necrosis may occur within 3–4 h. Decompression is best performed using the two incision technique (see Figure 46.1).¹ The cutaneous incisions are identical to those for wound extension: 1.5 cm medial to the medial subcutaneous border of the tibia and 2 cm lateral to the lateral subcutaneous border. The fascia is divided in line with the skin incision, avoiding any undermining. The deep posterior compartment is decompressed proximally by removing the origin of soleus from the tibia and distally by dividing the deep fascia over the flexor hallucis longus, taking care not to inadvertently injure the posterior tibial neurovascular bundle. The lateral incision decompresses the anterior tibial compartment. The dissection proceeds laterally in the subfascial plane until the vertical septum separating the anterior and peroneal compartments is encountered and divided. Care should be taken not to expose the peroneal tendons or injure the superficial peroneal nerve. Decompression must be accompanied by debridement of necrotic muscle. If extensive necrosis is encountered, a second look 24–48 h later is indicated. NPWT is then applied to the wounds.

The management of late diagnosis of compartment syndrome is controversial. Decompression may necessitate the excision of all non-viable muscle to avoid infection and subsequent free flap reconstruction may be necessary. Alternatively, closed treatment may be utilized ensuring adequate patient hydration, renal perfusion and functional joint splintage. The affected muscle will fibrose and the patient may have a reasonable outcome, depending on the function of the nerves traversing the affected compartment.

Vascular injuries

Vascular injuries are uncommon but potentially devastating. Prompt recognition is vital and requires a high index of suspicion. A compromised circulation results in irreversible muscle and nerve injury within 3–4 h.⁷⁶ Not all devascularized limbs should be reconstructed. Delayed reperfusion of ischaemic tissue results in systemic reperfusion syndrome, including renal failure and death. Furthermore, protracted attempts at limb salvage can adversely affect the patient.⁷⁷ Amputation should be considered and discussed preoperatively whenever possible. An algorithm for managing lower limb fractures with ischaemic vascular injuries has been formulated based on a meta-analysis of published series.⁷⁶

The pattern of injury is usually predicted by the fracture configuration. Common fracture patterns leading to an avascular limb include displaced femoral fractures, often with an associated butterfly fragment, fracture dislocations of the knee, proximal third tibial fractures and fracture dislocations of the

ankle.¹ Fracture reduction in the emergency department may improve blood flow, particularly in the case of ankle dislocations. Presence of capillary return in the toes can be misleading as blood pooling can give the same appearance. Palpation or Doppler ultrasound of the dorsalis pedis and posterior tibial arteries is preferable. Departmental preoperative angiography is contraindicated as it will delay revascularization.⁷⁶

If angiography is required, it can be performed in the operating theatre by locating the femoral artery via a groin incision, and directly injecting contrast medium while applying pressure proximally. If the patient is undergoing a CT scan for multitrauma, a CT angiogram of the injured limb may be performed at the same time. The suspected site of vascular injury should be explored and vascular shunts (e.g. Javid or Pruitt) inserted to restore the arterial supply. For injuries at the level of the popliteal vessels or more proximally, the vein is also shunted. The anaesthetist must be informed prior to reperfusion because the return to the systemic circulation of the metabolic products from the ischaemic limb may compromise systemic parameters such as blood pressure. Restoration of distal flow by shunting enables an assessment of tissue viability. If the limb is deemed salvageable, a bridging external fixator is then used to stabilize the skeleton. Finally, approximately 2 h after insertion of the shunts, definitive vascular repair can be performed. Our preference is for forearm cephalic or basilic vein grafts, which lie outside the zone of injury, are easily harvested and tend to adapt their calibre to accommodate the flow.

It is important to assess the patient for compartment syndrome by frequent serial pressure measurements in all four compartments. Unnecessary fasciotomies should be avoided to reduce bleeding as the patient often has a coagulopathy. If free flap reconstruction is indicated, the vein grafts can be used as recipient vessels if necessary.

Amputation

The surgeon's desire for reconstruction coupled with the patient's wish for limb salvage has to be balanced by the excellent functional outcomes of successful transtibial amputation. Published outcomes of salvage or amputation for severely injured limbs are similar^{78–80} but successful transtibial amputees enjoy shorter inpatient stays, fewer surgeries and a faster return to unaided ambulation. Early decision making is key and multiple unsuccessful attempts at limb salvage leading to delayed amputation must be avoided.

Absolute indications for immediate amputation include incomplete traumatic amputation, a devascularized limb with a warm ischaemia time in excess of 4–6 h, a severely crushed foot and severe open limb trauma in an unstable polytraumatized patient. Common scenarios where amputation may be preferred to reconstruction include loss of two or more muscle compartments in the leg, especially the posterior compartment and extensive bone loss involving one third or more of the tibia.

Combined open tibial and severe foot trauma may be best served by an early amputation. Suspected tibial nerve injuries must be explored and transected or avulsed neural injuries should be considered in the context of associated tissue trauma. It is not sufficient to rely on clinical assessment of absent or impaired plantar sensation.⁸¹

Despite recent advances, the decision to amputate remains difficult and scoring systems have not proven useful.⁸² We advocate a staged multidisciplinary approach to decision making in difficult cases. The zone of injury is assessed and photographed at the initial debridement. The patient and relatives are counselled and introduced to amputees and limb salvage patients before an informed decision is reached within 4–5 days. Delay beyond 5 days is associated with a rapid increase in deep infection rates.⁸³

Amputation surgery

The aim of surgery is the creation of an ideal residual limb with a cylindrical shape, a functioning myodesis, no end neuroma and sensate skin throughout. A functioning knee joint must be preserved whenever possible. Transtibial amputation results in 10–30%^{84–86} increase in ambulation energy expenditure with a prosthesis compared to 40–70%⁸⁵ for transfemoral amputation.

Through-knee amputations are occasionally preferred to transfemoral because of significantly improved limb fitting characteristics and faster mobilization.⁸⁷ However, the patient must be advised that the knee joint will be at a different level to the contralateral limb. We advocate early involvement of the limb fitting team who can provide valuable advice, particularly in complex bilateral limb trauma cases.

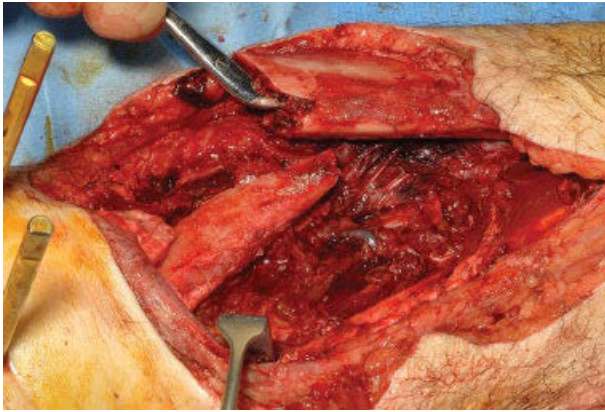
Amputation surgery in the presence of a wide zone of injury is challenging (Figure 46.7). Skin flaps can be difficult to plan at the outset so we use a fillet technique whereby all the soft tissues are initially preserved by making an incision along the pretibial border. The residual tibia should be approximately 15 cm long and ideally not less than 10 cm. The fibula is cut shorter, preserving the proximal tibiofemoral joint. The cut vessels are ligated but the perforators to the skin envelope are maintained as far as possible. Nerves should be cut short after infiltration of the epineurium with bupivacaine and allowed to retract into the muscles. Regional anaesthesia may reduce the incidence of phantom pain. Available calf muscles are used to achieve an end myodesis via drill holes through a well-rounded bone end. An adductor myodesis is essential to prevent a transfemoral limb drifting into valgus. The available skin is then fashioned to achieve tension-free closure using as much normal skin as possible and avoiding end-bearing wounds and skin grafts. Occasionally, special techniques such as the fillet of foot flap may be necessary.⁸⁸

Infection

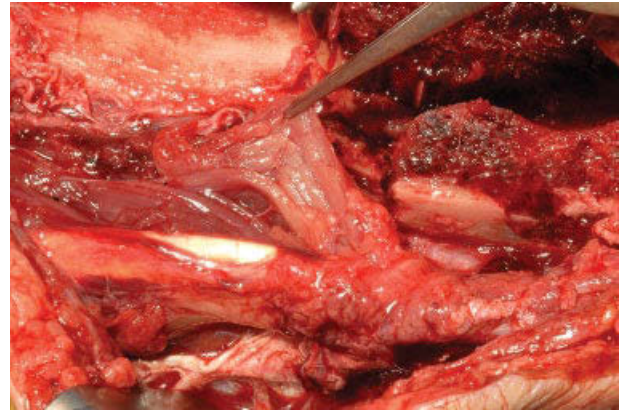
A severe open tibial fracture has all the ingredients for infection: a contaminated wound and devitalized tissue, ideal for bacterial proliferation; subsequent exposure to hospital pathogens which



(a)



(b)



(c)



(d)



(e)

Figure 46.7 Transtibial amputation. A 62-year-old man fell from a roof sustaining a grade IIIB open tibial fracture that was unsalvageable. (a) Initial radiographs show a displaced fracture of the distal tibial shaft with a significant intra-articular component. There were also segmental comminuted fractures of the fibula. (b) There was periosteal stripping of the exposed bone ends and approximately 4 cm bone loss following debridement. (c) The posterior tibial nerve, held in the forceps, was partially divided and severely contused. (d) Postamputation appearance highlighting a cylindrical-shaped residual limb with normal skin where possible and tension-free wound closure avoiding end-bearing wounds. (e) Progress at three months showing successful limb fitting and unaided walking.

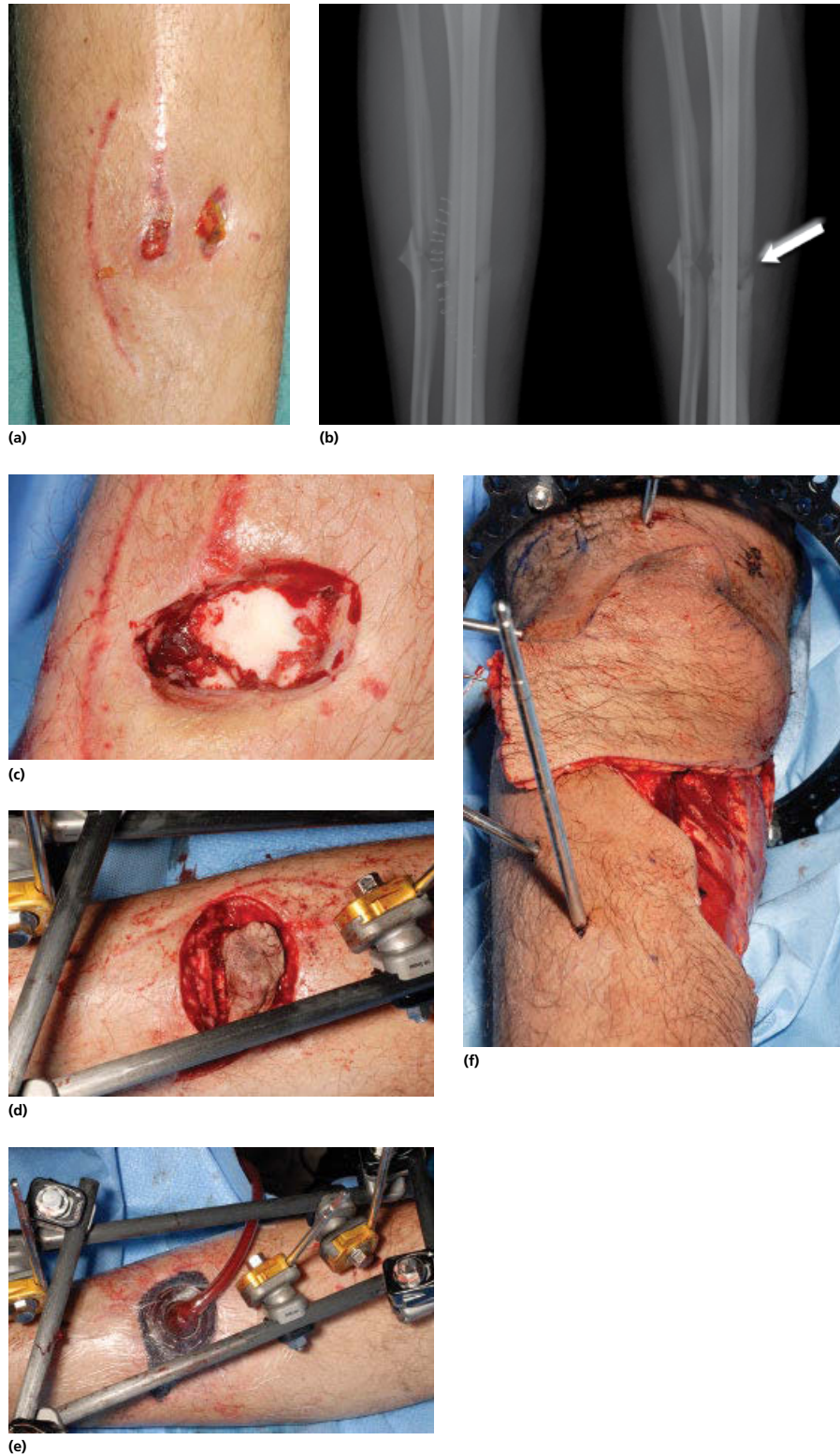


Figure 46.8 Infection of grade II midshaft tibial fracture. **(a)** Discharging wounds at eight weeks following injury. Note the separate lateral incision that had been used for the initial debridement without excision of the original open wounds. **(b)** Left AP X-ray immediately after nail fixation showing clips on the separate lateral surgical incision. At six weeks (right) bone resorption can be seen at the fracture site suggestive of deep infection (arrow). **(c)** Excision of the original injury wounds exposed the bone ends, which were devoid of periosteum. **(d)** The intramedullary nail was extracted, the canal reamed and lavaged and bone resected back to bleeding bone. Vancomycin-containing bone cement was inserted into the resection gap and a BEF applied for stability. **(e)** Negative pressure wound therapy (NPWT) was used as temporary dressing. All tissue specimens cultured sensitive *Staphylococcus*. **(f)** A proximally based fasciocutaneous flap was raised and the bone defect closed by acute shortening. A circular external fixator was applied for definitive fixation prior to proximal corticotomy. **(g)** Radiograph showing early bone formation in the distraction gap and compression at bone resection site. **(h)** Final radiograph showing healing of the regenerate bone and union at the fracture site.

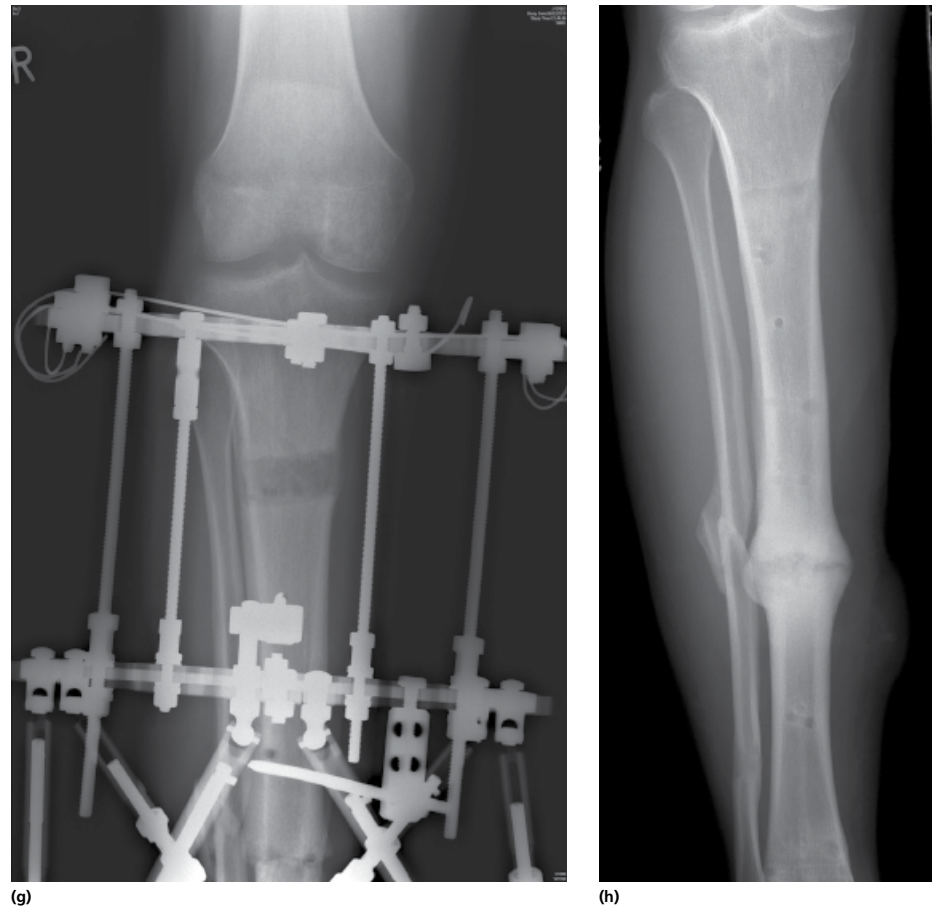


Figure 46.8 (Continued)

adhere to dead bone and metalwork and produce 'slime' or bio-film. Stagnant blood filling dead spaces can provide further enrichment in a microenvironment with limited penetration by host immune cells and antibiotics.

Prevention of infection in high-risk cases can only be achieved by a strict adherence to evidenced-based protocols and meticulous surgical techniques. Cases at high risk for infection include farmyard and agricultural injuries, delayed presentation or debridement, extensive zone of injury, vascular injuries and bone loss. A more aggressive debridement will be required and we advocate multiple tissue sampling after debridement in all potentially contaminated and high-risk cases. Definitive fixation and soft tissue cover are only performed when cultures are negative. Deep infection associated with an implant is a potentially devastating complication and the orthopaedic surgeon must be confident that the conditions are suitable before considering internal fixation. The plastic surgeon must also ensure the flap is not simply covering dead tissue which is unable to bring about a healing response and fracture union, but rather serves as a nidus of deep infection.

The incidence of infection after open tibial fracture has reduced with the advent of modern techniques but around 1 in 10 patients will develop deep infection^{4,5} and treatment depends largely on the time since contamination.⁸⁹

Acute Infection

Acute or early infection usually means within four weeks of injury. Careful clinical monitoring combined with a high index of suspicion enables early detection. Complications such as erythema, wound drainage and subflap collections must be expected and managed urgently to prevent established deep infection. Regular C-reactive protein (CRP) monitoring is the most valuable investigation and rising CRP levels invariably herald infection. Imaging modalities such as ultrasound and MRI are not very useful in the acute setting and may delay treatment. Prompt surgical exploration is mandatory to prevent deep infection, ideally after stopping antibiotic therapy for at least 24 h to enable representative microbiological sampling.

Fracture stability and tissue viability are carefully assessed at surgery and any collection is evacuated. Multiple deep surgical samples including bone are obtained before high-dose, broad-spectrum bactericidal antibiotics are given pending culture results. The common infective organisms are *Staphylococcus aureus*, *Enterococcus*, *Enterobacter* and *Pseudomonas*.⁶

The orthopaedic team must be prepared to undertake an aggressive debridement if devitalized tissue is encountered and contaminated internal fixation should be removed and replaced by stable CEF. We do not advocate retaining internal fixation in the presence of infection, even with sensitive organisms.

Published studies do not support this approach, particularly in cases of open fractures and IMN.^{90,91}

Flap tip necrosis will require debridement and flap advancement if possible (Figure 46.8). Alternatively, flap revision may be indicated particularly if the fracture site is exposed. Acute infection after grade I and II open trauma managed initially by wound closure, usually have a soft tissue defect after debridement and may require flap cover. Staged treatment should be considered with antibiotic-loaded cement spacers providing local antibiotic delivery and deadspace management until the bed is ready for cover.

An aggressive orthoplastic approach to acute infection is supported by our series of open tibial fractures initially managed in non-specialist centres and characterized by inadequate initial debridement, suboptimal fixation and contamination or frank infection at presentation.¹⁹ Despite late definitive treatment, the final outcomes in these patients were excellent and similar to patients receiving early treatment.

Chronic infection

Chronic infection implies the patient has lost the 'race for the surface' and deep infection of bone and hardware is established. Wound healing and fracture repair are severely impaired and definitive treatment usually requires demanding, complex surgical intervention. We recommend a multidisciplinary approach to select the most appropriate treatment modality involving orthopaedic and plastic surgeons, musculoskeletal radiologists, medical microbiologists and rehabilitation specialists.

The general health of the host is established:

- Type A: normal immune status;
- Type B: some degree of immunocompromise (e.g. smoking);
- Type C: significant immunocompromise (e.g. diabetes).⁸⁹

The functional limitation caused by the infection is considered and a thorough orthoplastic assessment of the involved limb is undertaken. Proximal and distal joint function is examined as well as limb alignment, local soft tissue status and limb vascularity. Imaging modalities are used selectively: CT scans define bone architecture and extent of necrosis, MRI and nuclear scanning help delineate the zone of injury and angiography will highlight local vascular abnormalities. Finally, accurate microbiological information obtained by multiple deep sampling allows the microbiologist to decide the most appropriate antimicrobial therapy.

Complex limb reconstruction in the presence of chronic sepsis can only be justified if salvage offers distinct advantages over a well-performed transtibial amputation. Chronic infection associated with a non-union may not cause major morbidity and definitive treatment may be worse than the disease. Low-risk treatment options such as intermittent antibiotic suppression or limb shortening with external fixation may be preferred for high-risk patients with low functional demands. Transtibial amputation should be considered, particularly if there is chronic ankle stiffness or significant muscle wasting which will preclude a good outcome from salvage.

If reconstruction is selected, an aggressive 'tumour excision' debridement of necrotic and ischaemic tissue is essential. The extent of surgical clearance is predictive of recurrent infection and a clearance margin of 5 mm is recommended.⁹² We use the presence of punctate bone bleeding (the 'paprika sign') to indicate viable bone margins. Staged treatment is preferred until post-debridement samples are culture negative and definitive cover can be performed. Cement spacers combined with NPW will eliminate deadspace between surgical procedures and antibiotic-loaded cement aid bacterial clearance.

We advocate skeletal stabilization by CEF, which provides long-term stability and addresses bone deficiencies while respecting the blood supply of remaining fragments. The use of CEF also limits the risk of cross-contamination associated with secondary internal fixation. Bone defects are managed principally by bone transport. Shortening of smaller defects may be also used to eliminate deadspace, improve limb stability and facilitate early fracture healing.

Soft tissue cover is selected according to the site and size of the defect. Microsurgical reconstruction can be challenging due to extensive perivascular fibrosis associated with chronic inflammation and anastomosis outside of the injury zone is recommended whenever possible. The Ilizarov method alone has been advocated for the treatment of both bone and soft tissue defects without the use of flap coverage.⁹³ However, the final soft tissue envelope is poorly vascularized and does not compare to the quality of vascularized soft tissue cover provided by modern plastic reconstructive techniques.^{94,95}

Conclusion

An integrated orthoplastic approach in a major trauma centre is the cornerstone of limb reconstruction after severe open trauma. Experienced orthopaedic and plastic surgical teams working closely with rehabilitation and infectious disease specialists provide the best chance for the successful restoration of limb function.

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CHAPTER 47

Lymphoedema

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Lymphoedema is a chronic debilitating disease resulting from an abnormal collection of protein-rich fluid within the subcutaneous tissues of the body. It may be due to aberrant development or malfunction of lymphatic channels (primary lymphoedema) or from permanent damage to a normal lymphatic system (secondary lymphoedema). This disruption to lymph drainage routes prevents the normal flow of interstitial fluid into and along lymphatic vessels with ultimate drainage back into the veins of the neck at the thoracic duct. The consequence is swelling within the affected drainage basin. This swelling, or lymphoedema, causes significant skin and subcutaneous tissue changes, as well as repeated infections which can be severe. The change in volume of the limb also results in an extremely unsightly and disfigured limb.

Embryology and anatomy of the lymphatic system

The Italian anatomist Gaspare Aselli published the first anatomical description of lymphatics as 'milky veins' in the mesentery of a dog in 1627. In 1651, Jean Pecquet in Dieppe discovered the thoracic duct and its entry into the left subclavian vein. At the same time, Olaus Rudbeck in Uppsala discovered that lymphatic vessels form a distinct vascular network throughout the body. His observations, together with those of Thomas Bartholin (who introduced the term *lymphaticus*) led to the first full description of the human lymphatic system, in which the role of lymphatic vessels in transporting fluid filtered from the blood was suggested.¹ In 1746 William Hunter demonstrated that lymphatic vessels are the absorbing vessels throughout the body. His comprehensive work laid the foundations for the modern knowledge of the anatomy, physiology and pathology of the lymphatic system. Subsequently Marie Sappey, a professor of anatomy from Paris, published his extensive work detailing the anatomy of the cutaneous lymphatic system,² using his procedure of injecting mercury into cadaveric skin.

By the end of the nineteenth century the anatomy of most of the lymphatic system had been described but its origins remained in dispute. Two theories were proposed regarding the embryological origin of the lymphatic endothelium. The first suggested that lymphatic endothelium derived from epithelial buds arising from venous channels in the early stages of embryologic life (centrifugal theory). The second suggested that lymphatic endothelium differentiates *in situ* from primitive mesenchyme, and secondarily acquires connection with the vascular system (centripetal theory).¹

Studies of mammalian embryos have shown that there are eight lymph sacs; three paired and two unpaired.³ The paired ones are the jugular, subclavian and posterior lymph sacs, and the unpaired are the cisterna chyli and the retroperitoneal lymph sac. Lymph sacs are venous derivatives that sprout into all parts of the body, except for the central nervous system and the bone marrow. According to this centrifugal theory, the peripheral lymphatic system originates from the primary lymph sacs and spreads by endothelial sprouting into the surrounding tissues and organs, where local capillaries are formed. Lymph sacs later will give rise to primary lymph nodes.

The alternative model proposed that lymphatic vessels developed independently from mesenchymal precursor cells and that connections with veins were only established later on during development. According to this model, lymphatic vessels originate by fusion of mesenchymal spaces into a primitive lymphatic network, which spreads centripetally (centripetal theory) and then establishes a connection to the venous system.¹ Both of these theories are now thought to play a role in lymphatic embryology: The main parts of the lymph sacs are derived from adjacent venous vessels, whereas the superficial, dermal lymphatics are derived from local lymphangioblasts. The two compartments fuse to form the lymphatic system.

Lymphatic vessels generally follow blood vessels within the tissues but the vascularity of a particular area is not always proportional to its lymphatic drainage. There are also areas that do not have lymphatic vessels, such as bone marrow and the central

nervous system as well as areas that have neither blood nor lymphatic vessels such as cartilage, the cornea of the eye and the epidermis of the skin. In order to provide the high cellular oxygen requirements, lymphatics have a rich network of blood vessels performing a nutritive function.

There are five main type of lymphatic conduit: initial lymphatics, collecting vessels, lymph nodes, trunks and ducts. Within the skin, there are two plexuses of lymphatics – superficial and deep. The superficial lymphatics are valveless and the vessels of the deep lymphatic plexus have valves.

Having left the interstitium, lymph enters the initial lymphatics, which include the smallest lymphatic capillary and the larger precollector vessel.⁴ The precollectors drain into collecting vessels, which pass through lymph nodes. From these collecting vessels the lymph drains into larger trunks, which then drain into ducts (apart from intestinal, hepatic and lumbar lymphatics which drain directly into the cisterna chyli). The ducts finally return the lymph back into the bloodstream.

Physiology

Oedema represents an increase in interstitial fluid volume sufficient to manifest with swelling. Whatever the cause of the oedema, it is due to an imbalance between capillary filtration and lymph drainage.⁵ Most examples of limb oedema are caused by capillary filtration overwhelming lymph drainage capacity. Lymphoedema generally occurs when swelling is due to a failure of lymph drainage in circumstances in which capillary filtration is not increased.⁶ There are a number of mechanisms for chronic oedema that may equate to lymphoedema:

- If there is simple lymphatic failure with no increase in capillary filtration (normal lymph load) then this is lymphoedema.
- If there is lymphatic failure with increased capillary filtration (high lymph load overwhelming lymph drainage capacity) then this results in filtration oedema with lymphatic failure.
- If there is lymphatic failure resulting from sustained increased capillary filtration exhausting lymph drainage capacity then lymphoedema is the result.⁷

The physiology of lymph drainage

The lymphatic system is crucially important for maintaining fluid balance in tissues. It is a one-way drainage route that removes from the interstitium unwanted material and excess fluid. Approximately half of the total circulating protein escapes from the blood vessels each day and the key function of the lymphatic system is to return to the blood vascular compartment protein, colloids, and particulate matter too large to re-enter the bloodstream directly.⁸ This maintains the plasma volume and prevents an increase in tissue pressure. The distances between cells and capillaries also need to be kept to a minimum to maintain cell nutrition and so any excess fluid within the interstitium must be constantly cleared.

The protein composition of lymph is similar to that of interstitial fluid and slightly less concentrated than that of blood plasma. The exception to this is intestinal lymph or chyle, which is a milky emulsion as a result of its high fat content. The lymphatic system is also involved in the body's response to infection: antigen-presenting cells are directed to the lymph nodes by lymphatic vessels and are essential for the development of cellular immunity. Impairment of these lymphatic functions results in a number of diseases that are characterized by oedema, impaired immunity, and fibrosis.⁹

The ends of lymphatic capillaries are open to the interstitium in order that fluid and molecules can pass into the vessels. In addition, the outer surfaces of the lymphatic endothelial cells are tethered to the surrounding tissues by anchoring filaments. If the interstitial volume increases, this stretches the surrounding connective tissue, thereby pulling on the anchoring filaments. This has the effect of widening the lymphatic lumen (allowing an increase in fluid transport), and also opening intercellular channels, which facilitates the passage of fluid and molecules into the lymphatic. Thus an increase in interstitial pressure results in an increase in lymphatic flow, but only up to a maximum level (approx. +2 mmHg). Once the pressure exceeds this level, there is no further increase in flow, and oedema results.⁹

The collecting lymphatics are the main limb lymphatic vessels that provide the afferent flow to the lymph nodes. The smooth muscle in their walls in conjunction with their valves is responsible for the intrinsic propulsion of lymph centripetally and this is the essential motor for lymph flow.¹⁰ However, flow can be only as good as the supply of lymph to these collectors and hence the physiology is often compared with Starling's law of the heart.

The supply of lymph from the initial lymphatics depends on a different mechanism. Flow of interstitial fluid and macromolecules into and consequently along initial lymphatics is caused by intermittent changes in hydrostatic and oncotic pressures locally and is termed convective flow.¹¹ Deformation or movement of the tissues by surface pressure or underlying muscle contractions and by other contractile structures, such as arterioles, causes compression of the initial lymphatics which forces lymph along initial lymphatics. Valves ensure that flow is unidirectional.

Differential diagnoses

Non-inflammatory oedema may occur whenever there is a disturbance to Starling pressures and an increase in microvascular fluid filtration. Circumstances where this occurs include an increase in intravascular hydrostatic pressure (e.g. heart failure) or a fall in plasma colloid osmotic pressure (e.g. protein losing state). Contrary to popular belief, kidney failure does not cause oedema except in nephrotic syndrome or endstage renal disease when salt and water retention occurs. There are numerous clinical settings where oedema is caused by increased fluid filtration overwhelming lymph drainage and they must be

distinguished from oedema caused by sole failure of lymphatic drainage as seen in true lymphoedema.

Increased hydrostatic pressure can result from either impaired venous return or from arteriolar dilatation. The commonest cause of impaired venous return is congestive cardiac failure, but pericarditis, liver cirrhosis and venous obstruction should also be considered. Arteriolar dilatation may result from heat, neurohumoral abnormalities or from calcium channel antagonists used to treat hypertension.

Reduced plasma oncotic pressure results from the proteinuria seen in nephrotic syndrome, impaired protein synthesis due to liver cirrhosis, gastrointestinal conditions that lose protein and malnutrition.

Advanced renal disease results in sodium retention and causes systemic oedema.¹²

Lymphatic malformations may exist in isolation, with no communication with a normally draining lymphatic system. In such cases swelling arises from lymph fluid distending malformed lymphatic vessels. If malformations communicate and interfere with lymph drainage then lymphoedema occurs. One such example is Klippel–Trenaunay syndrome (capillary malformations of an extremity overlying a venous or lymphatic malformation, associated with limb hypertrophy and bony enlargement), which has been shown to have an associated primary abnormality of the lymphatics in up to 55% of cases.¹³

Aetiology

Lymphoedema arises when there is an intrinsic fault within the lymph conducting system (primary lymphoedema) or when damage occurs from factors originating outside the lymphatic system (secondary lymphoedema).

Primary lymphoedema

Primary lymphoedema is caused by an intrinsic abnormality of the lymphatic drainage channels. This may be due to:

- *aplasia* – absence of subcutaneous lymph trunks;
- *hypoplasia* – commonest type in which there are an insufficient number of lymphatic collectors; demonstrated by lymphographic studies;¹⁴ or
- *hyperplasia* – enlarged lymphatics that are increased in number and may be tortuous.

The diagnosis of primary lymphoedema is made once other causes have been excluded. The frequency at birth of those who will develop primary lymphoedema is estimated to be about 1 in 6000, with a sex ratio of about one male to three females.

Inheritance has been thought to be one of the causes since 1892, when William Milroy from Omaha described a family with the condition passing through six generations.¹⁵ If swelling is present from birth (i.e. congenital) as well as being familial then it is most likely to be Milroy's disease. In such patients the form of inheritance is now known to be autosomal dominant caused by mutations in the *VEGFR3* gene.¹⁶ The

mechanism for Milroy lymphoedema is a failure of the initial lymphatics, despite their presence in the skin and subcutaneous tissues.¹⁷ Overall, nine discrete gene mutations have been linked with primary lymphoedema, among them *VEGFR3* (Milroy's disease), *FOXC2* (lymphoedema distichiasis syndrome) and *SOX18* (hypotrichosis lymphoedema telangiectasia syndrome).¹⁸

However, for the majority of cases of primary lymphoedema, swelling does not develop until puberty, in which cases it remains unclear as to whether the lymphatic vessels are abnormal at birth. In cases of hypoplasia, lymphoedema develops either at puberty (lymphoedema praecox or Meige's disease) or later in adult life (lymphoedema tarda), yet the genetic fault is present from birth. It is possible that the defect may be programmed from birth (as suggested by the familial nature of such cases of primary lymphoedema) and that a degenerative process develops to cause subsequent lymph drainage failure. The demonstration of severe atrophy of the smooth muscle layer in proximal limb lymphatics with relative sparing of distal vessels suggests that a degenerative process may begin proximally and spread distally.¹⁹ This could help explain the variable time for onset of all lymphoedema.

Another subset of primary lymphoedema is where lymphatics are excessive in size and number (hyperplasia). Lymphatic collectors that are expanded and enlarged are also termed megalymphatics. Lymphatic valvular insufficiency and reflux is, perhaps unsurprisingly, often seen in these cases.²⁰ Such cases may also be associated with the low flow vascular malformations known as lymphatic malformations.

Secondary lymphoedema

If a pathological cause for the lymphoedema can be determined then it is termed secondary lymphoedema and this is much commoner than primary. In the developed world the majority of secondary lymphoedema results from cancer and its therapy, but in developing countries various forms of infection can also result in reduced drainage. The causes can be summarized as:

- *Malignancy* – infiltration of lymphatics and nodes by cancer.
- *Damage* to nodes and or channels by surgery/radiotherapy/accidental trauma/silica absorption through skin (podoconiosis). The resultant lymph drainage impairment may arise from downstream obstruction, valvular incompetence and backflow (hence dermal backflow) or obliteration of peripheral lymphatics.²⁰
- *Infection* – chronic inflammation secondary to infection with filariasis caused by *Wuchereria bancrofti* is a classic infective cause worldwide. Not only can the worms cause mechanical obstruction but also the products of the filarial parasites depress lymphatic contractility.²¹ However, with respect to global causes of infective lymphoedema, tuberculous lymphadenitis and chronic sepsis are commoner. Recurrent bouts of cellulitis/lymphangitis result in obliteration of lymphatic vessels and deterioration of the lymphoedema.

Breast cancer-related lymphoedema

Chronic oedema following mastectomy for breast cancer was first described by Halstead in 1921.²² Modern trends in therapy for breast cancer, including the Z11 study²³ have resulted in more conservative surgery and fewer axillary clearances, but breast cancer-related lymphoedema (BCRL) remains a common problem. Reports of incidence vary, but one large cohort displayed a cumulative incidence of 28%.²⁴

Similarly, a 10-year follow-up of 256 patients treated with breast irradiation following sentinel node biopsy or axillary clearance showed chronic lymphoedema occurring in 34% of patients.²⁵ The same group have also proven the widely accepted fact that lymphoedema is significantly more likely to occur subsequent to axillary clearance than after sentinel node biopsy alone.²⁶ A recent meta-analysis still puts the incidence of arm lymphoedema at more than one in five women.²⁷ This takes no account of increasing levels of breast lymphoedema as a result of breast-conserving surgery, a problem that never existed with mastectomy.

Cases of lymphoedema continue to emerge up until 5 years after breast cancer surgery, if not longer.²⁸ Clearly in these cases relapsed carcinoma must be excluded in the first instance. After an initial rapid expansion period, arm volumes tend to stabilize and remain in a relatively steady state.⁶

The pathophysiology of BCRL would seem to be the result of damage to the axillary lymphatic system caused by surgery and/or radiotherapy, which thus impair lymph drainage from the arm. Lymphangiography performed subsequent to treatment but before swelling shows dilatation of the main collecting lymphatics in the arm²⁹ and contrast medium appears to be delayed in the axilla. Normally, lymph from the superficial system drains into the deep lymphatic system across fascial connections. As oedema develops, the deep lymphatic system is no longer visualized and there is much dermal collateralization with retrograde flow from deep vessels back to the superficial skin lymphatics.³⁰ This reversal of flow or 'dermal backflow' indicates valvular incompetence.

If lymph drainage is reduced (as is the case in lymphoedema) then capillary filtration and consequently protein concentration (and interstitial osmotic pressure) would be expected to rise. However, the analysis of interstitial fluid extracted from both swollen and non-swollen arms demonstrated that protein concentrations were *lower* in the swollen arm, to the extent that protein concentration correlated negatively with the severity of arm swelling.³¹ It was thought that this might be due to a vascular contribution such as increased capillary filtration.

Venous obstruction has been suggested to have a causative role – altered venous anatomy has suggested obstruction³² and duplex ultrasound imaging has shown that only 30% of patients with chronic arm swelling after breast cancer are normal. However, in contradiction of these facts, measurement of venous pressures in postmastectomy lymphoedema arms did not show an increase.³³

Another possible vascular cause would be failure to maintain precapillary resistance, allowing blood flow and capillary

pressure to rise and therefore increasing the filtration rate. Spectral Doppler studies of mean axillo-subclavian blood flow have demonstrated a 68% increase in lymphoedematous limbs.³⁴ Venous occlusion plethysmography has also shown that total arm blood flow is increased in the majority of such patients.³⁵

This finding of increased blood flow to the whole arm suggests either vasodilatation of existing resistance vessels or formation of new ones (angiogenesis). Studies investigating the possibility of vessel dilatation have shown that vasoconstrictor control and sympathetic vasodilator control were normal in BCRL, but local vasodilator control was impaired.³⁶ Sustained vasodilatation as a cause of the rise in capillary pressure was not seen. Regarding new vessel formation, a study has found evidence of an increase in total capillary numbers in BCRL.³⁷ Such capillary angiogenesis could be increasing the surface area for an exchange and so increase the filtration load.

All oedemas result from an imbalance between capillary filtration and lymph drainage and thus the state of the microcirculation must be considered as well as of the lymphatic drainage. Lymph flow was raised in both the subcutis and muscle of both arms in postsurgical breast patients who later developed BCRL, compared with those who did not. Changes in the contralateral arm suggest that systemic factors contribute to BCRL.³⁸ There is therefore strong evidence to believe that haemodynamic factors contribute to the oedema.

Clinical history, features and assessment

Lymphoedema can be difficult to differentiate from other types of oedema. However there are a number of clinical pointers that would suggest a diagnosis of true lymphoedema:

- Oedema that only slightly resolves with overnight elevation is likely to be a lymphoedema as elevation does not improve lymph flow (indeed the opposite because it reduces microvascular fluid filtration).
- Diuretics do not improve lymphoedema because diuretics reduce body salt and water with no stimulation of lymph drainage.
- More than one attack of cellulitis suggests lymphatic insufficiency.
- Exercise improves lymph drainage.
- Lymphoedema pits in its early stages like any other oedema.
- Lymphoedema causes skin thickening and hyperkeratosis, so-called elephantiasis.

Lymphoedema generally presents with limb swelling and this may be associated with symptoms of heaviness or aching of the affected limb. Pitting (pressure from a finger or thumb leaving an indentation after removal) is often seen in the early stages due to the fact that the increase in limb size is mostly due to its fluid component. As the disease progresses, fibrosis and fat deposition occur, with the result that the limb may no longer pit.

Historical teaching suggested that pitting oedema differentiated lymphoedema from other causes of swollen limbs. However oedema of many causes (including lymphoedema) may initially

show pitting but similarly oedema of any nature may not pit once it becomes chronic. All oedema represents a form of lymphatic failure. This is because, contrary to traditional teaching, the evidence indicates that all interstitial fluid is reabsorbed by the lymphatic in the steady state with no venous reabsorption. Hence any peripheral oedema is either solely due to lymph drainage failure in the face of a normal microvascular fluid filtration (normal lymph load) or lymph drainage capacity is overwhelmed by increased fluid filtration (increased lymph load). Therefore all oedema is either absolute or relative lymph drainage failure.^{5,7}

The swollen limb is disfiguring and commonly causes functional impairment, a feeling of heaviness and aching, psychosocial maladjustment and psychological morbidity.³⁹ Features of lymphoedema seen in the lower limb include loss of ankle contour, square profile of toes and lichenified filiform fronds. A positive Stemmer's sign,⁴⁰ which is the inability to pinch a fold of skin (due to increased skin thickness) at the base of the second toe, is pathognomonic of lymphoedema.

Recurrent acute inflammatory episodes ('cellulitis' or 'erysipelas') can affect up to one-third of patients. Such attacks increase swelling and compound the problem by causing further damage to lymph drainage routes. It has been suggested that this propensity for infection is due to increased fluid and protein content of the skin and subcutaneous tissues.⁴¹ A much more likely explanation is the effect disturbed immune cell trafficking has on local tissue immunity. Cellulitis exacerbates swelling, may require hospitalization for intravenous antibiotics and can destroy residual lymphatics. If multiple repeated episodes of cellulitis occur, the limb may reach massive proportions and may become pachydermatous (elephantiasis).

The psychological harm that results from lymphoedema is a significant problem that should not be underestimated. A study of a cohort of women presenting for therapy for their lymphoedema has shown that these patients have high levels of psychological distress and of functional and social dysfunction. There is no linear relationship between severity of lymphoedema and levels of distress. Women with pain of any intensity were the most distressed, and had the most significant difficulties in psychological and physical functioning.⁴² Unfortunately this psychological distress can result in an avoidant coping style and low perceived social support, which may interfere with any treatment. Efforts must therefore be made to strengthen the patient's coping skills, ideally through a multidisciplinary approach.⁴³

A thorough clinical examination is crucial, in particular of the vascular system, which can be assessed with suitable imaging such as a duplex scan or venogram. Assessment tools that can be used to clinically evaluate patients with extremity lymphoedema include circumference measurement, water displacement volumetry and perometry as well as bioimpedance spectroscopy. The latter two may be less prone to human error. Imaging techniques are also useful and are discussed elsewhere in this chapter.

The water displacement method (water plethysmography) is an accurate way of calculating limb volume and is the only reliable method available for the measurement of oedematous hands and feet. There are issues with hygiene and access to such equipment that limit its use.

Perometry uses infrared light beams to measure the outline of the limb; from which localized areas of differential oedema can be quantified, as well as an overall volume assessment. This is arguably the gold standard. Bioimpedance spectroscopy measures tissue resistance to an electrical current in order to determine extracellular fluid volume. Although not yet established in routine practice, it may prove useful in demonstrating early lymphoedema and in monitoring response to treatment.⁴⁴

There is no effective method for measuring the head and neck, breast, trunk or genitalia, although 3D photography or imaging may have a role for these areas.

Lifestyle questionnaires such as the Disabilities of the Arm, Shoulder and Hand (DASH score) have been used for the assessment of lymphoedema patients. They concentrate on evaluating the capability of the patient to carry out everyday tasks and can be used to assess the efficacy of different intervention according to these parameters.

Investigations/imaging

The search for the ideal method of imaging the lymphatic system has been a source of extensive research with some invaluable recent advances. Nineteenth-century anatomists used materials such as mercury, wax and gelatine to perform lymphography on cadavers. *In vivo* studies have used interstitial injections of contrast agents, which are then taken up by the lymphatic circulation. Radioactive proteins or colloids are imaged by a gamma camera (lymphoscintigraphy). Vital dyes such as patent blue, injected intra- or subdermally, can be seen with the naked eye and used to identify lymphatics *in vivo*. Traditionally, radiocontrast agents were injected directly into subcutaneous lymphatic collectors and imaged by X-ray (lymphangiography). The latter lost popularity because it is an invasive technique and can damage the lymphatic vessels and worsen lymphoedema.

In recent years the introduction of fluorescent tracers, water-soluble contrast agents and isotopes, in parallel with the advent of physiological surgical procedures, has reinvigorated the role of imaging in lymphoedema. Surgery planned to divert lymphatic fluid may only be successful if the surgeon can identify the position of the tiny lymphatic channels (on average 0.3–0.8 mm diameter) and establish whether they are patent and functional. Near-infrared spectroscopy, lymphoscintigraphy and MR lymphangiography have had particular success in the assessment of candidates for physiological procedures.

Lymphoscintigraphy is perhaps the most well-recognized modality used for the evaluation of lymphatic function in lymphoedema. Although it provides quantitative assessment of

lymphatic function and flow abnormalities, it is time consuming, expensive and unsuitable for perioperative use.

Near-infrared spectroscopy allows real-time visualization of lymphatic vessels with minimal invasiveness. It accurately shows the flow of lymphatic fluid without the need for radioactive isotopes. It uses the principle of fluorescence lymphography, which detects near-infrared light emitted by a fluorophore (commonly indocyanine green) that has been injected into the affected limb. The fluorophore binds with albumin, which is then selectively taken up by, and transported through, the lymphatic circulation. This not only demonstrates the position and path of lymphatic vessels within a limb, but also provides a dynamic functional assessment (Figure 47.1) that is comparable to lymphoscintigraphy.⁴⁵

Fluorescence lymphography using indocyanine green dye in this manner has previously been used for various clinical situations such as liver function evaluation, assessment of burn depth, sentinel lymph node detection, cardiac assessment, perforator identification and evaluation of flap viability.^{46–48} The only factor excluding patients from this technique is a history of allergic reactions to iodine. A recent anatomical and physiological study used near-infrared fluorescence to image lymphatics in normal volunteers and lymphoedema patients. It elegantly displayed active propulsion, architecture and extravascular leaks.⁴⁹ This imaging technique can also be used to show postoperative flow in the diverted lymphatic channels to prove patency.⁵⁰

Magnetic resonance (MR) lymphangiography, a technique in which lymphangiograms are obtained by injecting standard MR contrast agents such as gadopentate in the subdermal plane followed by a series of image acquisitions, has been used to image lymphatics with some success.⁵¹ Imaging is generally performed with a 3 or 4 tesla scanner using a 3D spoiled gradient-echo sequence. Although this provides reasonable imaging of the lymphatics, it is both more invasive and expensive than near-infrared fluorescence. There have also been challenges in the

ability of these scans to differentiate between the venous and lymphatic circulation.

Lymphatic function can also be assessed in terms of tissue clearance rate, which describes the rate of removal of an injected radiolabelled substance from the tissues. This provides a quantitative measure of function in an easily reproducible manner.⁵² Lymphatic function can also be assessed by measurements of solute concentration ratios between plasma and lymph, as well by direct measurements of lymphatic capillary pressures.⁵³

Classification

The fact that there are multiple systems for classification of lymphoedema perhaps suggests that no single method is ideal. Generally, staging of lymphoedema can be differentiated into systems that assess clinical factors, immunohistochemical appearances and disability.

The increase in use of near-infrared spectroscopy by indocyanine green lymphography has resulted in accurate delineation of lymphatic anatomy. This imaging modality has been used to create a new classification system of lymphography findings in secondary lymphoedema of the lower limb reflecting both pathophysiology and severity of lymphoedema.⁵⁴ The findings are largely classified into 'linear' or 'dermal backflow' patterns and each of these are subclassified into three groups. This system certainly has merit for planning of microsurgical lymphatic bypass procedures but it remains to be seen whether the scientific community as a whole adopts it.

One of the most commonly cited classification systems for severity of lymphoedema is that of Campisi (Table 47.1).

The International Society of Lymphology (ISL) produces a consensus document on behalf of the international community. In its most recent publication⁵⁶ it summarizes the ISL classification of lymphoedema as follows:

- *Stage 0 (or Ia)*: A subclinical condition where swelling is not yet evident despite impaired lymph transport.
- *Stage I*: Early accumulation of fluid relatively high in protein content which subsides with limb elevation and may display pitting.

Table 47.1 Campisi extremity lymphoedema staging system

Stage 1A	No oedema despite the presence of lymphatic dysfunction
Stage 1B	Mild oedema that spontaneously regresses with elevation
Stage 2	Persistent oedema that regresses only partially with elevation
Stage 3	Persistent, progressive oedema; recurrent acute erysipeloid lymphangitis
Stage 4	Fibrotic lymphoedema with column limb
Stage 5	Elephantiasis with severe limb deformation, including sclero-indurative pachydermitis and widespread lymphostasis warts

Source: Campisi and Boccardo, 2004.⁵⁵

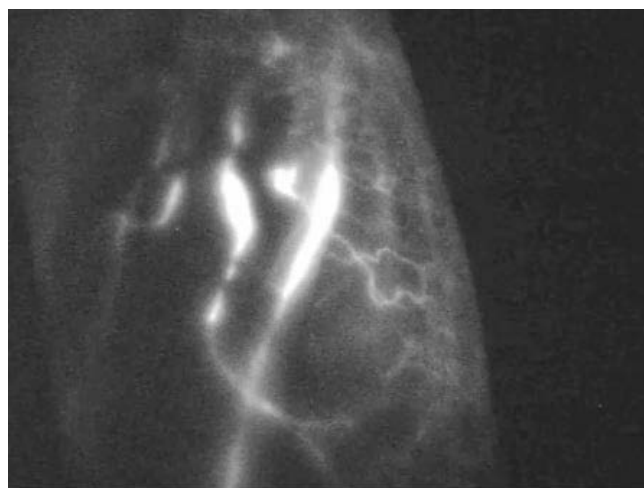


Figure 47.1 Indocyanine green lymphography showing propulsion of a bolus of dye along a linear lymphatic in the antecubital fossa. Source: Campisi & Boccardo, 2004.⁵⁵ Reproduced with permission of Springer.

- *Stage II*: Limb elevation alone rarely reduces swelling. In early stages pitting is manifest but as it progresses the limb may or may not pit as a result of excess fat and fibrosis.
- *Stage III*: encompasses lymphostatic elephantiasis where pitting may be absent and trophic skin changes such as acanthosis, further fatty deposition and fibrosis, and hyperkeratoses have developed.

Although this is an internationally accepted staging system, it only refers to the physical condition of the lymphoedematous limb and not pathophysiology. Another consensus document has been recently published by the International Union of Phlebology, but the classification is confined to primary lymphoedema although it does consider aetiology including causal mutations.⁵⁷ Another recently published classification system uses an algorithm based on phenotype and genotype to decipher clinical presentations of primary lymphoedema.⁵⁸

As understanding of the pathological, physiological, genetic and clinical features of lymphoedema improves, this will certainly lead to further evolution in our staging systems.

Management, complications and outcomes

Non-operative

The main challenges in the management of lymphoedema are:

- reversal of increased interstitial volume;
- amelioration of some of the secondary skin and subcutaneous tissue changes;
- prevention of infection.

Conservative measures are the mainstay of treatment designed to manage the condition and to minimize complications rather than to provide a cure.⁵⁹

Initial management of lymphoedema includes education and support, skin care, stimulation of lymph drainage and management of any concomitant conditions. The effect of exercise and movement on lymph flow is enhanced by compression. Intensive treatment using manual lymph drainage, multilayer lymphoedema bandaging and exercises is designed to reduce limb volume, restore limb shape and improve the condition of the skin. Once completed, maintenance treatment is in the form of exercises and compression. Compression can be implemented by prescription hosiery or Velcro wraps (e.g. Farrow wraps). Elevation is to be recommended when relaxing but never undertaken in preference to exercise.

Compression hosiery is designed to (a) reduce capillary pressure and consequently microvascular fluid filtration and (b) increase tissue interstitial pressure and so enhance fluid absorption into, and transport along, initial lymphatics. Clearly the presence of comorbidities, in particular vascular disease and heart failure, as well as the ability of the patient to tolerate them, must be considered with any compression techniques.

The use of *multilayer compression bandaging* has been shown to reduce volumes more effectively than compression garments. A mean overall reduction rate of lower limb lymphoedema of up

to 31% can be achieved after bandaging, followed by compression stockings compared with 15% using compression stockings alone.⁶⁰ However, stockings are more convenient and allow easier continuation of routine activities.

Manual lymphatic drainage (MLD) is designed to direct lymph drainage to normal lymph drainage basins. This manual technique concentrates on central lymphatics to 'clear the way ahead' for lymph draining from limbs. For example in BCRL, lymph fluid around the shoulder and on the trunk is directed through collateral routes to the contralateral upper quadrant lymph drainage basin before the ipsilateral arm oedema is drained by bandaging. The evidence in support of MLD is limited although one prospective trial supports the combination of MLD with compression bandaging for mild lymphoedema.⁶¹

Decongestive lymphatic therapy/complex decongestive therapy comprises MLD, multilayer compression bandaging, therapeutic exercise and skin care.⁴⁴ Földi defined two treatment phases of this (initial intensive intervention followed by multiple maintenance treatments), which have since been modified. A systematic review of these techniques combined together indicated that they reduce arm volumes more effectively than compression hosiery alone.⁶² However, multiple subsequent randomized trials have failed to show that it confers a significant benefit beyond that of compression alone, and therefore is difficult to justify as first-line therapy for early lymphoedema.⁶³

Intermittent pneumatic compression devices sequentially inflate and deflate along the length of the limb in order to push lymph and interstitial fluid proximally towards the trunk. Their benefit is not well proven.

Pharmacological treatment plays little role in the management of lymphoedema. Benzopyrones and diuretics have been used but there is little evidence to support their use.

Surgical management of lymphoedema

Many surgical procedures have been described, broadly divided into 'excisional' or 'physiological' techniques. Physiological operations are designed to improve the lymphatic function of the limb, whereas the other techniques involve debulking or removing skin and/or subcutaneous tissue.

In addition to these, simpler surgical procedures may be used to manage the cutaneous vesicles of lymphangiectasia such as diathermy, hyfrecation or laser therapy.

Excisional procedures

These surgical procedures have involved excision of some or all involved tissue, but can be severely mutilating operations. Homans' operation (1936)⁶⁴ involved raising flaps of skin and removing a longitudinal segment of involved skin and subcutaneous tissue. This was popularized by Miller in 1973 and was one of the most accepted surgical treatment for lymphoedema. Scars were extensive and wound complications common.⁶⁵ Kondolean procedure (1912) involves resection of the lymphoedematous tissues as well as creating a fascial window as a means of establishing communication with deeper lymphatics.

Thompson's procedure (1962) involved advancement of de-epithelialized dermal flaps into muscles in the hope of drawing lymphatics into the deep circulation.⁶⁶ The most severe of these procedures was the Charles procedure (1912),⁶⁷ which involved the circumferential excision of all involved skin and subcutaneous tissue before covering the areas with split-thickness skin grafts. Proponents who quoted symptomatic improvement justified the cosmetically shocking results, but these procedures are now rarely performed.

Liposuction

As lymphoedema becomes chronic, the volume differential between limbs may be increasingly due to fatty deposition rather than fluid accumulation,⁶⁸ although the rate of this transition from fluid to fatty excess appears to vary greatly.

In order to treat fat deposition within a lymphoedematous limb liposuction techniques have been utilized. An assessment of the affected limb is essential to determine the fat to fluid ratio within the swollen limb. If fluid dominates then decongestive lymphatic therapy (DLT) or microsurgical bypass techniques should be considered. If the excess volume is due to fatty deposition rather than excess interstitial fluid, then liposuction should be considered.

The excessive growth of adipose tissue associated with lymphoedema led to the proposal that lymph is adipogenic, but there has been limited evidence to support this until now. A landmark study from Harvey *et al.* described a mouse model of lymphoedema that progresses to obesity.⁶⁹ They have suggested that a factor normally present in chyle or lymph cooperates with insulin to drive adipocyte differentiation.

Non-pitting, non-fibrotic lymphoedema treated with liposuction has resulted in some excellent volume reduction outcomes.⁷⁰ Hakan Brorson from Sweden is a leading proponent of this and is renowned for his regime of lymphoedema management. This involves an initial intensive course of compression, using custom-made garments, which is termed 'controlled compression therapy'. Liposuction is undertaken under tourniquet control, with the aim of removing all excess tissue from the arm and this is followed by maintenance compression therapy in the same manner as before. Although labour intensive, this regime has shown dramatic reductions in volume. One prospective study combining liposuction and compression therapy in this manner showed oedema reduction figures of 104% compared with 47% for controlled compression alone.⁷¹ It has also been shown that the aspirate removed in this manner under tourniquet control contains up to 93% adipose tissue.⁷²

There is concern that the instrumentation of the surgery further damages the lymphatics of the limb, although lymphoscintigraphic studies have shown that it may not further decrease the already impaired lymph transport capacity.⁷³ However, concerns that the intervention may worsen the fibrosis and so exacerbate the condition have not been fully realized.

Liposuction does not address the cause of the lymphoedema, but simply reduces the volume of the limb. It cannot be used in

isolation but requires the use of lifelong maintenance compression therapy, which is a significant disadvantage for some.

Reconstructive procedures

These techniques involve the use of a conduit (usually lymphatic collector or an interposition vein) to restore lymphatic continuity in lymphoedema resulting from an anatomical interruption. The volume reduction achieved in some cases is reported to reach a level of 80% and the transport index, as demonstrated by imaging methods, has shown an improvement of 30%.⁷⁴ The technique is said to imitate the normal physiology and has shown long-term patencies of more than 10 years.⁷⁵

Autologous lymphatic grafts used to bridge obstructed lymphatic segments in this manner may be useful in some cases, but data are lacking. The method necessitates the harvest of the conduit from a healthy limb, and there are concerns regarding the donor site morbidity that need to be addressed in further studies. It has also been shown that LVAs are less susceptible to thrombosis than lympholymphatic where the pressure within the lymphatics is very low.⁷⁶

Physiological/derivative procedures

The pathogenesis of lymphoedema is related to several physiological factors including increased lymph load, lymphatic obstruction, the number and pattern of lymphatic/lymphaticovenous (reduced compensatory capacity) communications and lymph vessel obliteration. Physiological surgical strategies for lymphoedema are predicated on these principles of pathogenesis in an attempt to restore physiological lymphatic function.

Omental transfer has been used whereby a pedicled portion of omentum is transposed to bridge an area of lymph vessel hypoplasia causing obstruction to the affected limb drainage but omentum lymphatics were found not to generate sufficient collateral drainage. Bridging procedures, initially popularized in the setting of lymphoedema by Sir Harold Gillies in 1935,⁷⁷ have generally failed because lymphatic communication was not achieved. However, the enteromesenteric flap was initially thought to be a breakthrough in the lymphoedema management.⁷⁸ This bridging procedure required covering transected iliac or inguinal nodal basins with a segment of ileum. However, the fact that it involved major surgery and carried the risk of small bowel obstruction means that such procedures have now been abandoned.

Lymph node transfer

The surgical technique of transplanting a healthy, vascularized lymph node into a lymphoedematous limb was initially investigated in the rat⁷⁹ and canine models.⁸⁰ Superficial lymph nodes and perinodal tissue were moved as a free vascularized transfer and this resulted in reduction of the lymphoedematous legs compared with controls. Performing simultaneous lymphaticolymphatic anastomoses to re-establish lymphatic circulation was shown to convey no benefit, and it had previously been

shown that lymph glands will spontaneously re-establish afferent and efferent continuity following division if the vascular supply is retained.⁸¹

Subsequent to these animal studies, there have been a number of *in vivo* studies suggesting that lymphoedema can be treated by lymph node transfer. In order to minimize donor leg lymphoedema, superficial inguinal lymph nodes located along the superficial circumflex iliac artery (SCIA) were selected for transplantation. These more external superolateral nodes had been previously shown to receive drainage from the lower limb in a minority of cases. Corinne Becker and colleagues showed that these nodes based on the SCIA drained mainly the lymph from the abdominal wall and could be safely transplanted with a skin island supplied by the same vessel.⁸² This group has published modest improvement in cases of post mastectomy⁸³ and primary lymphoedema⁸⁴ with this technique, although others have struggled to produce similar results. What is not clear is whether the transplanted node confers benefit in isolation or whether it is the axillary scar release and vascularized transfer of tissue that confers benefit.

The recipient site for lymph node transfers is usually the affected nodal basin, so as to re-establish drainage routes so far as is possible. However, recipient vessels for anastomosis can be challenging in the setting of a scarred and possibly irradiated nodal basin. Hence other options for recipient sites have been sought, such as the wrist, which has yielded reasonable improvements in limb size and lymph drainage.⁸⁵ Although this technique resulted in statistically significant mean reduction rates there are concerns regarding the cosmetic outcomes of such transfers.

Nodal transfers and autologous breast reconstruction with the deep inferior epigastric artery perforator (DIEP) flap have been performed simultaneously. The concept of performing a breast reconstruction at the same time as attempting to manage the lymphoedema is certainly an attractive one and has had success.⁸⁶ However it may be that breast reconstruction itself has an impact on lymphoedema.⁸⁷ A review of 574 cross-matched patients showed that patients who did not undergo reconstruction (implants and autologous in equal numbers) were significantly more likely to develop BCRL.⁸⁸ Further evidence for this comes from a recent large study wherein breast cancer patients who underwent modified radical mastectomy with immediate autologous breast reconstruction had a significantly lower incidence of lymphoedema than those in the non-reconstruction group.⁸⁹

To date, the technique of lymph node transfer has been the source of some controversy. The suggestion that the transplanted node encourages lymphangiogenesis (development of new lymphatic connections) is countered by the argument that this may happen even in the absence of a transplant. Hence the mechanism as to the success of the technique is unclear, as is the optimal recipient site for the transplantation (axilla/wrist etc.), whether a skin flap is necessary in addition to the node itself and whether a lymphatic anastomosis is required.

Donor site morbidity is perhaps the largest area of controversy for this technique. Given the fact that removing a single lymph node from a lymphatic basin carries a risk of lymphoedema (sentinel node biopsy), it follows that harvesting the node for transplantation must also carry the same risk. Lymphoscintigraphy has shown that postoperative lymphatic vessel function of the donor limb may be impaired.⁹⁰ A more recent review from Paris showed significant complications in 38% of patients who underwent autologous lymph node transplantation.⁹¹ These were not only chronic lymphoedema in the donor limb, but also lymphocoeles, a hydrocoele requiring surgery and persistent donor site pain.

It is well established that the development of the lymphatic system is mainly regulated by the VEGF-C/VEGF-D/VEGFR-3 signalling system. Knowledge of the different molecules involved in lymphangiogenesis has rapidly accumulated in recent years. It has been suggested that combining growth factor therapy with vascularized lymph node transfer will result in increased lymphangiogenesis around the implanted node and therefore improved drainage of the limb. However there are a number of potential technical and oncological obstacles. The role of growth factors in cancer upregulation is one such concern, as numerous studies have also shown a direct correlation between VEGF-C and VEGF-D expression in human cancers and tumour metastasis, suggesting that lymphangiogenesis has an important role in promoting the metastasis of human tumours.⁹²

There have been some promising early results from animal studies in Finland using such growth factor therapy in combination with lymph node transfer.^{93,94} Growth of lymphatic channels around the transferred nodes was seen and results from initial clinical trials are keenly awaited.

Lymphaticovenous anastomosis

The concept of lymphatic reconstruction by connecting obstructed lymphatic channels onto veins in order to improve drainage remains an attractive one.

This lymphaticovenous anastomosis (LVA) surgery was first performed in the late 1960s^{95,96} with perhaps the most well-known initial work published by O'Brien and colleagues.⁹⁷ Early studies proved the technique to be advantageous for sufferers from postsurgical lymphoedema, although results were modest and variable.⁹⁸ With the advent of recent advances in supermicrosurgery, such techniques have become more popular.

One prospective study showed that performing this LVA procedure on patients with BCRL could result in symptom improvement for 95% of patients and a quantitative improvement in 65%.⁹⁹ An impressive series of results from an Italian team found that, of those patients followed up, 85% were able to discontinue the use of conservative measures such as compression sleeves. With an average follow-up of more than 10 years an average reduction in excess volume of 69% was achieved.¹⁰⁰ A large series from Poland has also shown benefit from lymphovenous shunt procedures using the femoral vein, although this recipient vein is not widely used.¹⁰¹

Many researchers have had conflicting results, with significant variation between patients in terms of the amount of reduction in size of the limb that occurs. Indeed some small series have had very limited success.¹⁰² It appears, therefore, that patient selection is crucial in order to select those patients who will benefit from surgery. This may be due to a number of factors, including the severity of lymphoedema-related fibrosis and the availability of viable lymphatic vessels.⁹⁹ It is for this reason that imaging techniques such as indocyanine green lymphography have been employed.¹⁰³

Dynamic imaging techniques are used to delineate functioning lymphatics to be used for anastomosis, and this combination of imaging with surgery has had more favourable results.¹⁰⁴ Once the lymphatics and venules have been isolated, the anastomoses are generally performed using 12/0 Ethilon on a 50 μ m needle, using supermicrosurgical instruments (Figures 47.2, 47.3 and 47.4).

One series combining this technique with ultrasonography to identify veins, reported 78% of patients having at least a 50% reduction of the proximal, distal or whole limb.¹⁰⁵ Further studies have refined the technique by using shorter incisions,¹⁰⁶ performing anastomoses at multiple sites simultaneously¹⁰⁷ and using intravascular stents.¹⁰⁸

If there is a dermal backflow pattern whereby no linear lymphatics can be found, some surgeons have advocated the use of predictive lymphatic mapping, whereby the linear pattern of lymphatics on the unaffected limb are mapped onto the affected limb, and this technique is used to guide the site of surgical incision for lymphatic surgery.¹⁰⁹

Several reports have described the effect of such microlymphatic surgery on peripheral lymphoedema, but details of the exact treatment protocols used are limited. Many of the series published are thought to combine DLT/complex decongestive therapy (CDT) with LVA bypass surgery and thus the separate net effects of surgery versus DLT/CDT have not always been made clear. A recent publication sought to address this and considered that oedema can be reduced using DLT/CDT before surgery. The authors evaluated the net effect of DLT/CDT and then evaluated the net effect of subsequent LVA surgery.¹¹⁰ The approximate reduction achieved by preoperative DLT/CDT in affected limbs was significantly better than that achieved by surgery, but the net effect of LVA surgery on volume reduction was confirmed. They also indicated that the surgical effect should include not only the amount of oedema reduction but also changes in skin stiffness, subjective symptoms and the frequency of cellulitis. The series included some patients who required no DLT/CDT after surgery and many for whom the class of pressure in the elastic stockings could be decreased. Both of these outcomes should also be evaluated when considering the outcomes of any surgical technique.

The anatomical configuration of the lymphaticovenous anastomosis varies between series (i.e. whether to perform an end-to-end or end-to-side anastomosis and whether to use the proximal or distal ends of the lymphatic and venule). The

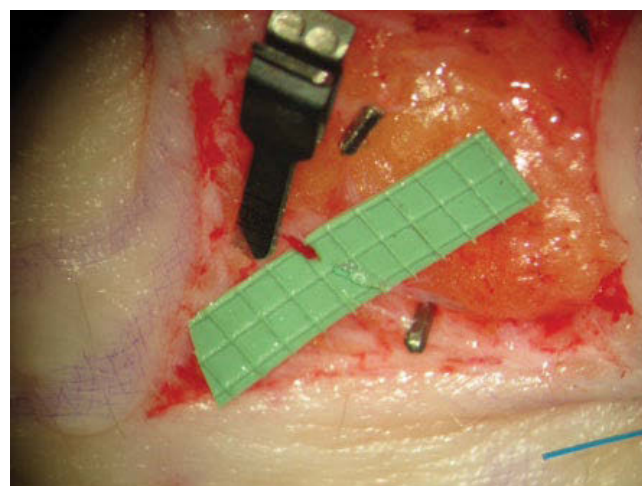


Figure 47.2 Lymphatic and vein prior to anastomosis, with 1 mm gradations on background.

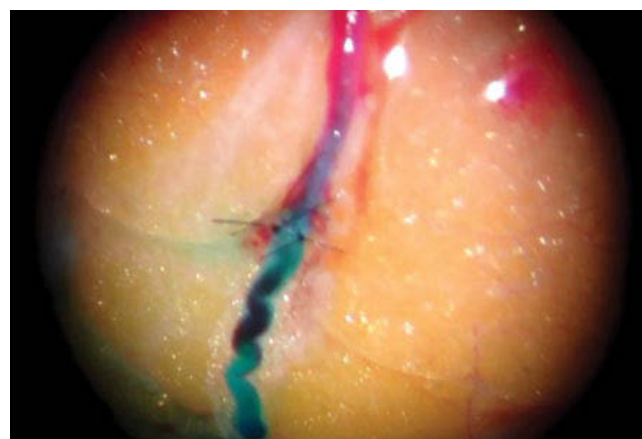


Figure 47.3 Lymphaticovenous anastomosis immediately after completion.



Figure 47.4 Lymphaticovenous anastomosis 2 s later, showing flow of dye from lymphatic into vein.

majority of series comprise end-to-end anastomoses between the distal stump of the lymphatic and the proximal stump of the vein but good results have also been achieved with multiple alternative configurations.^{108,100}

The number of anastomoses performed may also be relevant to the success of the surgery. One large retrospective review has suggested a relationship between the numbers of anastomoses and volume reduction achieved,¹¹¹ but not all centres have shown similar findings. However the patency rate of the anastomoses decreases over time and thus it may be reasonable to suggest that many anastomoses are needed to achieve good long-term outcomes.

The timing of surgical intervention is critical, but there are some conflicts between pathophysiology and clinical indication. The concept of undertaking LVA at a much earlier stage to restore some of the disruption/blockage of superficial lymphatics is based on the physiological rationale that this will reduce increased hydrostatic pressure in other parts of the lymphatic system and promote absorption of fluid back into the lymphatic vessels. However, performing LVA surgery at the time of lymph node clearance in a non-selective fashion would be difficult to justify as only about one-quarter of patients would be expected to subsequently develop lymphoedema. On the other hand, the procedure may be less effective when done following onset of established lymphoedema and of course this entails an additional surgical procedure.

It has been proposed that LVA surgery should be performed at the same time as axillary node clearance (ANC), in order to attempt to prevent the onset of lymphoedema in the first place. The LYMPHA technique, conceived by Corradino Campisi in Genoa, is such a technique and consists of performing LVA between the arm lymphatics and collateral branches of the axillary vein at the same time as axillary dissection.¹¹² In a recent series from this group, one cohort of patients had ANC and LVA during the same operation, whereas the others had the best conservative treatments.¹¹³ The study has short follow-up and small numbers, but the results showed that 30% of the control group developed lymphoedema whereas only 4% of the treatment group were affected.

The surgical technique used by this group varies slightly from the standard LVA. Usually, the divided end of a single lymphatic is anastomosed to a similar-sized venule. Campisi's group insert multiple lymphatics into the divided end of larger veins, using their 'sleeve technique'. Although there are strong data to support LVA surgery on its own, some find it difficult to justify prophylactic LVA surgery at the time of an axillary clearance if only 20–30% of those patients treated would have developed lymphoedema.

A technique designed to mitigate the damaging effects of axillary surgery is axillary reverse mapping. The lymphatic drainage of the arm is mapped with blue dye prior to surgery, so that the identified lymphatics can be preserved.¹¹⁴ However, to truly preserve the lymphatic flow of the arm, not only the afferent lymphatics but also the lymph nodes belonging to the

arm lymphatic pathways must be preserved. There remains controversy regarding the oncological safety of preserving lymph nodes during an axillary clearance in this manner. Similarly, the identification of efferent lymphatic pathways in order to bypass the axilla is hindered by the fact that these join the common lymphatic pathways draining the breast. For these reasons further studies are required to confirm the feasibility and long-term efficacy of this technique.

Lymphangiosarcoma

Lymphangiosarcoma is a rare tumour that presents with localized swelling and multiple, subcutaneous nodules occurring in chronic lymphoedematous limbs. It is an aggressive tumour that is rapidly fatal unless extensive surgery is performed.

Summary

Both excisional and lymphatic reconstruction surgical techniques have been employed to treat lymphoedema but published results have been variable and difficult to assess in terms of efficacy due to small numbers and limited statistical analysis.

Despite the exciting potential of combining imaging with lymphatic diversion surgery, there is a clear need for an unbiased scientific assessment of these techniques with larger randomized studies. Research is needed to understand which patients are most likely to benefit from surgical techniques and the optimal timing. This can only be achieved with standardization of diagnosis, classification, assessment and outcome measures with international consensus.

Clearly, it would be preferable to prevent development of more established forms of lymphoedema by intervening at an early stage to impact on pathogenic mechanisms.

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PART VII

Upper limb

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CHAPTER 48

Hand examination and investigations

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The medical student adage of 'look, feel and move' applies to hand examination at all clinical levels. Having been put in harm's way more than any other part of the body, this extension of our mental will into the external world doubles up as a tactile organ as well. A thorough knowledge of its anatomy and biomechanical principles assists the clinician in narrowing down the pathology. Recognition of patterns of pathology and injury are as important in examining the hands as they are in any other form of clinical examination.

Examination principles: 'look, feel, move'

Inspection ('look')

Prior to the physical examination a medical history should have been taken. The inspection is an important part of the examination as the appearance may give valuable information prior to the palpation of the hand and wrist, especially in cases of pain or discomfort. In this phase patients – especially children – will be more relaxed. It is important to compare the involved hand and/or wrist with the contralateral side.

The inspection should include:

- colour,
- deformity, including alignment,
- swelling,
- muscular appearance (atrophy, hypertrophy, spasm),
- trophic changes (e.g. sweating, hair growth),
- skin creases.

Specific appearances should be noted.

- For rheumatoid arthritis a well-known pattern can be seen in advanced disease. This includes radial deviation of the wrist and ulnar deviation of the metaphalangeal joints with swollen joints and dorsal aspect of the wrist in case of active disease. Attention should be given to potential loss of extension of the thumb and the fingers.
- For nerve palsies, specific postures can be seen: 'dropping hand' indicates radial nerve palsy with loss of wrist and finger extensors.

- 'Preacher's hand' is caused by pathology of the proximal median nerve. The index and middle finger are in a relative extended position, whereas the ring and little finger are in more flexed position. The flexor digitorum of the ring and little fingers are innervated by the ulnar nerve and are the only functioning long finger flexors.
- 'Claw hand' or 'spinster's claw' is an abnormal hand position that develops due to pathology of the ulnar nerve. The hand will show hyperextension of the metacarpophalangeal (MCP) joints and flexion at the distal and proximal interphalangeal (IP) joints of the 4th and 5th digits (ring and little fingers). The clawing will become most obvious when the person is asked to extend their fingers. The pathogenesis is a partial or complete denervation of the two ulnar lumbricals as well as the interosseous muscles to these fingers. The ulnar paradox is so called because the more distal ulnar palsy sparing the flexor digitorum profundus (FDP) supplies to the ring and little fingers is more pronounced.
- Dupuytren's contracture can present itself as a small, hard nodule at the base of the finger. It predominantly tends to affect the ring and little finger as puckering and adherence of the palmar aponeurosis to the skin. Progression may lead to a flexion contracture of the MCP and IP joints of the affected digits.
- The clinical presentation of osteoarthritis may vary from swollen joints to abnormal joint position.

Palpation ('feel')

The first orientation should be the evaluation of:

- temperature,
- oedema,
- tenderness, pain,
- passive (joint) stiffness,
- presence of skin lines,
- stability of joints (specific tests will be discussed later in this chapter).

Range of motion ('move')

Evaluation should include:

- measurement of the active range of motion of the hand and wrist joints;

- relation of movement with pain or discomfort;
- presence of snaps, clicks and clunks;
- specific tendon tests (see below Clinical tests);
- specific nerve tests (see below Clinical tests).

Clinical tests

Tendon testing

The Bouvier test

The Bouvier test or manoeuvre is generally used to assess the nature of a claw hand. This test was named after the French orthopaedic surgeon Sauveur Henri Victor Bouvier (1799–1877). In 1851 he described¹ the observation of one of his patients, the carpenter Henri Marlier, who was suffering from a temporary, effort-induced, left-sided ulnar palsy, that passive flexion of his 4th and 5th MCP joint enabled him to actively extend the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints in these fingers. A comparable phenomenon was later described by Beevor² in 1903 and even later by several other distinguished hand surgeons.³ The biomechanical basis of this test is that blocking extension of the MCP joint increases the tension in the finger's extrinsic extensor mechanism up to a level that, when besides the ulnar nerve palsy all other systems are working properly, it is able to fully extend the PIP and DIP joints. In this situation the test is considered to be positive. In a negative test additional factors such as insufficiency (rupture, adhesion, lengthening) of the central slip or a volar subluxation of the lateral bands also play a role in the origin of the claw hand.



(a)



(b)

The Bunnell test

The Bunnell test assesses the length of the intrinsic muscle-tendon units to the fingers. The test was named after the American surgeon Sterling Bunnell (1882–1957), one of the founding fathers of hand surgery and the first president of the American Society for Surgery of the Hand (ASSH). This test is also named the Finochietto test, the Finochietto–Bunnell test and the Bunnell–Littler test. The underlying biomechanical phenomenon was first described⁴ in 1920 by the Argentinean surgeon Ricardo Finochietto (1888–1962), who later became the head physician for Eva Perón, the wife of Juan Perón the tenth president of Argentina. Later descriptions are by Parkes in 1945⁵ and Bunnell in 1948,⁶ albeit apparently without knowledge of and therefore reference to their predecessor(s).

The biomechanical basis of this test is that when the MCP joint is held in extension, the tension in the MCP joint capsule and the aforementioned tendons as well as the more distal portion of the extensor apparatus increases, allowing less flexion of the PIP and DIP joints. Conversely, when the MCP joint is held in flexion, the tension in the MCP joint capsule and the aforementioned tendons as well as the more distal portion of the extensor apparatus decreases, allowing more flexion of the PIP and DIP joints (Figure 48.1).

The Boyes test and the Elson test

The former test was named after the American surgeon Joseph Henry Boyes (1905–1995), one of the founding fathers of the ASSH, its first secretary–treasurer and its ninth president.⁷ In this test the PIP joint is held passively extended and an attempt is made to flex the DIP joint (Figure 48.2a). In a normal finger

Figure 48.1 (a) Bouvier test. (b) Bunnell test.

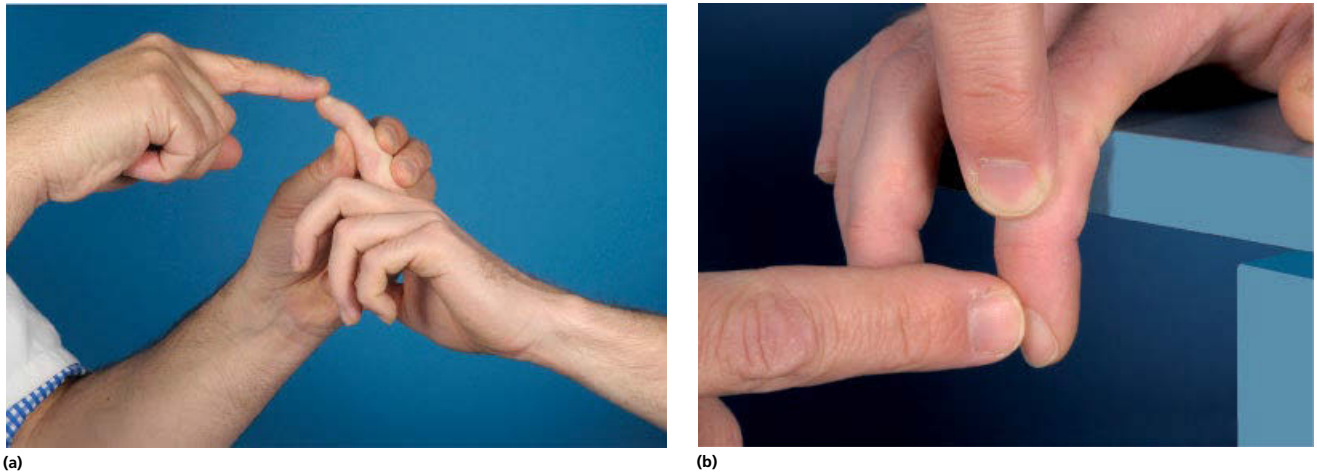


Figure 48.2 (a) Boyes test. (b) Elson test.



Figure 48.3 Finkelstein test.

this is possible and the test is negative. When the central slip has been ruptured, the tension in the lateral bands is increased and DIP flexion becomes increasingly difficult. This test is not very sensitive and only becomes positive later when the proximal part of the ruptured central slip has become retracted and adherent to the surrounding tissues.⁸

In more acute situations, when the Boyes test is less sensitive, other tests can be used, of which the Elson test was found the most reliable.⁹ This test is named after the British orthopaedic surgeon Reginald A. Elson MB BCh Cambridge, FRCS (1930). In this test the PIP joint is flexed 90° over the edge of a table, and the patient is asked to extend the joint against resistance. In a healthy finger the examiner can feel the pressure of the extending PIP joint and the DIP remains flail. In case of a central slip lesion, the possibility of active PIP joint extension is severely diminished and the lateral bands remain proximal and consequently taut, rendering the DIP joint rigid with a tendency to extension (Figure 48.2b).

The Finkelstein test

The Finkelstein test for de Quervain's disease was first described by and named after the American surgeon Harry Finkelstein (1865–1939). In the original implementation of this test the

examiner grasps the patient's thumb and deviates the hand ulnarly. The test is positive when this movement elicits excruciating pain over the tip of the radial styloid. Another test for de Quervain's disease was described earlier by the German surgeon Erich Eichhoff.¹⁰ In the Eichhoff test the patient's thumb is placed in the patient's hand and the other fingers closed around the thumb, holding it tightly. Then the examiner bends the hand severely in ulnar abduction. The test is positive when an intense pain is experienced on the styloid process of the radius. Often the Eichhoff test is assumed to be the Finkelstein test. This is probably due to the fact that both are being mentioned in Finkelstein's original paper in which the quoted description of the Eichhoff test takes up an elaborate 236 words while the description of Finkelstein's own test takes up only 19 words and is therefore easily overlooked.

A disadvantage of both Eichhoff and Finkelstein tests is that they can elicit pain in healthy subjects and therefore have a high sensitivity but a low specificity. This is the reason that nowadays more subtle versions of the latter are being used with a more staged loading of the involved first dorsal extensor compartment and tendon (Figure 48.3).¹¹ Quantitative information about the sensitivity, specificity or the positive or negative predictive value of the Eichhoff and Finkelstein tests or their more sophisticated offspring is not yet available.

Nerve (specific nerve testing)

Many techniques for evaluating sensibility have been described. Interestingly, even today consensus is lacking. How do we test sensibility in the hand? This may appear an easy question to answer. However, simplicity may deceive, in many hand surgery departments different tests scoring gnostic modalities are being used. If we unravel the problem, it can be brought down to the following: there are a number of requirements for adequate testing.¹²

- The test should have a quantitative outcome. Simply asking 'Do you feel this, does it feel different from over here or different from the same area of the other hand?' is not sufficient. This may all seem exaggerated, but we are often confronted with information about the sensibility of the hand that resembles this. It can be even worse: 'We already applied local anaesthesia, but I think that the sensibility was normal'.
- It should be a test that correlates with function of the hand. For example, the fact that touch is being perceived does not mean that that sensibility is useful in daily living and in the workplace.
- It should be tolerable for the patient. Testing sensibility with a needle is cruel to the patient, especially if the sensibility is not abnormal! In children, introducing such an instrument will

directly end any cooperation and repeated tests will not be accepted.

- The test should be reproducible in time and it should be reproducible between different technicians.
- Especially nowadays, the test should be affordable and it should not be time consuming.

Tests can be divided into innervation density and touch threshold tests. The *innervation density test* scores the number of available and working axons and receptors, whereas the *threshold test* gives information on the required force of the stimulus to elicit a response by the mechanoreceptors. Innervation density is tested by static and moving two-point discrimination tests. Temperature and pain are seen as protective sensibilities: temperature thresholds can be measured and pain is scored on a visual analogue scale to demonstrate the severity. The use of these different modalities is demonstrated by evaluating the results of median nerve reconstruction as an example. Numerical grading scales to evaluate the function of sensory and motor nerves have been described. Different aspects of combined sensory and motor recovery will be scored as a number. Its applicability is demonstrated by comparing pre- and postoperative results of ulnar nerve decompression at the elbow.¹³



(a)



(b)



(c)



(d)

Figure 48.4 Hoffman/Tinel's examination. (a,b) In carpal tunnel syndrome. (c) In cubital tunnel syndrome. (d) Sensory branch of radial nerve.

Tinel/Hoffmann sign

The Tinel sign is named after the French neurologist Jules Tinel (1879–1952). Both he and, only months before him, the German physiologist Paul Hoffmann (1884–1962) described the generation of paraesthesias radiating into the area of distribution of a particular nerve due to putting pressure on it or tapping on it respectively. The hypothesis is that the sign occurs because the incompletely myelinated sprouting fibres of damaged or compressed nerves are increasingly sensitive to mechanical stimuli.

The Hoffmann/Tinel's test is a specific test for carpal tunnel syndrome and can be used as indication for medical management (Figure 48.4). The Hoffmann/Tinel sign is positive in 51–54% of cases of symptomatic carpal tunnel syndrome and is 80% positive in pronator syndrome. No numbers are known for cubital ulnar neuropathy.^{14–17}

For carpal tunnel syndrome:^{18–24}

- Sensitivity: 23–73%
- Specificity: 87–94%
- Positive predictive value: 0.75
- Negative predictive value: 0.41
- Level of evidence (Oxford Centre for Evidence-based Medicine): 1c.

Phalen's test

Phalen's test is named after the American orthopaedic surgeon George S. Phalen (1911–1998) and is used to aid in the diagnosis of carpal tunnel syndrome. Phalen's test consists of passive flexion of the patient's wrist for 15–60 s. The test is considered positive when the patient experiences tingling or numbness in the median nerve distribution. This test increases carpal tunnel pressure (CTP), and therefore the pressure on the median nerve, which is believed to be one of the causes of carpal tunnel syndrome. The increase in CTP during Phalen's test is probably caused by a decrease of the cross-sectional area of the carpal tunnel.

The reverse Phalen's test consists of passive extension of the patient's wrist for 15–60 s. The test is considered positive when

the patient experiences tingling or numbness in the median nerve distribution. The increase in CTP in this test is higher than the normal Phalen's test. A possible explanation for this is that wrist extension not only results in a decreased cross-sectional area of the carpal tunnel at the pisiform level but also a constriction of the proximal end of the carpal tunnel due to the incursion of the flexor digitorum superficialis and flexor digitorum profundus muscle bellies.

Phalen's test is positive in 48–87% of cases of symptomatic carpal tunnel syndrome, with a mean time of 18.4 s.^{24–26}

- Sensitivity: 10–80%
- Specificity: 55–86%
- Positive predictive value: 0.78
- Negative predictive value: 0.49
- Level of evidence (Oxford Centre for Evidence-based Medicine): 1c.

The Phalen's manoeuvre has been found to be more sensitive than Tinel's sign.²⁷

Reverse Phalen's test

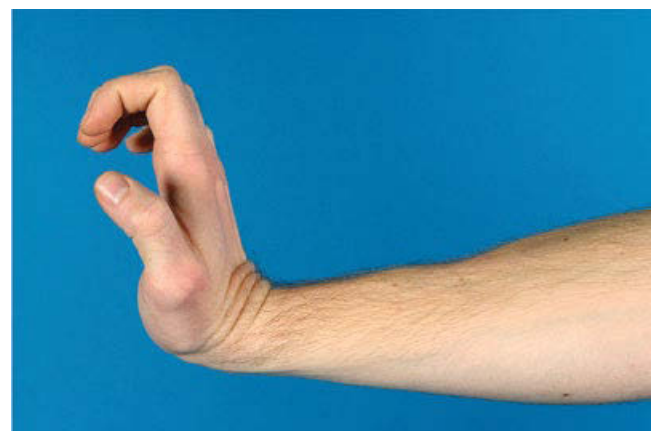
This test can be performed by having the patient maintain full wrist and finger extension for 2 min (Figure 48.5). The reverse Phalen's test significantly increases pressure in the carpal tunnel within 10 s of the change in wrist posture and the CTP has the tendency to increase throughout the test's duration. In contrast, the change in CTP noted in the standard Phalen's test is modest and plateaus after 20–30 s.

Froment's sign

Froment's sign is used to evaluate the motor function of the ulnar nerve. The patient is asked to hold a piece of paper between the volar side of the thumb and the radial side of the index finger. The examiner pulls on the paper while instructing the patient to hold on it. Patients with adequate function of the first dorsal interosseous and adductor pollicis muscles will keep the IP joint of the thumb in an extended position. In case of weakness, the patient will need to flex the IP joint to be able hold on



(a)



(b)

Figure 48.5 (a) Phalen's test. (b) Reverse Phalen's test.

to the paper using the flexor pollicis longus (and also (hyper) extending the thumb MCP joint to stabilize it: Jeanne's sign).

Jeanne's sign

Jeanne's sign is used to evaluate the motor function of the ulnar nerve. In case of pathology of the ulnar nerve, the patient needs to hyperextend the MCP joint of the thumb to create lateral stability to compensate for the thumb adductors.

Wartenberg's sign

This is used to evaluate the motor function of the ulnar nerve. The patient is instructed to keep the fingers extended. If the patient has pathology of the ulnar nerve, the little finger deviates ulnarly because of dysfunction of the third palmar interosseus and the ulnar directing force of the extensor digiti minimi. This is not present in all the patients. This is related to the eccentric insertion and pull of the extensor digiti minimi onto the 5th digit abducting the digit unopposed by intrinsic hand adductors.

Scratch collapse test

This is a new diagnostic test for carpal tunnel syndrome and cubital tunnel syndrome in which the examiner scratches the patient's skin lightly over the area of nerve compression, while the patient performs resisted shoulder external rotation bilaterally.

The test is performed as follows: patient facing examiner, arms adducted, elbows flexed, hands outstretched and wrist neutral (Figure 48.6). The examiner gently pushes against both the forearms, asking the patient to sustain steady resistance. With the fingertips, the examiner scratches/swipes the skin overlying the potentially compressed nerve. A positive test is recorded if the patient demonstrates a loss of resistance in the affected side after 'scratching' the respective tunnel. Loss of resistance is brief, with the patient regaining strength essentially immediately.

The positive predictive value for all tests was high for carpal tunnel syndrome, with the scratch collapse test having the highest value (99%), followed by wrist flexion/nerve compression (98%) and Tinel's (96%). Negative predictive values were also highest for the scratch collapse test (73%) followed by wrist flexion/nerve compression (65%) and Tinel's (59%). For cubital tunnel syndrome, the scratch collapse test had the highest positive predictive value (99%), followed by Tinel's (98%), and elbow flexion/nerve compression (96%). Negative predictive values, however, were highest for Tinel's (98%), followed by the scratch collapse test (86%) and elbow flexion/nerve compression (78%). Data analysis showed that the scratch collapse test was reproducible, with excellent interrater reliability, and had higher sensitivity than the other tests performed, for both carpal and cubital tunnel syndromes. The accuracy rate was 82% for diagnosing carpal tunnel syndrome and 89% for cubital tunnel syndrome.²⁸

Traditional nerve tests

Traditional tests are also divided into innervation density and touch threshold tests.

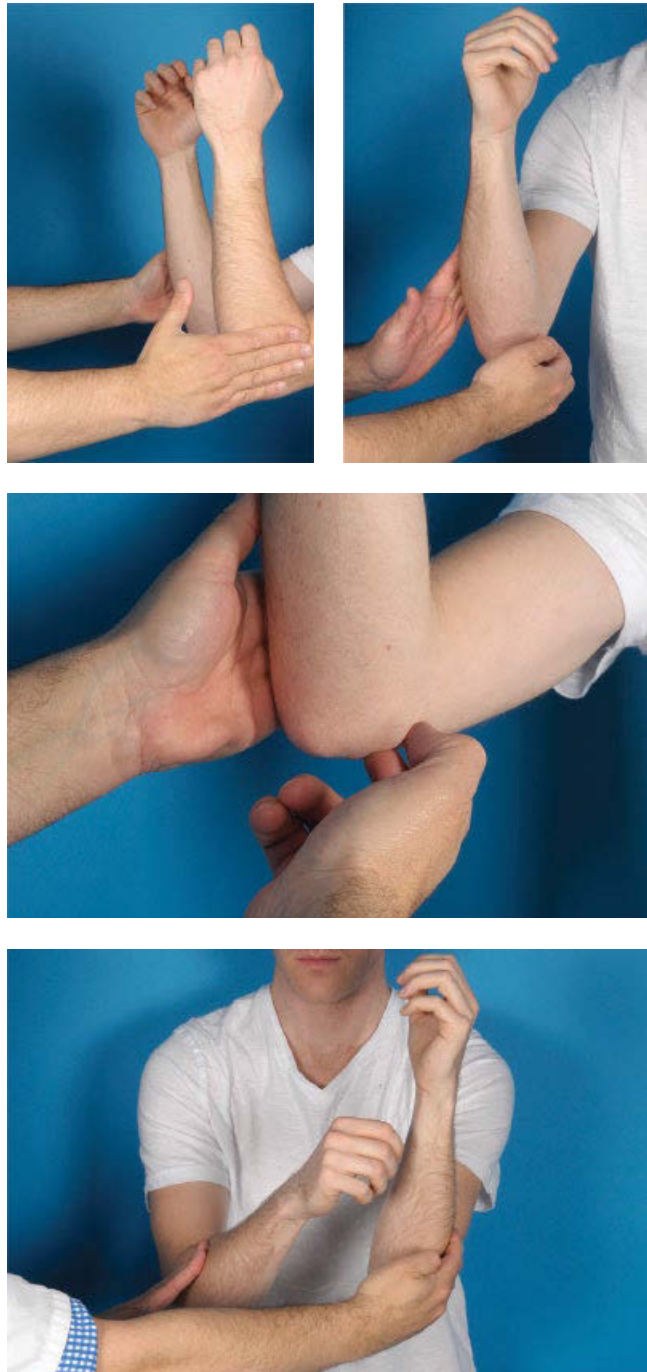


Figure 48.6 Scratch collapse test.

One-point discrimination test (Von Frey hairs)

The cutaneous threshold can be evaluated by the static and moving one-point discrimination tests and vibration. A static one-point discrimination test can be performed by either Semmes Weinstein filaments or quantitative sensory testing (QST) (Figure 48.7). Other tests that are available are combined tests that will score both sensory and motor function, for example the pick-up test and the Moberg recognition test.

Semmes Weinstein filament test, and its variant the Weinstein Enhanced Sensory Test (WEST, e.g. WEST-hand), present nylon monofilaments of roughly the same length (38 mm) but of varying diameters. The diameter and length are used to control the force applied. Whereas Dr Weinstein used three-digit numbers to reflect the force of the Semmes Weinstein aesthesiometer (three-digit number equals the common log of the force measured in tenths of a milligram), the WEST aesthesiometers (also created by Weinstein and group) use grams (e.g., 0.70 g) to describe the force.

There is a low correlation between pressure sensitivity and spatial threshold (divergent validity) and between pressure threshold and cortical area. However, the Semmes Weinstein monofilaments correlate with object recognition time and object identification.^{29–32}

- Level of evidence (Oxford Centre for Evidence-based Medicine): 3a.

Two-point discrimination test

This test will demonstrate the innervation density. Originally described by Weber and modified by Moberg, it will evaluate the slow-adapting fibres. In compressive neuropathies the reproducibility has been difficult due to bias in the application forces of the two prongs.

The test can be performed as a static or dynamic test. In static two-point discrimination the device will be applied to a small area and will require near-normal sensation to be discerned by the patient. In the moving variation a larger area is tested. This can still be felt after static two-point discrimination is not being detected. If the prongs can only be felt at a wider interval, the innervation density is diminished.^{33–35}

There is a strong correlation between two-point discrimination and object identification. Poor two-point discrimination can be functionally limiting. The cutaneous pressure threshold is

inversely related to static two-point discrimination (regardless of age or presence of compression or neuropathy).^{36–40}

Quantitative sensory testing

Quantitative sensory testing (QST) has been described as the result of subjective tests of sensibility that require conscious recognition of the stimulus in contrast to electrodiagnostic testing. It can be used to evaluate thermal, pressure and vibration perception. Advantages of QST over traditional nerve testing are the uniform non-logarithmic scale for one-point discrimination and the known application force for two-point discrimination (see Figure 48.7). The multiplication of the distance between the prongs and the application force can be used as a constant factor (e.g. if the prongs are at the 4 mm distance the force to discern may be 6 g/mm, if the prongs are wider apart, to 6 mm, the application force needed will be smaller: 4 g/mm. However, the multiplication will remain constant.⁴¹

Unfortunately we need more evidence from future studies that the use of QST for the non-invasive assessment and quantification of sensory nerve function is more accurate than conventional tests. Cutaneous pressure threshold measurements with QST had a statistically significant correlation with the small-object subset of the Mayo Dexterity Test and with the object recognition test, demonstrating that QST is a valid tool for evaluating the sensory aspect of hand function. Also QST has a high sensitivity but a low specificity when compared with electrodiagnostic testing for diagnosis of peripheral nerve entrapment.^{42,43}

Moberg pick-up test

This test will evaluate motor and sensory function. The Moberg recognition test has also been described. Both tests will score the handling and recognition of small objects within a certain time frame.

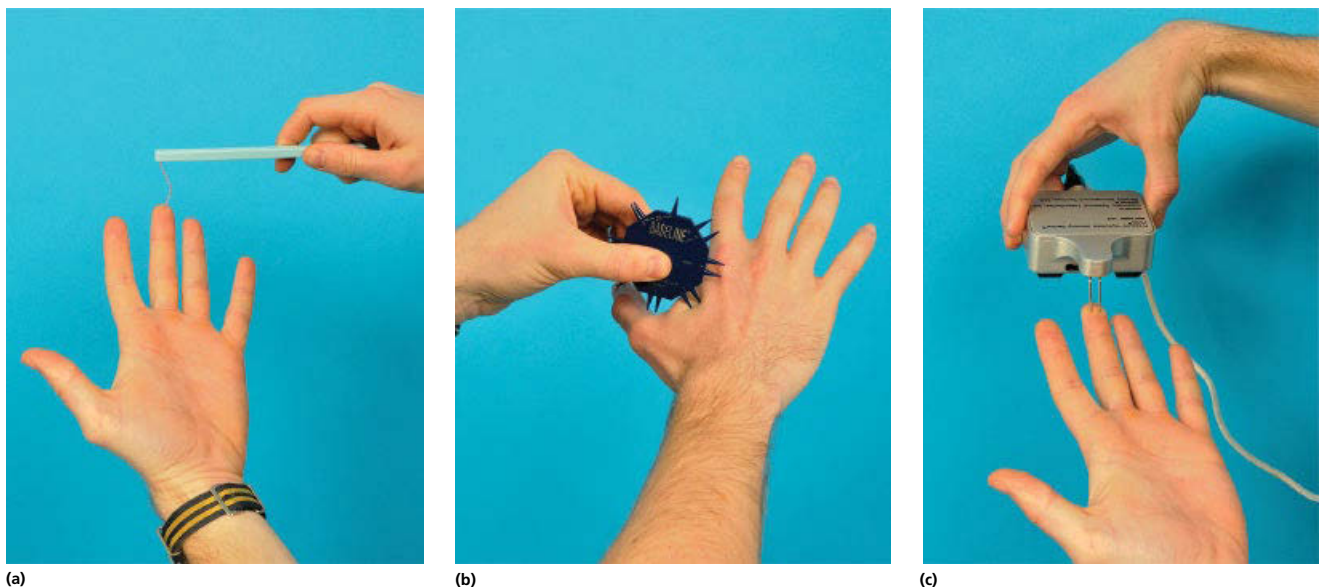


Figure 48.7 Sensibility testing. (a) Monofilament. (b) Disk-Criminator. (c) Pressure-specified sensory device.

In the pick-up test, small objects used in daily life, such as buttons, keys, paperclips, screws, bolts etc., are picked up and placed into a small box. This can be performed with or without visual aid. The times that are needed to complete the tasks are recorded. For the recognition test, the number of objects identified without visual support in a given time frame is scored.³⁵

Electrodiagnostic testing

In 1666 Francesco Redi described the first experiments in clinical neurophysiology. He discovered that a specific muscle in an eel created electricity. It was Luigi Galvani who described in his publication ‘De viribus electricitatis in motu musculari commentaries’ the fact that electricity could cause muscle contraction. It was not until 1960 that electrodiagnostic testing (EDT) testing could be used clinically.

EDT is critical in the diagnosis of many neuromuscular and peripheral nerve problems (Table 48.1).

EDT can be divided into nerve conduction velocity (NCV) and electromyography (EMG). NCV describes the speed with which an electric pulse is transmitted along a sensory or motor nerve, and the strength of the conducted impulse: amplitude height. If two different sites along the nerve or two different nerves are being compared, this can be used to obtain the conduction velocity. If only one site can be recorded for the impulse to travel distally over the nerve, this is called distal latency.

Table 48.1 Main indications and disadvantages of electrodiagnostic testing

Main indications	
Localization of nerve lesion	Radiculopathy
Progression of nerve lesion	Resolution neuropraxia
	Reinnervation following nerve repair
	Progression inflammatory neuropathy
	Status brachial plexus
Evaluation of systemic disease	Peripheral neuropathy
	Drug toxicity
	Neuromuscular dysfunction
	Myopathy
	Inability of patient to communicate
Verification of suspected nerve problem	Suspected malingering
	Martin–Gruber connection
Disadvantages	
Neurophysiology	Large myelinated fibres preferentially recorded
Pathophysiology	Sparing central fibres
	Uneven fibres loss among fascicles
	Incomplete remyelination
Anomalous innervation	Radial sensory nerve to thumb
	Overlap lateral antebrachial cutaneous nerve with radial sensory nerve
	Lateral sural nerve entrapment
	Temperature
Technique	Patient age
	Patient height
	Site along nerve

EMG evaluates the status of innervated muscle through a needle placed into the muscle. A normal muscle will display an M-wave, which is a depolarization with a short interval. If there is no or diminished activity, this suggests a denervated or fibrotic muscle, or both. A normal muscle is electrically quiet, however in denervated muscle spontaneous depolarization will occur, showing fibrillation potentials. These are, in fact, small M-waves.³⁵

In a third of the clinically symptomatic patients for carpal tunnel syndrome, the EDT is normal.

For ulnar nerve compression at the elbow, the results of the EDT do not predict the results of conservative or surgical management.^{44,45}

Neuropathic pain: diagnostic blocks

Diagnostic blocks are used to obtain information about the source of a patient’s pain. As such they differ in principle and in practice from regional anaesthetic blocks. In order to be valid, diagnostic blocks must be precise and target-specific. The involved nerve should receive a field block proximal to the point of maximal pain. Lidocaine (lignocaine) is most frequently used due to its early action and its short duration. We have shown that that a positive nerve block can predict a successful denervation.⁴⁶

Ligament (systematic exam) – scaphoid–lunate interval

Scaphoid shift test (Watson)

This test is used to diagnose insufficiency of the scaphoid-lunate (SL) ligament, scaphoid fracture and radio-scaphoid osteoarthritis. The arm is resting on the elbow. The examiner places his or her index and middle finger on Lister’s tubercle, with the thumb is on the distal pole of the scaphoid (Figure 48.8). The wrist is in a neutral position for flexion and extension. The wrist is in ulnar deviation and is brought to radial deviation. The scaphoid will move from an extended position to a flexed position. If the SL ligament is insufficient, the scaphoid will move dorsally in relation to the scaphoid fossa of the radius. Releasing the pressure on the scaphoid will cause a clunk. If this clunk is painful, a ligament tear is suspected. If the test is painful without a clunk, pathology of the scaphoid or the radiocarpal joint is possible.⁴⁷

Ballottement test

This is used to detect pain over the SL interval indicating a complete or partial rupture or occult ganglion. The examiner fixes the lunate palmar and dorsal with the thumb and index and the scaphoid with the same fingers of the other hand. When moving in opposite directions this will cause translation over the SL interval. The test is positive if pain and/or increased movement is present.

Finger extension test (Schuck test)

This test is used to detect pain over the SL interval, which indicates predynamic instability or occult ganglion. The examiner holds wrist and MCP joints in flexion. The patient is asked to extend the PIP and DIP joints of all fingers against resistance. If pain is elicited, the test is considered to be positive.⁴⁸

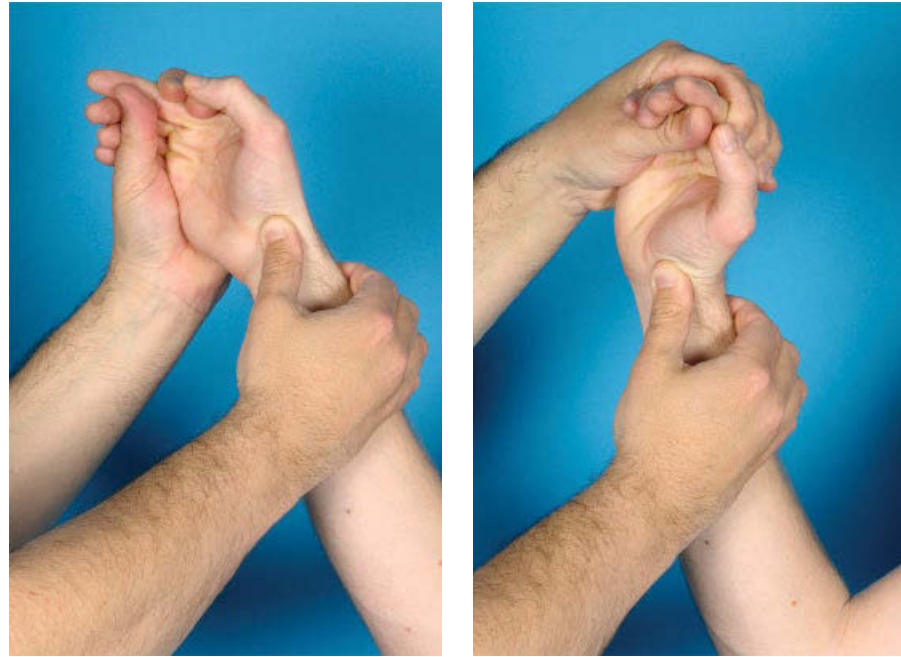


Figure 48.8 Watson test.

Ligament (systematic exam) – lunotriquetral interval

Ballottement test (Reagan's test)

Reagan's test is used to detect pain over the lunotriquetral (LT) interval, indicating a complete or partial rupture or occult ganglion. The examiner fixes the lunate palmar and dorsal with the thumb and index and the triquetrum with the same fingers of the other hand (Figure 48.9 top). When moving in opposite direction this will cause translation over the LT interval. The test is positive if pain and/or increased movement is present.

Lunotriquetral shuck test (Kleinman's shear test)

This is used to detect pain over the LT-interval, indicating a complete or partial rupture or occult ganglion. The patient's elbow is positioned on the table in a neutral position. The examiner fixes the lunate palmar and dorsal with the thumb and index, while the examiner pushes the pisiforme dorsally.

Midcarpal instability (Lichtman's test)

Lichtman's test is used to diagnose midcarpal instability. The examiner fixes the all metacarpals with one hand and the forearm with the other hand. Under axial loading the wrist (neutral for flexion and extension) is brought from ulnar to radial deviation. The test is positive if a painful clunk is felt.

Distal radioulnar joint translation test

This is used to evaluate the integrity of the volar and dorsal radioulnar ligaments (RUL). The patient's elbow is flexed 90° resting on a table. The wrist is neutral in regard to pronation and supination. The radius and the ulna are both stabilized between a thumb and an index finger. The amount of translation is

evaluated in neutral, full pronation and supination. The latter two are used for evaluation of both dorsal and volar RULs, respectively (Figure 48.9, lower panels). A test is considered positive if a clear difference with the contralateral side is detected.

Ulnar abutment

The ulnar abutment test is used to evaluate the integrity of the volar and dorsal RULs. The examiner stabilizes the patient's hand and places his or her thumb on the distal ulna. The other hand is used to stabilize the forearm. Then the patient's wrist is fully deviated in ulnar direction and the forearm is supinated and pronated. A test is considered positive if a pain and/or a click is present.

Pisiform grind test

This is used to diagnose pathology in the piso-triquetral (PT) joint. The examiner holds the hand while palpating the pisiform, applying shear force pushing it into the triquetrum. The test is positive if pain is present during the manoeuvre.

Trapeziometacarpal joint translation test

Test used to assess the laxity of the trapeziometacarpal (TMC) joint. The base of the first metacarpal is fixed between thumb and index and translation should be evaluated and compared with the contralateral side.

Grind(ing) test

The grind test or grinding test of the carpometacarpal (CMC) joint of the thumb implies a combination of rotation and axial compression of the joint. The test is considered positive if it

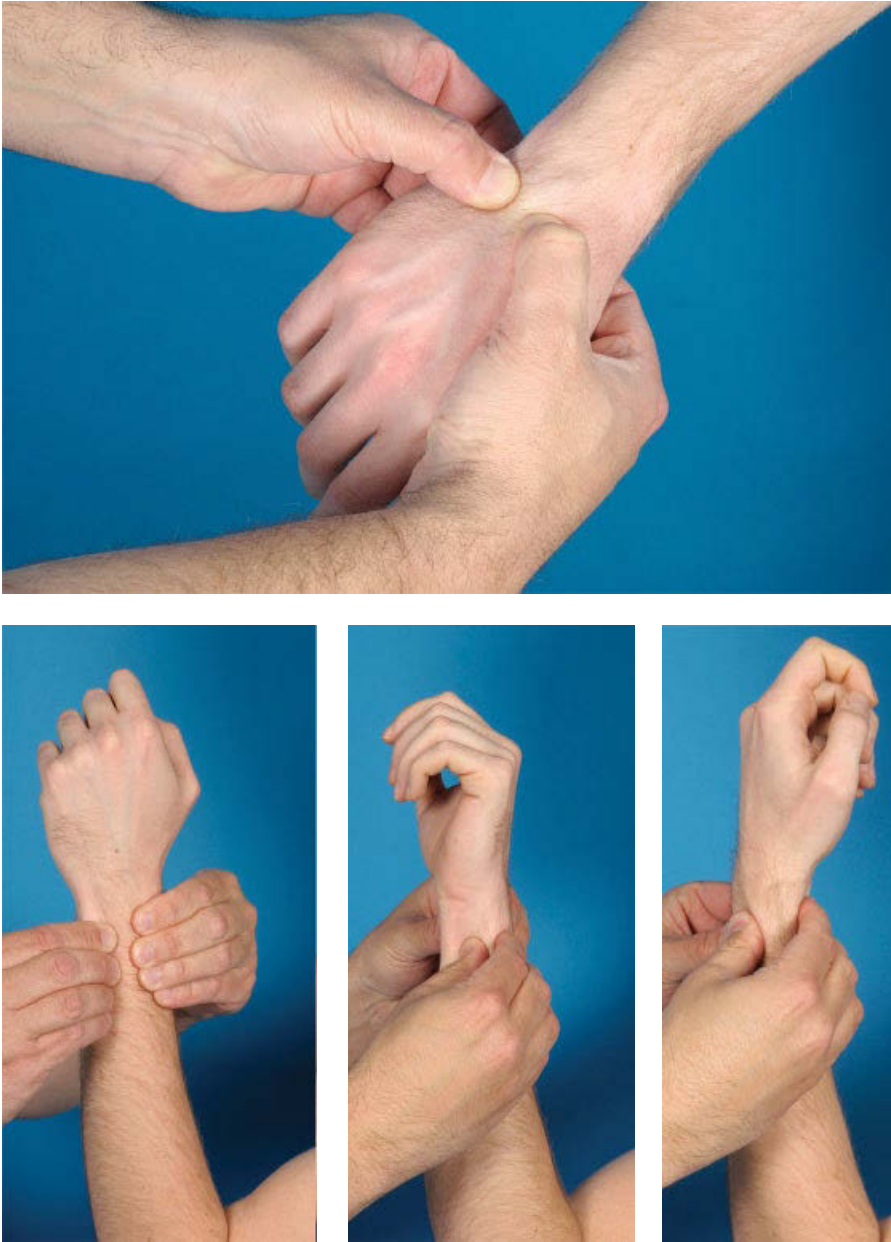


Figure 48.9 Distal radioulnar joint test.

elicits pain in the examined joint. The test can be executed by grasping the basal phalanx but this also stresses the MCP joint. To pressure only the thumb CMC joint, the thumb metacarpal should be grasped instead.

When the grind test is compared with the Eaton and Glickel system for radiologic staging of CMC osteoarthritis as the gold standard, the interrater reliability and the criterion validity of the grind test are as follows:

- Kappa value: 0.48
- Sensitivity: 42–53%
- Specificity: 82–93%
- Positive predictive value: 91–96%
- Negative predictive value: 68–70%.⁴⁹

Motor assessment

The Medical Research Council scale

In 1911 the Parliament of the United Kingdom passed the National Insurance Act, proposed by David Lloyd George as Chancellor of the Exchequer. Although its primary purpose was to provide the British working classes with their first contributory system of insurance against illness and unemployment, it also created a national fund for medical research. The Research Committee and Advisory Council that was set up in 1913, later in 1919 becoming the Medical Research Council (MRC), coordinated this research. Against the background of the vast numbers of nerve injuries in World War I and the start of World War II in 1939 the MRC formed a Nerve Injuries Committee that in 1942

published the first edition of *Aids to the Investigation of Peripheral Nerve Injuries*.⁵⁰ This atlas shows doctors how to examine limbs for peripheral nerve lesions and also contained the MRC scale for muscle strength. This scale divides strength into grades 0–5:

- Grade 0 – No contraction
- Grade 1 – Flicker or trace of contraction
- Grade 2 – Active movement, with gravity eliminated
- Grade 3 – Active movement against gravity
- Grade 4 – Active movement against gravity and resistance
- Grade 5 – Normal power.

Contrary to what these numbers suggest, this scale is not linear but more of an exponential nature because most muscles only need a minor percentage of their normal strength to overcome gravity. This percentage is different for every muscle, being about 2% for the biceps muscle and about 24% for the hip abductors.⁵¹ Consequently, for these muscles grades 0–3 cover respectively 2% and 24% of the normal muscle strength and grades 4 and 5 cover the remaining 98% and 76%, respectively. This means that only two grades (4 and 5) are available to describe the entire functional range of muscle strength. Knowing that manually tested muscle strength in the higher ranges is often overestimated⁵² up to a level that patients having only 50% of their normal muscle strength can be judged as normal,⁵³ it can be concluded that the MRC scale has its limitations with respect to the assessment of the percentage of normal strength. For this purpose dynamometry seems to be more suitable, except in clinical situations where a dynamometer cannot be used.

Grip strength

A sufficient amount of grip strength is necessary for the independent execution of the activities of daily living. When grip strength is going to be measured it should be noted that not all grip types are equally relevant. When measuring strength after carpal tunnel surgery, tip pinch dynamometry is more sensitive to median nerve function because it specifically targets the thenar musculature.⁵⁴ Maximum grip strength is achieved with the wrist in 35° extension.⁵⁵ In the case of five different hand dynamometer grip sizes the graph of the strength measurements at the five handle positions forms a bell-shaped curve with the greatest strength in the middle position. In the widest handle position the force is predominantly generated by extrinsic muscle action and in the smallest handle position predominantly by the intrinsic muscle action. When the strength of one hand is compared with the healthy contralateral one there is usually assumed to be a 5–10% difference in favour of the dominant hand. However, in 20–30% of the normal population the non-dominant hand is equally strong or stronger than the dominant hand.^{56,57}

Imaging

Plain film

Plain films are the basis of the work-up for pathology of the hand and wrist. Correct positioning of the hand and forearm is critical to obtain the required information.

For the fingers, views in three directions are needed in order to be able to evaluate pathology: posterior–anterior (PA), lateral and three quarters views.

The hand should be evaluated using PA and three quarters views. In most instances the lateral is not useful, but may be required on indication (e.g. carpal bossing, dorsal avulsion fracture of carpus, etc.).

For plain films of the hand and wrist, the shoulder should be abducted 90° and the elbow flexed 90°. This ensures a true posterior–anterior view of the wrist.

Special views can be obtained on indication, for example:

- Robert's view (to evaluate the TMC joint);
- carpal tunnel view (to evaluate fracture of the hook of the hamate);
- special fist view, ulnar/radial deviation (to evaluate the intercarpal ligaments of the proximal row: SL and LT).

There is only anecdotal evidence to be found on the clenched fist view for X-radiographs when SL ligament injury is suspected. One study comparing different views of the SL ligament concluded that a clenched fist view with 30° ulnar deviation (AP) resulted in the biggest SL gap: a Terry-Thomas sign of 4.55 mm (same as in a clenched pencil view).^{58–61}

- Level of evidence (Oxford Centre for Evidence-based Medicine): 4.

Ultrasound

Today musculoskeletal ultrasound offers quick, safe and relatively cheap visualization of the musculoskeletal system and the accompanying vessels and nerves. Initially the hospital's radiology department performed the majority of all ultrasound examinations. Nowadays gynaecologists and cardiologists perform their own ultrasound examinations and the rheumatologists are about to follow. According to this development it seems logical that in medical centres specializing in the treatment of patients with disorders of the hand and wrist, just as hand surgery became a specialism separate from orthopaedic or plastic surgery, the ultrasound examination of the extremities becomes a separate field of study. One of the major advantages of this is that the person performing the ultrasound examination can not only make a nice picture of the field or anatomical structure of interest but can also, because he or she is knowledgeable with respect to the disorder(s) the patient has or may have, confirm or deny diagnostic assumptions and actively pursue new diagnostic or therapeutic pathways. An additional advantage is the availability of more complex procedures such as the localization of nerves or other structures prior to operation, diagnostic ultrasound-guided nerve blocks using less local anaesthetics⁶² and ultrasound-guided palpation for a more precise localization of pressure-dependent pain.

CT scan

This modality is an important imaging technique for the hand and wrist. It was initially primarily used to evaluate fractures in three planes and in a three-dimensional fashion. Fractures of

Table 48.2 Comparison of MRI and MR arthrography for diagnosis of triangular fibrocartilage complex (TFCC) tears

	MRI				MRA			
	Sens (%)	Spec (%)	PPV	NPV	Sens	Spec	PPV	NPV
TFCC central	70.3	100	100	50.0	95.0	100	100	85.0
TFCC peripheral	60.0	100	100	85.0	93.0	97.0	88.0	97.0
TFCC full thickness	75.0	81.0			84.0	95.0		
1.5T	70.0	79.0			83.0	95.0		
3.0T	86.0	100			100	100		

MRI, magnetic resonance imaging; MRA, magnetic resonance arthrography; Sens, sensitivity; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value.

the carpal bones can be visualized especially well (e.g. avulsion fractures of the triquetrum).

In osteoarthritis of hand and wrist, a CT scan can show the exact extent of the damaged cartilage in a multiplanar fashion. The distal radial ulnar joint is difficult to evaluate on plain films due to the configuration of the joint. For example, we have shown that CT will show the extent of scaphotrapezotrapezoidal joint arthritis in more detail than plain films. Other areas for CT scans are, for example, bone tumours and vascular pathology (CT angiography). For soft tissue and in the presence of metal implants CT scanning has clear limitations.

With the use of intra-articular dye, CT arthrography has been shown to be useful for detecting SL ligament, LT ligament and central triangular fibrocartilage complex (TFCC) tears with high sensitivity and specificity. It is not as accurate at identifying peripheral tears.^{63,64}

MRI scan

In the last decade MRI has earned its place in the care of the hand and wrist. It is the scan of choice for soft tissue pathology. The improvement in the quality of the coils and the scanners has catapulted the modality forward. Also, avoiding radiation has clear health benefits.

Clinical applications are soft tissue tumours: ganglion cysts, giant cell tumours, lipomas, vascular tumours and bone tumours (e.g. enchondromas).

Occult fractures of the forearm, wrist and hand can be detected by a cortical break with bone bruising. In case of suspicion of a fracture non-union, MRI is better at showing this than plain films. Also ulnocarpal abutment can be seen by the early bone marrow oedema prior to the formation of cysts.

Infections can be detected, especially (chronic) osteomyelitis in the absence of clear abnormal infectious parameters.

In case of avascular necrosis of bone, MRI can demonstrate reduced vascularization in the early stages, making it useful in the diagnosis of Kienbock's and Preiser's disease.

The use of intra-articular dye has improved MR imaging for ligamentous injuries in the wrist, ligamentous injury to the thumb, MCP collateral ligaments, SL and LT ligaments and the TFCC.

When ulnar wrist pain exists without clinical instability on the ulnar side, additional work-up should be considered. MRI with or without intra-articular contrast is the choice. Pathology of the TFCC, LT interval, extensor carpi ulnaris and subsheath, quality of cartilage on the lunate fossa, lunate, distal radioulnar joint and distal ulna and synovitis can be seen. High-resolution MRI was able to diagnose distal radioulnar ligament, palmar radioulnar ligament or ulnolunate ligament injuries accurately.⁶⁵ The major limitations are asymptomatic tears in the central and/or peripheral TFCC and the LT ligament due to normal wear. For pathology of Palmer class 1B or 1C, MRI may give false-negative results.

Rüeggger reported that these tears often appear as non-communicating tears extending from the distal radioulnar joint into the triangular fibrocartilage.⁶⁶

With regard to the diagnostic accuracy of MRI and MR arthrography in the diagnosis of TFCC tears (central and peripheral), overall we can state that MR arthrography has a better prognostic value in diagnosing TFCC tears (Table 48.2).⁶⁷⁻⁶⁹

- Level of evidence (Oxford Centre for Evidence-based Medicine): 1a.

Bone scan

A bone scan or bone scintigraphy or three-phase radionuclide bone imaging (TPBI) is a technique in which a phosphorous-based compound attached to a radioisotope is administered to the patient by an intravenous injection. The most popular radioisotope is technetium-99m, which has a short (6h) half-life, limiting the radiation exposure of the body. Although the exact mechanism by which the phosphorous-based compound is metabolized and associates with the bone is unknown, three phases can be discerned in its distribution through the body. In the first phase, the first seconds after the injection, which is called the intravascular radionuclide angiogram phase, the tracer predominantly occupies the blood vessels. This phase can be used to check for the presence of vascular abnormalities with decreased or increased perfusion. In the second phase, 5–8 min after injection, which is called the blood pool or tissue phase, the tracer predominantly occupies the extravascular, extracellular spaces. This phase can be used to assess the relative vascularity to the area of interest, which can be useful in inflammatory lesions. In the third phase, 3–4h

after the injection, which is called the delayed or metabolic image phase, the majority of the radioisotope has been metabolized. This phase can be used to assess the location of areas with an increased level of bone turnover, such as fractures or osteomyelitis. Sometimes fourth-phase images can be made, 18–24 h after the injection, showing further increased uptake. This phase can be used to diagnose osteomyelitis in patients with delayed metabolism of the tracer due to decreased perfusion, such as in diabetes mellitus and/or peripheral vascular disease.⁷⁰

When radioactive substances are administered to a patient the resulting amount of ionizing radiation is always an important issue. A bone scan for a 70 kg adult results in a total body radiation load of about 1.3 mSv, which is about 216% of the worldwide average annual artificial radiation exposure of about 0.6 mSv. This being said it should be noted that the ovaries load (2.4–3.4 mSv), the testes load (1.6–2.2 mSv) and especially the bladder wall load (26–62 mSv), are considerably higher, although the latter disappears with voiding after 2–5 h.⁷¹

Dynamic conventional X-ray/fluoroscopy

The first fluoroscopic image was observed on 8 November 1895 by Wilhelm Röntgen when he saw a barium platinocyanide screen lighting up while being exposed to X-rays. The earliest fluoroscopes consisted of nothing more than an X-ray source and a fluorescent screen, hence the name fluoroscopy or fluorography. Unlike conventional X-ray examination, fluoroscopy allows the visualization of movement of structures within the body, although at the expense of a prolonged exposure to X-rays. The detrimental effects of the latter were already known to Thomas Alva Edison, who therefore in 1903 terminated his research on fluoroscopes. Nevertheless, between 1930 and 1950 fluoroscopes could be found in shoe shops in the United States, Canada and the United Kingdom to aid in the fitting of shoes.⁷² The mediocre quality of the created image and the relatively high radiation exposure initially limited the usefulness of fluoroscopes in the medical context. Later the invention of the X-ray image intensifier and the more sensitive flat-panel detector solved the first problem and, together with the invention of mini C-arms, decreased the second one, allowing fluoroscopy to become a generally accepted technique in hand surgery. Nevertheless, the longer exposure times in fluoroscopy and therefore the increased exposure to artificial radiation compared to conventional roentgenograms remained a controversial issue.

In patients with an SL distance of ≤ 3 mm, the sensitivity and specificity of cineradiography for ligament injury of the wrist^{73–75} is high (accuracy 0.93). This accuracy is higher than that for radiography alone (0.81). The accuracy of cineradiography is 0.90 when the SL distance is ≥ 3 mm (on conventional radiography).

- Sensitivity: 85–90%
- Specificity: 95–97%
- Positive predictive value: 0.94–0.98

- Negative predictive value: 0.86
- Level of evidence (Oxford Centre for Evidence-based Medicine): 2b.

Safety issues

During hand surgery with an average fluoroscopy time of 51 ± 36.9 s the surgeon's hands are exposed to an average $\pm$ SD of 0.2 (range 0.05–0.8) ± 0.12 mSv/case. This means that it will take an annual average of 2500 fluoroscopy-assisted operations to reach the 500 mSv/year limit for hand exposure. It can be concluded that it is virtually impossible for a hand surgeon to even approach this limit. That being said, one should know that the given annual dose limits have been calculated by extrapolation of data from medical occupational exposures, atomic bomb survivors and nuclear accident survivors and are not based on hard science. Consequently, the risk of low-dose exposure resulting in life shortening, somatic and heritable mutations is difficult to calculate. Currently most radiation biologists and geneticists assume that no dose of radiation can be considered completely safe and its usage must be determined on a risk-versus-benefit analysis. As a result of this it is advisable to use the as low as reasonably achievable (ALARA) principle with respect to the fluoroscopy-related radiation exposure by following the generally available suggestions regarding the settings of the mini C-arm and the distance and the position relative to the fluoroscope.

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CHAPTER 49

Congenital hand differences

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Embryology of congenital hand differences

Introduction

The upper limb bud develops from the flank of the developing embryo from day 26.¹ Somatic lateral plate mesoderm forms the skeleton and mesoderm from somites migrates in to form the musculature and contributes to the vascular system. Fibroblast growth factors initiate development along three axes with the interaction of signalling centres: *proximodistal* axis controlled by the apical ectodermal ridge (AER);^{2,3} *anteroposterior* (radioulnar) controlled by the zone of polarizing activity (ZPA); and the *dorsoventral* (or dorsovolar) controlled by the wingless gene encoded protein (Figure 49.1). Genes and their encoded proteins control growth and differentiation and act as messengers between signalling centres and developing cells. Mutations, whether inherited or spontaneous, disrupt secreted proteins, ligand factors and transcription factors. Environmental factors including drugs (e.g. thalidomide), radiation, nutrition and infection can also disrupt molecular pathways.

Timeline

The limb bud contains a core of proliferating mesenchymal cells covered by ectoderm which thickens at the tip to form the AER. The spinal nerves grow into this and an initial capillary network coalesces into a main stem artery, which drains into a marginal vein. The mesoderm organizes into the ZPA and a progress zone (PZ). By day 33, there is a paddle-shape limb with pre-chondrogenic condensations at sites where cartilaginous skeletal elements will form. Chondrogenesis takes place and myoblasts migrate into the limb and aggregate into ventral and dorsal muscle masses by day 36. At six weeks, digital rays form, giving the hand a webbed appearance and at seven weeks the upper limb rotates by 90° and the elbow flexes, bringing the palm anteriorly. Ossification occurs in parallel and apoptosis between rays leads to tissue involution and digital separation. Thus by week 8 a miniature adult upper limb is formed¹ (Table 49.1).

Limb patterning

Research into the developing chick embryo has provided a great deal of information on the process of limb patterning. In the proximodistal axis, the AER drives proliferation and bud outgrowth, secreting fibroblast growth factors, in particular FGF 2, 4 and 8.^{4,5} AER resection leads to a truncated limb, whereas AER grafting extends the limb. In the anteroposterior (radioulnar) axis, the ZPA is on the ulnar side between the polarizing region (posterior limb bud mesenchyme) and limb bud tip mesenchyme. This secretes Shh, which is the product of the *sonic hedgehog* gene.⁶ If the polarizing region is grafted anteriorly, a mirror set of digits results. In the dorsoventral axis, the somite provides a dorsal signal to the mesenchyme, which dorsalizes ectoderm. Right ectoderm transplanted onto a left limb results in a right hand developing. *Wnt7a* from the dorsal ectoderm and *LMX1B* from the mesoderm coordinate tendon and ligament formation.^{7–9} Other important genes in limb patterning include *HOXD* (*HOXD13* mutations are associated with central polydactyly) and *GLI3* (mutations lead to polydactylies).^{2,10–12}

Classification of congenital hand differences

Although there have been multiple attempts to classify congenital hand differences, an entirely inclusive system remains elusive. A largely morphological system, first proposed in 1968¹³ then refined in 1976¹⁴ and 1983¹⁵ by Swanson, was adopted as the standard by the International Federation of Societies for Surgery of the Hand in 1976 and remains the most widely used (Table 49.2). However, approximately 10% of congenital hand differences do not fit into this classification, and some anomalies fit into more than one category. Extensions to this classification include those by Knight and Kay in 2000¹⁶ and Upton in 2006.¹⁷

In 2013, Tonkin¹⁸ presented a classification system based on causation, taking into account the growing body of knowledge

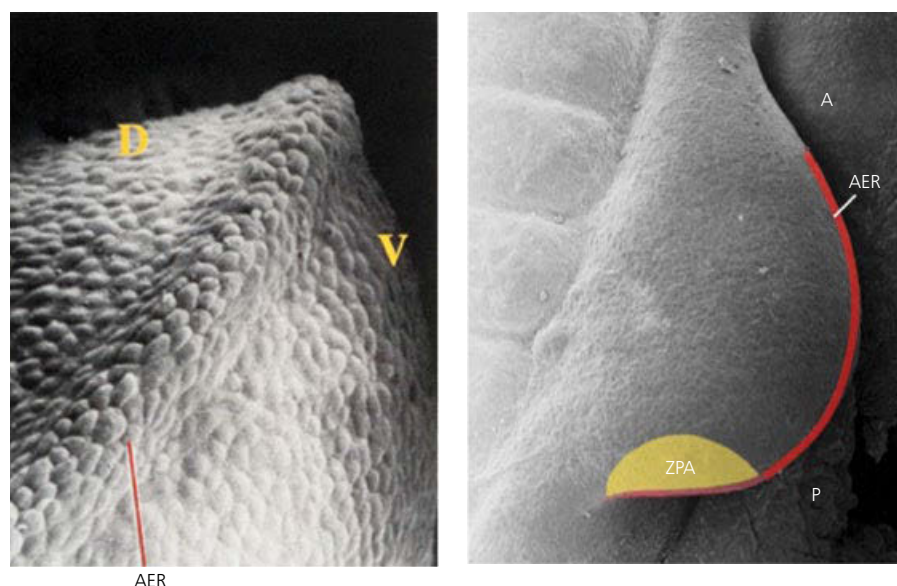


Figure 49.1 Development and patterning centres of the upper limb. Limb patterning is coordinated along three axis that are intimately linked through feedback loops. Outgrowth of the limb from proximal to distal is driven by the apical ectodermal ridge (AER). The anteroposterior axis is controlled by the zone of polarizing activity (ZPA) through the morphogen sonic hedgehog (*SHH*). Dorsal (D) and ventral (V) patterning is controlled via engrailed-1 (*EN1*), which is expressed in the ventral ectoderm and *WNT7A* whose expression is limited to the dorsal ectoderm.

Table 49.1 Timetable of upper limb development

Time point	Upper limb development
Day 26	Limb bud first appears
Day 33	Paddle-shaped hand present
Day 42	Digital rays present
	Hand assumes a webbed appearance
7th week	Upper limb rotates 90° laterally to bring the palm anteriorly
	Elbow begins to flex
	Ossification begins
	Involution of tissue between fingers occurs by apoptosis
8th week	Upper extremity resembles miniature adult upper limb

from molecular biology. He categorizes anomalies into malformations (an abnormal formation of tissue resulting from abnormal cell function), deformations (an insult to cells which have already formed normally) and dysplasias (a lack of normal organization of cells into tissue). Disruptions (alteration of tissue that is already formed) are considered alongside deformations. However, from a clinical point of view Swanson's system has proved the most practical to hand surgeons.

Syndactyly

A normal web ends at the midproximal phalanx on the volar side¹⁹ and an abnormal continuation beyond this level is termed syndactyly (from the Greek *syn*, together, *dactylos*, digit). Syndactyly is the commonest congenital hand abnormality with an incidence of 1 in 1000 live births. It is more common in white populations than in black and affects males more than females (46–84%). It is familial in 15–40% and can be unilateral or bilateral (25–50%).^{20,21} Syndactyly may affect the fingers and/or toes and most commonly presents as an

Table 49.2 Swanson classification of congenital hand differences

Failure of formation	Transverse arrest	Complete deficiencies
	Longitudinal arrest	Intercalated deficiencies
Failure of differentiation	Soft tissue	Radial (preaxial) longitudinal deficiency
	Skeletal	Central longitudinal deficiency
Duplication	Ulnar polydactyly	Ulnar (postaxial) longitudinal deficiency
	Central polydactyly	Syndactyly
Overgrowth	Radial polydactyly	Camptodactyly
	Ulnar dimelia	Trigger thumb
Undergrowth	Macrodactyly	Clasped thumb
	Brachydactyly	Clinodactyly
Constriction ring syndromes	Thumb hypoplasia	Symphalangism
	Groups 1–4	Synostosis
Generalized skeletal abnormalities	Achondroplasia	Arthrogryposis
		Windblown hand

First proposed in 1968 and adopted as the standard by the International Federation of Societies for Surgery of the Hand in 1976.

incomplete second web syndactyly in the foot. In the hand, the third web is most frequently involved (50%) (Figure 49.2a,b), followed by the fourth (30%), second (15%) and first (5%).^{19,20} A failure of apoptosis or programmed cell

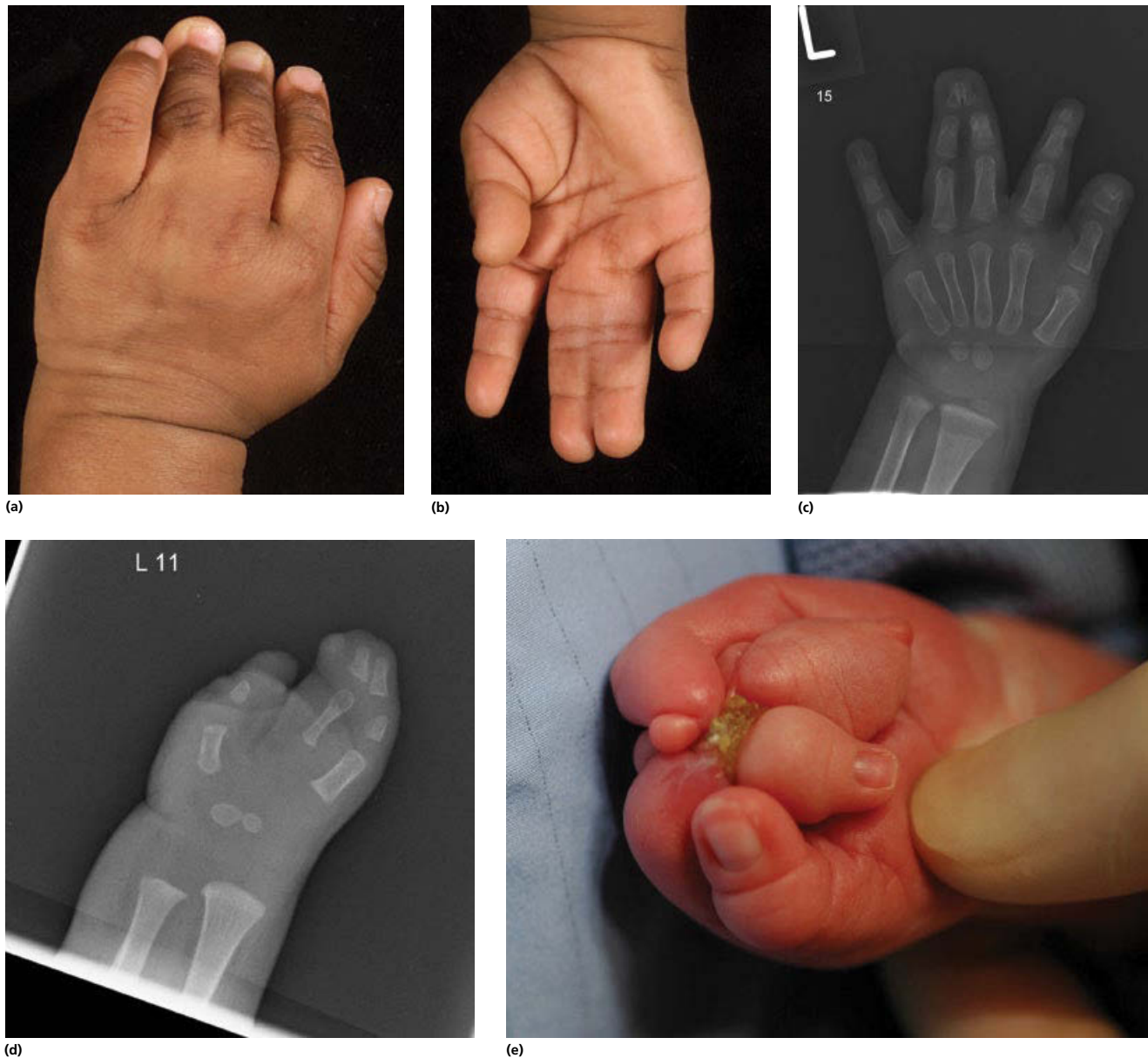


Figure 49.2 Images of different types of syndactyly. (a) Incomplete, (b) complete, (c) complex, (d) complicated, (e) acrosyndactyly.

death is thought to be responsible during digit separation from days 41 to 53.²²

Syndactyly may occur in isolation. However, it is also associated with a number of syndromes including Poland's, Apert's and Greig's syndromes. It can also occur in conjunction with other limb deformities such as polydactyly and constriction rings.

Classification

Syndactyly is classified as follows²⁰ (see Figure 49.2)

- Incomplete or complete (extending to the distal phalanx)
- Simple (soft tissue only) or complex (with bony synostosis)
- Complicated (a haphazard arrangement of bones with loss of normal ray patterning)

- Acrosyndactyly (fusion of digits distally, from the Greek *acro*, meaning tip, with proximal fenestrations commonly associated with constriction ring syndrome).

Clinical examination

Inspect the syndactylized digits. With complete syndactyly, the nails may be fused with an inward rotation of the digits, which suggests the presence of a complex distal bony union (Figure 49.2c). The degree of skeletal disruption within a syndactyly tends to parallel the degree of soft tissue abnormality, including that of tendons and digital neurovasculature structures. It is important to examine for the presence of digital creases, which provides valuable information about the passive

and active joint range of motion which may also be abnormal. The status of the joints pre-separation must be established to ensure adequate counselling of patients and their families with regard to postoperative motion and function. Often a child and family will opt for the correct complement of digital number over function in equivocal cases.

In complicated syndactyly there is more than a just a bony synostosis, with a haphazard arrangement that can include extraskkeletal elements concealed within a digit with associated fusions, rudimentary bones, missing bones, abnormal joints and sometimes cross bones, which may span several rays²³ (Figure 49.2d). It can be uncertain which bones best match with which digits. In acrosyndactyly, there is a complex syndactyly characterized by fusion of the bone distally but not proximally with often fenestrations or sinuses proximally (Figure 49.2e). This is discussed later under Constriction Ring Syndrome.

Surgical principles and management

Timing of surgery

Timing is controversial. With a minor syndactyly the later the release the less likely there will be recurrence in the form of web creep.²⁴ To reduce anaesthetic risk most surgeons operate over the age of 1 year. Delaying separation in this way has the added advantage of larger anatomy, including that of the vasculature. Indications for earlier surgery include syndactyly involving the border digits (i.e. between the thumb and index and the little and ring fingers) where tethering of growth can occur due to marked discrepancy in digital length. This can result in compromised function, and, in neglected cases, flexion contractures. Early release is also advised in cases of complex and acrosyndactyly, where bony

fusions can also affect growth.²⁵ The aim is for all possible separations to be complete by the age of 5.²⁴

Technique

The aim is to achieve both optimal function and cosmesis. Release involves separation of conjoined skin and subcutaneous tissue, ligaments and bony synostosis while preserving the integrity of the neurovascular bundles. Subcutaneous fat may be removed in effort to facilitate closure and contouring.

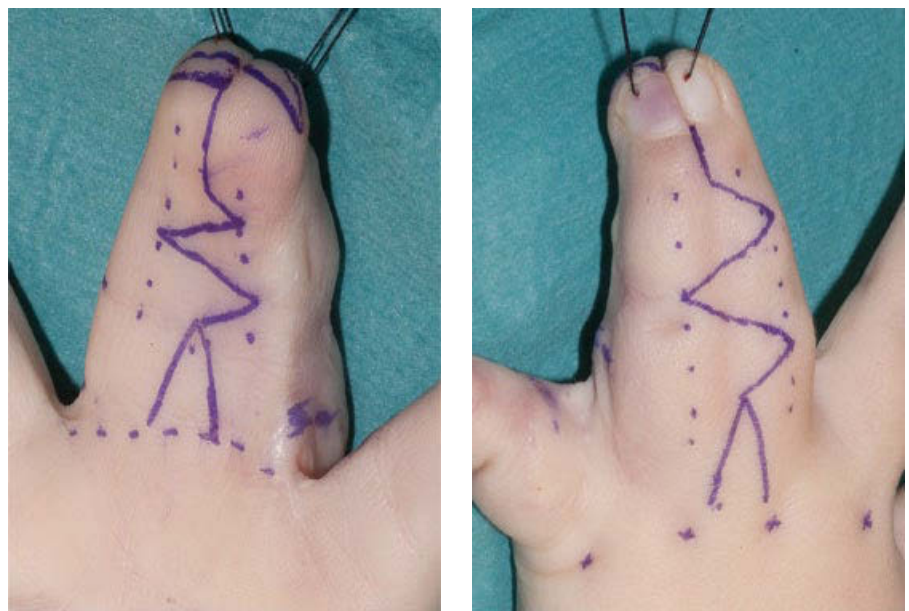
Separation can be divided into reconstruction of the web, sides of the digits and tips including the paronychia folds.

Web reconstruction

Multiple techniques have been described using volar, dorsal or a combination of flaps of varying sizes and designs.^{26–31} The web is generally reconstructed with a proximally based dorsal flap. Grafts are best avoided in the webspaces as they can lead to problems of poor pliability and delayed healing with associated web creep. Large triangular flaps are practical and versatile (Figure 49.3). They allow for adjustments to be made around abnormal distal branching of digital vessels and avoid transverse scars that can recontract in the future. They must be designed in such a way as to create a gentle slope from dorso-proximal to volar-distal.

Release of the first web involves the use of Z-plasties (four-flap (Figure 49.4), double, double opposing, five-flap), transposition flaps (from the dorsum of the hand and index or thumb), pedicled and free flaps depending on the defect size following thumb and index separation.^{32,33} It is important to release the fascia on the first dorsal interosseous and the adductor muscles. Sometimes it is necessary to reinsert the adductor muscle more proximally.

Figure 49.3 Author's preferred technique for syndactyly release using interdigitating triangular flaps. Design of interdigitating flaps marked on volar and dorsal sides of simple complete syndactyly.



Reconstruction of the sides of digits

Most commonly, interdigitating flaps from the volar and dorsal aspects are used to close the defects along the borders of the digits (see Figure 49.3). Straight-line incisions along the borders should be avoided as they have a high incidence of scar contracture and subsequent digital deformity. A skin graft is usually needed in all but the most minor cases, as even in a simple syndactyly skin shortage is at least 36% of the circumference.³⁴ The graft should be full thickness from the groin, inner arm or antecubital fossa. Thick split-thickness grafts from the instep can be used in some cases of toe separation. Graft take may be improved with quilting onto the underlying soft tissues, and in the case of complex, directly onto bone. Graftless techniques tend to reduce abduction. Withey *et al.* described an open technique with



Figure 49.4 Preferred method of first web release using a four-flap Z-plasty.

thick split-thickness grafts used at the base of the inner sides of the released digits with a large number of triangular flaps. Any open areas were left to heal by second intention with good cosmesis.³⁵

Single-stage releases of both sides of a digit should be avoided as this may jeopardize digital vascularity in cases of anomalous vascular anatomy. For example, in short multiple syndactylous fingers, dominant digital vessels can only exist on the lateral sides of the conjoined fingers. Sometimes it is necessary to sacrifice one of the vessels in a distal bifurcation. The digital nerves can usually be gently separated more proximally.

Tips

In a complete syndactyly with separate nails or partly fused nails, on separation of the pulps the skin can generally be advanced to close the resultant defects. In conjoined nails without a ridge, the lateral nail fold requires reconstruction, most commonly with interdigitating flaps from the hyponychium (e.g. Buck-Gramcko 'stiletto' flaps)³⁶ (Figure 49.5). Care must be taken to ensure these paronychia reconstructions are not too bulky and do not leave an excessively narrow tip.

Clinodactyly

Clinodactyly (from the Greek *clino*, deviated, *dactylos*, digit) is a congenital deformity in the radioulnar plane at the phalangeal level. Incidence is reported as 1–19.5% depending on the degree of angulation used to define it.³⁷ It is inherited as an autosomal dominant trait with incomplete penetrance though the exact aetiology is unknown. The angulation commonly occurs as a result of a triangular-shaped 'delta' or

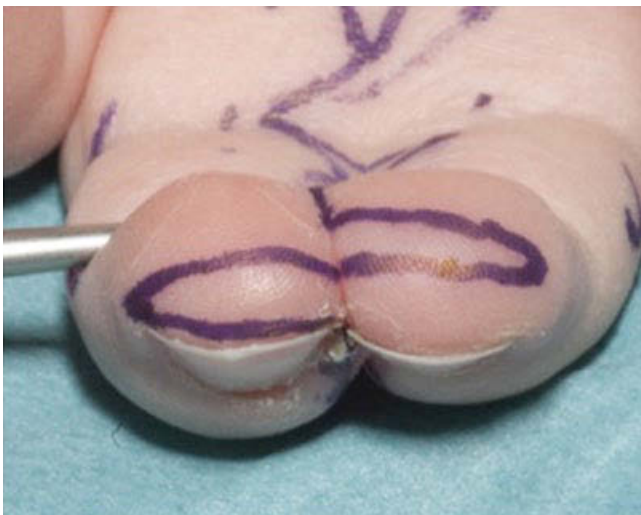


Figure 49.5 Buck-Gramcko stiletto flaps used for reconstruction of paronychia folds.

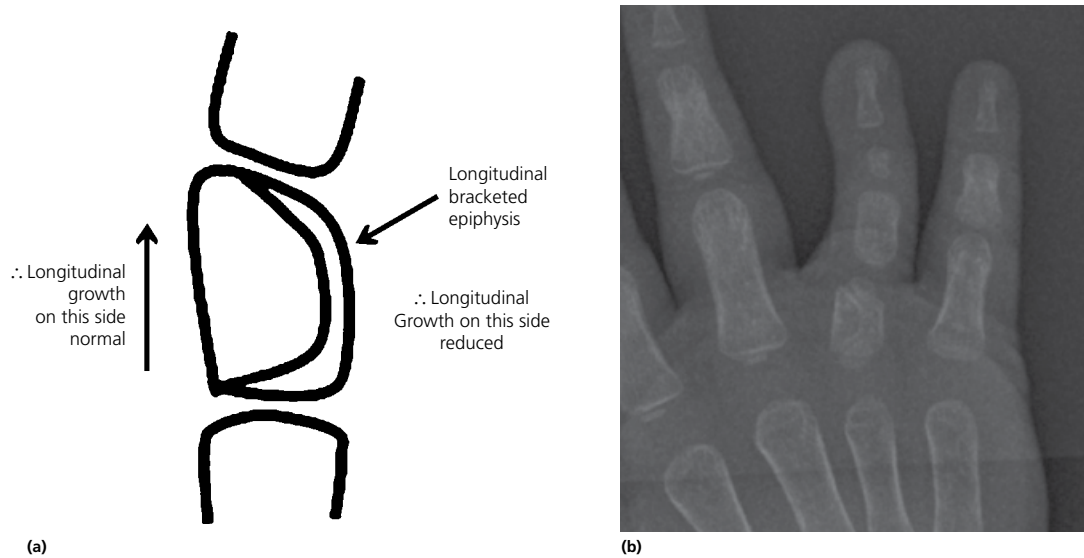


Figure 49.6 Formation of a trapezoidal phalanx as a result of a longitudinal bracketed epiphysis. (a) Growth on the side of the bracketed epiphysis is compromised. (b) Radiograph of a clinodactylous digit with a trapezoidal proximal phalanx.

trapezoidal middle phalanx (Figure 49.6), though more rarely the proximal phalanx can be involved. This is formed by an abnormal C-shaped epiphysis (longitudinal bracketed or comma-shaped), the arm of which extends onto the ulnar side of the middle phalanx (Figure 49.6a). The articular surface of the phalanx does not lie perpendicular to the longitudinal axis of the finger.^{38–41} The middle phalanx ossifies later than the other phalanges and latest in the little finger, hence clinodactyly occurs most commonly in the little finger. The thumb is the second most common. Clinodactyly most frequently occurs as an isolated familial abnormality. However it may also be linked to syndromes including Apert's syndrome (thumb), Trisomy 21, Poland's syndrome, Treacher Collins syndrome and Klinefelter's.⁴²

Clinical assessment

The most common presentation congenitally is in the little finger with radial deviation of more than 10°, seen at the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints, and a positive family history.⁴³ An acquired deformity may occur due to growth plate injury or deviation following syndactyly release. Kirner's deformity may be mistaken for clinodactyly. However Kirner's deformity represents a volar and radial curvature at the DIP joint. It most commonly affects females and appears between the ages of 7 and 14 years, often presenting as a painless swelling at the base of the nail.⁴⁴

A common presenting complaint with clinodactyly is cosmesis, as milder forms are not associated with any functional impairment. However with worsening angulation, function can become impaired. Radiographic assessment allows identification

of an abnormal or bracketed epiphysis. The status of the growth plate is an important factor in deciding treatment.

Surgical principles and management

As the abnormality is primarily skeletal and not corrected with growth there is little role for splintage or stretches. The main indication for surgery is worsening deviation with associated functional impairment. This commonly occurs with deviations of over 20–30°. Age and the status of the epiphysis are critical factors. If growing physiolysis with resection of the lateral physis and interposition of a fat graft can achieve good results with reliance then on the finger growing straight and correcting itself.⁴⁵ This is best done before the age of 6 years. Durability of this procedure is influenced by the extent of soft tissue release at the time of surgery.⁴⁶ Epiphyseal bar resection with fat grafting is best avoided in small triangular-shaped bones with minimal growth potential and best not repeated to avoid premature epiphyseal closure.⁴⁷ With persistent deformity or after completion of growth, correction is best achieved via osteotomy procedures. These include a closing wedge osteotomy where bony wedge is removed from the longer side in order to correct angulation. If length preservation is more critical an opening wedge, reverse wedge or dome osteotomy (Figure 49.7) may be more appropriate. However in this form of correction the soft tissue deficit on the shorter side must be addressed in order to facilitate correction and prevent recurrence in the long term. This can be achieved through techniques such as Z-plasties or local transposition flaps. Our preference whenever possible is for an opening wedge or dome osteotomy (see Figure 49.7) with longitudinal K-wire fixation.

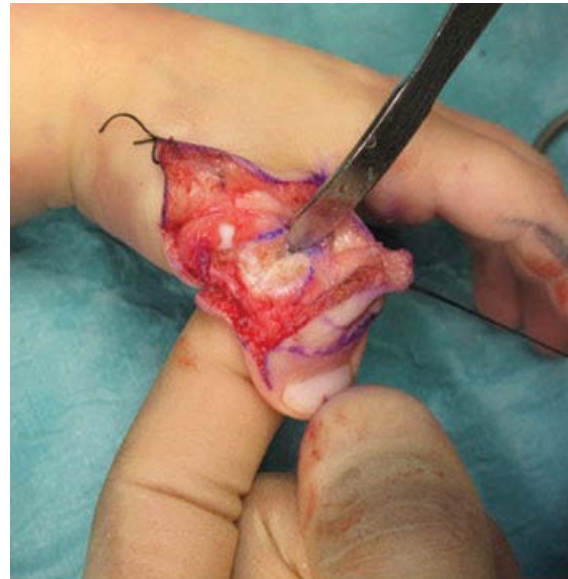


Figure 49.7 A dome osteotomy for correction of clinodactyly. The dome is created via multiple K-wire holes in an arch configuration linked through of the use of osteotome or fine side cutting burr.

Camptodactyly

Camptodactyly (from the Greek *kampylos*, arched, *dactylos*, digit) is a congenital flexion deformity of the PIP joint (Figure 49.8). There are approximately 1 million cases in the United Kingdom per year. The little finger is affected most commonly (70%), then the ring (20%) and then the other digits (10%). Bilateral involvement is common and multiple digits can be affected.⁴⁸ Two common patterns of presentation exist. First, in the newborn age group, worsening during the 1–4 year growth spurt. In this group males and females tend to be equally affected. Second, in the adolescent growth spurt between 10 and 14 years of age, in which there is a female preponderance. The condition tends to not progress beyond skeletal maturity. Most cases are sporadic. However in 30% of cases it is inherited as an autosomal dominant trait.^{49,50}

Pathology

Virtually every structure crossing volar to the PIP joint has been implicated in the condition. However abnormalities can be divided into primary causal anomalies and those that are secondary effects. The primary causes are commonly abnormal flexor digitorum superficialis and lumbrical anatomy. Tightness and aberrant distal insertions lead to a flexor–extensor imbalance. Secondary effects include abnormalities of the retinaculum cutis, volar plate, collateral ligaments lateral bands, central slip and skeletal changes.⁵¹ Skeletal changes such as joint space narrowing, flexion of the proximal phalangeal head and flattening of the base of the middle phalanx augur a poorer response to treatment and prognosis⁴⁹ (Figure 49.9).



Figure 49.8 Image of a camptodactylous little finger. A web can be seen across the proximal interphalangeal joint.

Clinical assessment

Functional impairment can occur when the flexion contracture exceeds 60° and multiple digits are involved. Otherwise issues tend to be more cosmetic. Foucher described four types⁵² (Table 49.3).

Factors associated with a poor response to treatment include skeletal changes, older patients (teenagers) with stiff joints and those who demonstrate poor compliance with hand therapy.⁵²

Surgical principles and management

The mainstay of initial management is stretching and splintage. We reserve surgical intervention for patients with a progressive condition despite compliance to hand therapy and flexion contractures of more than 60°. Outcomes from surgery range



Figure 49.9 Radiographic image of camptodactylous digits, demonstrating narrowing of the joint space in the proximal interphalangeal joint, flattening with volar curvature of the head of the proximal phalanx and flattening of the base of the middle phalanx.

Table 49.3 Foucher classification of camptodactyly⁵²

Grade	Description
1a	Early and stiff
1b	Early and correctable
2a	Late and stiff
2b	Late and correctable

from 14% to 35% improvement.^{51,53} Outcomes from hand therapy in young children in the Japanese population have been excellent with an 80–92% improvement reported^{54,55} though poorer in white population series (mean improvement of 40°).^{52,56}

Preferred method of surgical correction

All abnormal structures, both primary and secondary, must be addressed. We use a volar longitudinal approach with Z-plasty lengthening as required. Steps include

- Release of the retinaculum cutis, lateral digital sheets and Grayson's ligaments.
- Release of any abnormal insertions of the lateral bands and interosseus.

- The lumbricals are inspected and abnormal insertions are released.
- If the flexor digitorum superficialis (FDS) is functional and traction on the lateral bands produces PIP joint extension, an FDS transfer to the extensor mechanism via the lumbrical canal can be performed.
- If the FDS is deficient proximally and not usable it is released.
- If the flexor digitorum profundus (FDP) or FDS musculotendinous units are short but functional they can be fractionally or step lengthened in the forearm.
- Joint release may be necessary, including release of the check-rein ligaments, accessory collaterals and volar plate in turn as required.
- Our preference is to avoid skeletal correction as this leads to further stiffness of the PIP joint and a poorer outcome.

However, no single procedure has proved to be universally successful. Other techniques described include Glicenstein's subperiosteal release,⁵⁷ angulation osteotomy and arthrodesis.

Duplication

Duplication may involve the whole limb or part of a limb. Polydactyly occurs when greater than five digits or toes are present. It may occur as an isolated anomaly, either sporadic or inherited, or syndromic with a multitude of associated syndromes described. Syndromic polydactyly may occur in combinations such as radial with ulnar and may coexist with other hand and foot anomalies. The aims of surgical intervention are to improve function, especially in terms of glove and shoe fitting, and cosmesis. Upper limb polydactyly is the focus of this section.

Radial polydactyly

Radial, or preaxial, polydactyly occurs in 1 in 3000 live births.³⁷ It is usually unilateral and sporadic with no associations. The expanded Wassel classification with contributions from Wood in 1978 and Hung in 1996 is generally the most widely used system^{58–60} (Table 49.4). A significant proportion of cases may not fit completely into this classification and so additional terms such as deviation or triphalangism can be used. Chromosome 7q36, harbouring the regulatory element of the SHH protein, is implicated in the rarer dominantly inherited radial polydactyly.²⁴ Typically there is some degree of hypoplasia of both duplicates, with the radial usually the smaller of the two (Figure 49.11a). The ulnar-innervated intrinsics to the thumb typically insert onto the ulnar-most duplicate and the median-innervated onto the radial duplicate.

Clinical examination

Other anomalies may be present and so it is always important to examine all four limbs. More specifically, in radial polydactyly the thenar musculature may be hypoplastic, especially with increasing Wassel grade.⁴³ The extrinsics are usually Y-shaped

Table 49.4 The expanded Wassel classification system of thumb duplication based upon radiographic appearance^{58–60}

Grade	Description
Type I	Bifid distal phalanx
Type II	Duplicated distal phalanx (approximately 20%)
Type III	Bifid proximal phalanx
Type IV	Duplicated proximal phalanx (commonest, approximately 50%)
Type V	Bifid metacarpal
Type VI	Duplicated metacarpal
Type VII	Triphalangeal, in either or both thumbs (approximately 12%)

and asymmetrical with hypoplasia or absence involving the smaller digit. Simultaneous flexion or extension may occur with or without deviation. The joints may be stiff, hypermobile or hypoplastic. Note the presence of creases and hence the chance of active movement following surgery. There may be asymmetry of the pulps and nail beds. The first web may be tight, especially with more proximal duplications.

Surgical principles and management

Our preference for timing of surgical correction is between 12 and 18 months of age as long as other, more pressing comorbidities have been addressed. In an unbalanced duplication the thumb to be retained and reconstructed should be first determined. Different parts from each thumb may need to be used in order to provide the best complement of structures for stability and function.^{24,43} Careful dissection of intrinsic, extrinsic muscles and tendons and ligamentous structures must be carried out. Pollex abductus – an abnormal deviating radial connection between the flexor and extensor apparatus – must be looked for and divided if present to allow for unrestricted motion and growth.^{61,62} Preservation of an optimal neurovascular supply is important for thumb viability and the preservation of sensibility. One-stage reconstruction, including osteotomies to ensure correct skeletal alignment, is recommended.

Correction of distal duplications (Wassel types I–III)

Unbalanced distal duplications

In unbalanced distal duplications where one side is larger with a well-formed nail, excision of the deficient partner, most commonly the radial, is best. The preserved duplicate – usually the ulnar – is augmented with tissue salvaged from its radial partner. Incisions are planned high in the axial line dorsal to the junction of glabrous and non-glabrous skin. The radial border of the ulnar thumb is excised to allow inset of the flap. During the dissection, the radial nail and bone are excised, and the tendons are sectioned at the level of their division. Only one nail is preserved. A broad proximal phalangeal head can be trimmed. Residual deviation of the thumb can be corrected via a closing wedge osteotomy of the distal phalanx in type II duplications or

proximal phalanx in type III. Reconstruction of a radial collateral ligament using local tissue is important in this technique.

Balanced distal duplications

Decision making becomes more complex when the duplications are hypoplastic but of equivalent size (balanced type). One approach in this situation is a modified Bilhaut–Cloquet procedure using asymmetric skeletal elements from each phalanx in an effort to preserve joint surface integrity and an entire nail bed from one duplicate to avoid a nail ridge.^{63,64} However, this approach can still result in a broad and aesthetically suboptimal thumb so the aforementioned method of retaining one duplicate and augmenting it with tissue salvaged from the other is preferred whenever possible.

Correction of type IV duplications

In general, the best approach for these, the most common of preaxial duplications, is the preservation of the ulnar-more developed thumb and reconstruction of motion and stability using elements from its radial partner. The principles and steps are similar to those in type II reconstruction with also initial detachment and subsequent reattachment of the intrinsic muscles. Incisions are planned around the radial duplicate to be excised, preserving skin to allow for soft tissue recontouring of the retained thumb. Decisions around which duplicate to retain becomes more complex in a balanced duplication (Figure 49.10). Skeletal work is often necessary to achieve long-term correction of alignment.⁶⁵ The bifid head of the metacarpal must be trimmed to prevent residual exostosis distorting the radial contour of the reconstructed thumb. An additional osteotomy of the metacarpal may be necessary to correct any residual clinodactyly. This involves the removal of a triangular wedge of metacarpal neck to create a perpendicular articular surface. The osteotomy is fixed with a longitudinal K-wire and the radial collateral ligament should be reconstructed if possible. The latter may not be possible due to the extensive dissection required for skeletal realignment and stability relies upon correct re-insertion of intrinsic musculature, in particular the abductor digiti minimi.

Correction of proximal duplications (Wassel types V, VI and VII)

The basic principles outlined above also apply to type V, VI and VII duplications. The ulnar-more developed thumb is usually retained and reconstructed (Figure 49.11). The intrinsic muscles must be repositioned, the lax radial collateral ligament must be tightened and the extrinsic tendons centralized if necessary.⁶⁶ Adduction contractures in the first web must also be released to improve function. In type VI duplications where both the metacarpophalangeal (MCP) and carpometacarpal joints are abnormal, an epiphyseal sparing chondrodesis at the MCP joint will provide the necessary stability for a functional thumb. In type VII duplications the thumb with the two phalanges is retained if it

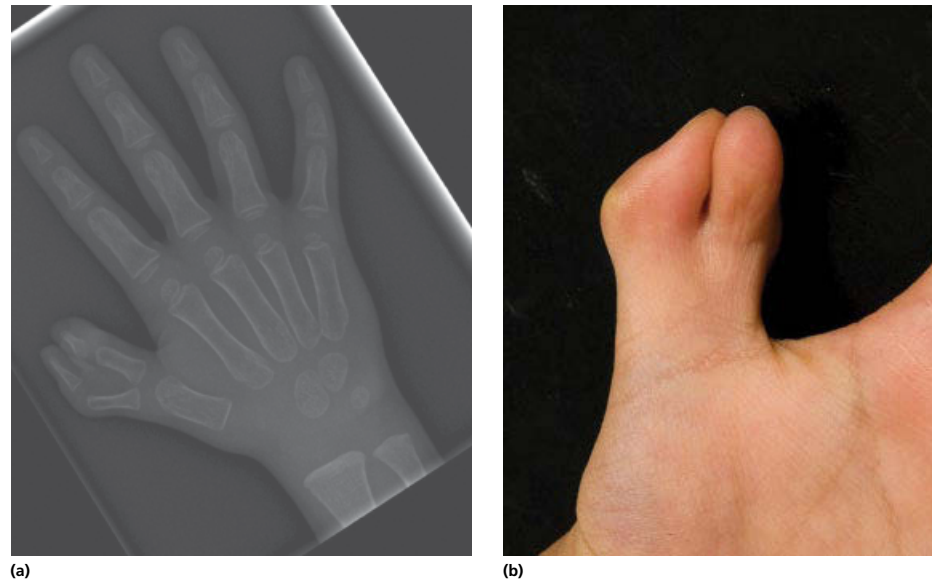


Figure 49.10 Wassel IV thumb duplication. (a) Radiographic image of a balanced Wassel type IV duplication demonstrating duplication from the proximal phalangeal distally. (b) Image of a patient with a Wassel type IV thumb duplication.

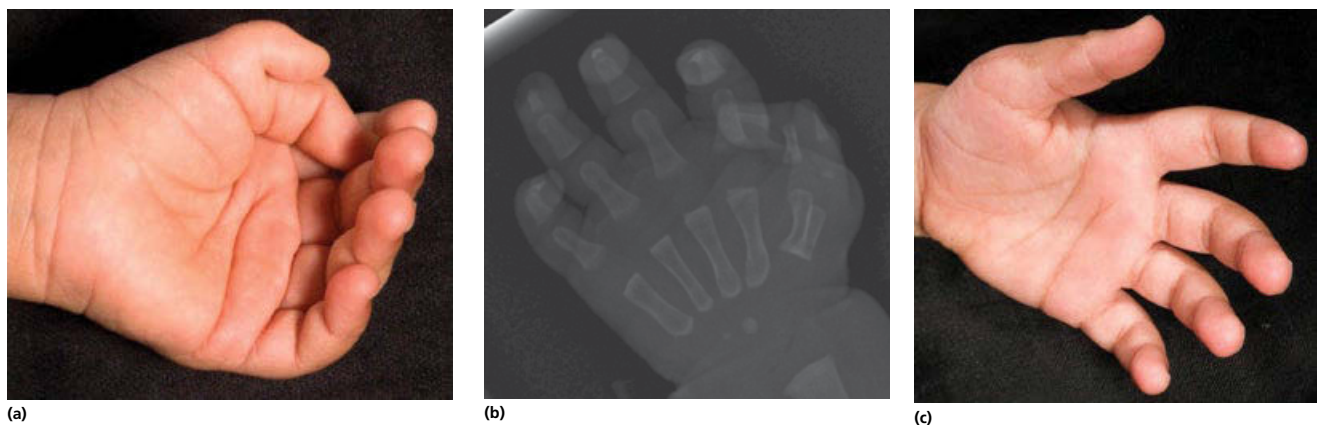


Figure 49.11 Reconstruction of a Wassel type VII duplication. (a) Preoperative image of an unbalanced type VII duplication. (b) Preoperative radiograph demonstrating smaller triphalangeal radial duplicate. (c) Postoperative image of corrected type VII duplication after removal of radial duplicate.

is the more stable and components of the triphalangeal duplicate used for reconstruction. If the thumbs are comparable in size and functionality then preference should be given to that with the more stable ulnar collateral ligament at the MCP joint.³⁷ In some instances triphalangeal thumb reconstruction may require an on-top-plasty, whereby the distal portion of one thumb is transferred to the proximal part of the other with the better basal joint.

Ulnar polydactyly

Ulnar, or postaxial, polydactyly occurs in 1 in 500 live births (37). It is the most common congenital hand anomaly. It is often bilateral and usually of autosomal dominant inheritance. Temtamy and McKusick in 1969 divided ulnar polydactyly into

type A duplications which were more developed and substantial and type B which were rudimentary and pedunculated.⁶⁷ Type B are ten times more common in black populations.⁶⁸ Whereas type A is rarer and its incidence is more common across races.⁶⁹ Ulnar polydactyly, in particular the more substantial type A, is associated with a number of different syndromes, especially in white and Asian populations. These include chromosomal abnormalities such as Trisomy 13 and 18 and bone dysplasias such as Ellis-van Creveld.

Classification

Stelling and Turek in 1963 classified ulnar polydactyly into three types based on the degree of duplication as shown in Table 49.5⁷⁰ (Figure 49.12). Type I within this classification is the most common (Figure 49.12a). However, clinically there is a spectrum

of complexity and the common small pedunculated digit usually has at least a nail and distal phalanx. A neurovascular bundle is always present, however rudimentary the appendage. The digit may auto-infarct and auto-amputate prior to presentation.

Clinical examination

Clinical assessment follows the principles of radial polydactyly. The most common site of attachment when a more developed digit is present is at the MCP joint level. It is often abducted

Table 49.5 Stelling and Turek classification of ulnar polydactyly⁷⁰

Grade	Description
Type I	Soft tissue mass without skeletal structure and skin bridge only (pedunculated)
Type II	Digit with all normal components and articulating with a normal or bifid metacarpal or phalanx
Type III	Complete digit articulating with an extra metacarpal

at the MCP joint and radially deviated at the PIP joint. The extrinsic tendons are usually Y-shaped and asymmetrical as in radial polydactyly.⁴³

Surgical principles and management

Type I

We find ligaclip ligation or crushing with diathermy to the neurovascular bundle at the base in an outpatient setting effective, being less likely to leave a residual nubbin than the more traditional tying off (Figure 49.12a). Alternatively, formal excision under local anaesthetic can be performed within the first three months of age.

Type II

The ulnar duplicate is usually the most hypoplastic and is therefore removed with preservation of the ulnar collateral ligament at the MCP joint if required and re-insertion of any aberrantly positioned hypothenar musculature (Figure 49.12b, c). Trimming of a broad metacarpal head may be required to create

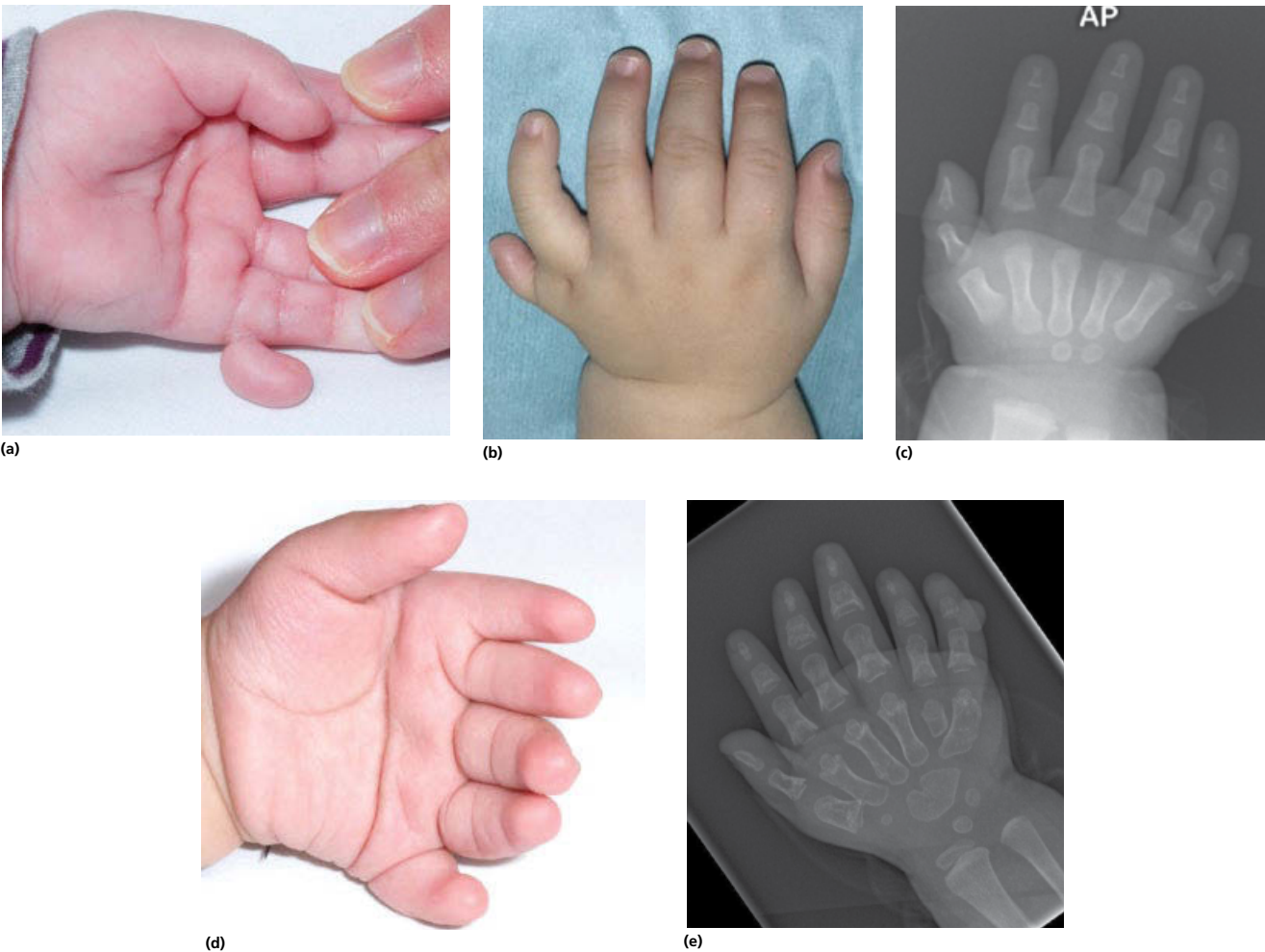


Figure 49.12 Images of three Stelling grades of ulnar polydactyly.⁷⁰ (a) Type I – pedunculated soft tissue digital mass. (b, c) Type II – complete digit articulating with bifid phalanx or metacarpal. (d, e) Type III – complete digit along with extra metacarpal.

the optimal ulnar contour. Resection of the metacarpal head and MCP joint reconstruction may be necessary.

Type III

Principles detailed above with reference to type II corrections apply in particular with unbalanced type III polydactyly (Figure 49.12d,e). In balanced type III situations it is preferable to retain the ulnar ray with its intact complement of hypothenar muscles.

Central polydactyly

Central polydactyly is rare condition. The ring finger is most often involved, followed by the middle then the index. A complete extra independent digit is rare. Often the situation is more complex, involving more than one digit with aberrant bones and abnormalities of the extrinsics, intrinsics and neurovascular supply.²³ Examples include duplication of the middle finger proximally at the metacarpal level, which shifts to an aberrant duplication of the ring finger within the digit. Duplication of the ring finger is often partial and hidden by a syndactyly with the middle digit. Polysyndactyly can be associated with *HOXD19* gene mutations with involvement of the little toe of the foot, together with a suppressed polysyndactyly of the ring finger.⁷¹ Surgical management should be considered carefully in central polydactyly as involved digits are often deficient, with significant soft tissue anomalies with poor function upon release.⁷²

Macroductyly

Macroductyly involves the localized enlargement of all structures of an affected digit (Figure 49.13 and 14). This distinguishes it from conditions where only part of the digit is involved



Figure 49.13 Image of a patient with macroductyly affecting predominantly the central two rays with an associated syndactyly.

(e.g. vascular malformations). It is rare, with an estimated incidence of 0.2 per 10 000 births. It affects the index most commonly and is usually unilateral.⁷³

The enlarged area often corresponds to the cutaneous distribution of specific nerves (i.e. nerve territory-orientated macroductyly).⁷⁴ If one digital nerve is affected, the macroductyly on that side will push the unaffected side away from the midline, causing a clinodactyly. If the common digital nerve is affected, both sides of adjacent fingers will be involved, leading to divergent clinodactyly.⁷⁵ Macroductyly tends to follow two patterns of progression: a *static form*, where growth is commensurate with the child's limb, and a *progressive form*, in which the growth is out of proportion (see Figure 49.13).⁷⁶ It can also be classified into five types according to specific disease entities, as described by Flatt and expanded by Upton.^{37,77} Type I is associated with lipofibromatosis (see Figure 49.14), II with neurofibromatosis, III with hyperostosis, IV with hemi-hypertrophy and V with additional conditions linked to overgrowth.⁷⁷ Conditions associated with overgrowth include Proteus and Beckwith-Wiedemann syndromes.⁷⁸

Surgical principles and management

Management of these cases is challenging due to the extensive nature and relentless progression of the condition. Surgery can be complicated by problems of delayed healing, infection

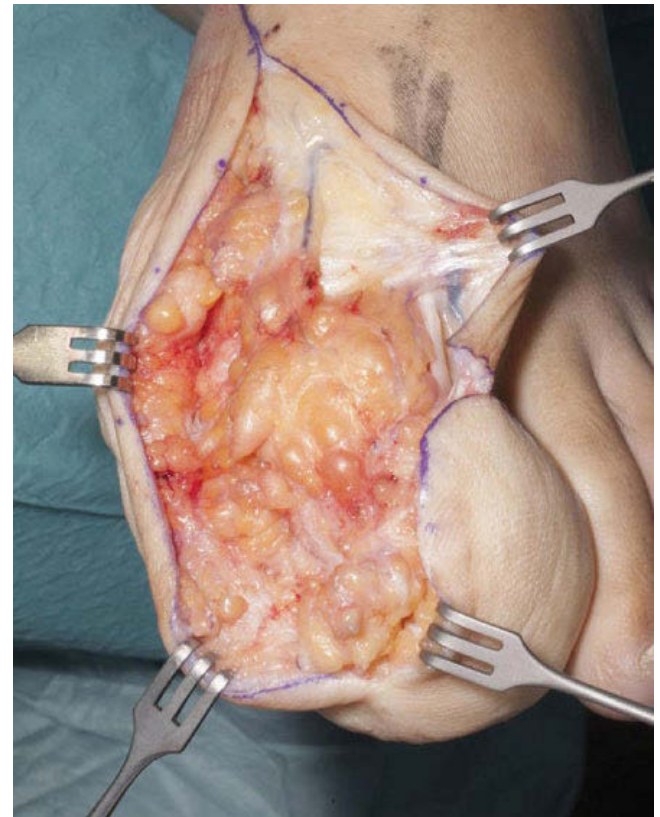


Figure 49.14 Intraoperative image of a case of macroductyly demonstrating harmartomatous overgrowth of all tissues.

and hypertrophic scarring.⁷⁸ The aims of treatment are to reduce bulk, maintain sensation and preserve joint motion whenever possible.⁷⁹ Careful counselling and the individualization of treatment is crucial. Psychological support is important as the psychosocial distress macrodactyly causes to patients and their families should not be underestimated.⁸⁰ The main surgical options are epiphyseal ablation (epiphysiodesis), soft tissue debulking with or without skeletal reduction (see Figure 49.14) and finally amputation. Nerve stripping or resection has also been recommended by some authors in an effort to suppress the neurotrophic drive seen in cases of nerve-orientated or neurofibromatosis type overgrowth.⁸¹ However, the long-term results of nerve exision/neurolisis are unpredictable and do not reliably prevent overgrowth.⁸² Epiphysiodesis is carried out to control longitudinal growth.⁷⁵ It is best carried out when the size of the affected digit reaches that of the parents. Destruction of the growth bar can be achieved with a needle, burr or osteotome under radiographic guidance. This often requires repetition and can be combined with debulking procedures to control circumferential girth. However, results from serial debulking surgery can be frustrating and despite early and repeated intervention the endpoint is often amputation, particularly in cases with a progressive growth pattern. Amputation should not be considered a treatment failure as it can significantly unencumber the hand, thereby improving overall function and cosmesis. Careful joint decision making with the patient and family must be carried out to avoid a protracted course with repeated hospital admissions and surgical procedures.^{78,79}

Thumb hypoplasia

Thumb hypoplasia can significantly impact on hand use as the thumb contributes to at least 50% of overall hand function.⁸³ Thumb hypoplasia represents a spectrum of abnormalities that range from structurally normal but small thumbs, through varying degrees of skeletal and soft tissue deficiency to complete thumb aplasia. It can exist as an isolated entity but is commonly seen as a component feature of radial ray deficiency. Its true incidence is hard to ascertain as thumb hypoplasia is present in a multitude of congenital malformations, in particular central and transverse deficiencies. Incidences of up to 37% of all congenital hand anomalies have been reported.⁸⁴ The aetiology is complex, encompassing multiple genetic and environmental factors.⁸⁵ This is confirmed through links to many different conditions, each with distinct aetiologies, including Fanconi's anaemia, thrombocytopenia with absence of radius (TAR) syndrome, Holt–Oram syndrome and the vertebral, anal, cardiac, tracheal, oesophageal, renal and limb association (VACTERL).^{86,87} Therefore involvement of a geneticist is advised.

Table 49.6 The modified Blauth classification of thumb hypoplasia^{89–92}

Grade	Features
1	Mild hypoplasia with all structures present
2	Moderate hypoplasia Thenar muscle hypoplasia First webspace adduction contracture MCP joint laxity Modified by Smith: 2A = ulnar collateral ligament laxity only 2B = global instability of MCP joint
3	Severe hypoplasia Metacarpal hypoplasia Thenar muscle hypoplasia Extrinsic hypoplasia Modified by Manske and McCarroll: 3A = Relatively stable CMC joint present 3B = unstable CMC joint
4	No metacarpal present Floating thumb (<i>pouce flottant</i> in French or <i>Pendeldarmen</i> in German)
5	Complete absence

Classification

The classification of thumb hypoplasia has evolved since that first proposed by Muller in 1937.⁸⁸ Currently the most widely accepted is the Blauth classification,⁸⁹ which has been refined through contributions from Buck-Gramcko in 1981,⁹⁰ Manske *et al.* in 1995⁹¹ and Smith in 2002⁹² (Table 49.6). Complete assessment of thumb hypoplasia must incorporate detailed analysis of its size and position in relation to the other digits, the first webspace, intrinsic musculature, extrinsic tendons and quality and stability of the thumb osseo-articular column to ensure optimal management. Accurate classification of anatomical and functional deficiency allows planning of surgical treatment.

Grade 1

There may be hypoplasia of the phalanges and metacarpal with weak intrinsics. The first web may be marginally tight. Joints are all present and stable. There is excellent function and parents may be unaware of any hypoplasia. This form is common in those with a contralateral more severe radial dysplasia.

Grade 2

The metacarpal and phalanges are present but may be narrower and smaller than usual. In addition the radial carpus particularly the trapezium, trapezoid and scaphoid may be hypoplastic.⁹³ A large spectrum of soft tissue abnormalities can occur, affecting both the extrinsic and intrinsic radial musculature. The median innervated thenar muscles, in particular the opponens pollicis and abductor pollicis brevis, are underdeveloped and weak. In contrast the ulnar-innervated intrinsics, particularly the adductor pollicis, are strong, which results in an abnormal adduction of the thumb and resultant narrowing of the first webspace.⁹³ Hypoplasia of the thenar intrinsics leads to a

weakness of thumb opposition, another defining feature of grade 2 hypoplasia. This, in combination with instability of the MCP joint, further compounds thumb dysfunction. In particular, laxity of the ulnar collateral ligament leads to radial collapse of the thumb during pinch. Grade 2A thumbs are defined by uni-axial (ulnar) instability and IIB by multiaxial instability of the MCP joint.⁹² Pollex abductus may also be present, causing radial deviation distal to the MCP joint through abnormal connections between the flexors and the extensor mechanism.⁶¹

Grade 3

Features of grade 2 thumbs are present but with more pronounced osseo-articular and extrinsic musculature deficiency. The osseous deficiency is most significant at the proximal metacarpal level, rendering the thumb even more unstable.^{90,94–96} There is in addition a degree of hypoplasia of the radial carpus, in particular the scaphoid, which is often absent. The distal radius is smaller and there is hypoplasia or total absence of the radial styloid.⁹³ The thumb MCP joint is unstable in all directions with severe underdevelopment of the collateral ligaments and volar plate. Intrinsic and extrinsic muscle abnormalities are pronounced. Flexors and extensors are usually present but are markedly weak. Abnormal tendinous interconnections can be present, producing a pollex abductus deformity.⁶¹ Intrinsic muscles are similarly underdeveloped, particularly those innervated by the median nerve, whereas ulnar-innervated adductor is relatively spared, compounding the first web adduction contracture.^{94,97} Manske *et al.* subdivided this category into grades 3A and B.⁹¹ In grade 3A thumbs the metacarpal, although hypoplastic, is present, forming a relatively stable articulation at the carpometacarpal (CMC) joint, whereas in the grade 3B thumb the proximal end of the first metacarpal is absent, which leads to instability of the CMC joint. More recently, in 2002, Smith suggested that all hypoplastic thumbs with a metacarpal present should be classified as grade 2. In other words Manske grade 3A should be classified as a grade 2C. He further suggested that Grade 3 should be divided into 3A where there is loss of the proximal one third of the metacarpal and 3B where there is a loss of the proximal two thirds.⁹²

Grade 4

These 'floating' thumbs arise most commonly distally along the radial midaxial line of the hand. They usually comprise two hypoplastic phalanges within a pedunculated soft tissue envelope that is connected to the hand by a narrow skin bridge containing a neurovascular pedicle. There are no metacarpal or intrinsic muscle insertions into the phalanges. A first dorsal interosseous may, however, be present.^{89,97}

Grade 5

This is defined by complete absence of the thumb and in 50% of cases there is a degree of radial deficiency.³⁷ The degree of radial hypoplasia correlates with deficiency within the index finger.⁹⁷ Hence those with a normal radius may try to 'auto-pollicize'

their index finger by pronating and abducting it and widening their second intermetacarpal space.^{37,97}

Surgical principles and management

Grade 1 function well and no intervention is required. The aims of reconstruction in grade 2 thumbs is widening of the first webspace, stabilization of the MCP joint and the provision of opposition. All three aims can be achieved through one surgery. Release of the adduction contracture allows the thumb to passively abduct and oppose, utilizing the shape of the CMC joint. Simultaneous opponensplasty adds the active ability to do so.

Addressing the first web contracture must ensure adequate release of all structures limiting thumb abduction, including the tight investing fascia over the adductor and first dorsal interosseous musculature. In addition, in severe cases release of the musculature itself from its origin or insertion may be required. Resurfacing the new opened web can be achieved in a number of ways depending on the degree of skin deficit. Mild to moderate contractures can be dealt with adequately using a single or four-flap Z-plasty technique. These also allow access to the MCP joint to facilitate joint stabilization and opposition transfers. More severe contractures may necessitate a large dorsal rotation-advancement flap to resurface the first web.^{98–100} More complex options include regional pedicled flaps such as the radial forearm or posterior interosseous flap or free tissue transfer with thin pliable flaps such as the groin flap.^{33,101}

Management of the lax MCP joint and the provision of opposition should be guided by the type of joint instability seen.¹⁰² Uni-axial (usually ulnar) instability of the MCP joint (grade 2A) can be corrected in the majority of cases as part of an FDS opposition transfer⁹⁵ (Figure 49.15a). The two slips of the FDS, usually from the ring finger, can be split and passed through drill holes in the base of the proximal phalanx and the head of the metacarpal, in a square configuration, as originally described by Lister.⁹⁵ Our preference, however, is to place the tendon repair on the radial side of the MCP joint so that in pinch tension is taken off it¹⁰² (Figure 49.15b). The use of the superficialis transfer in this way enables both MCP joint stabilization and thumb opposition using the distal border of the carpal tunnel as a pulley. In contrast, those with poor bone stock and instability in all planes (grade 2B) require an epiphyseal plate-sparing fusion of the MCP joint – a chondrodesis – in combination with an abductor digit minimi opponensplasty¹⁰³ (Figure 49.16) in order to produce equivalent functional results.^{102,104,105}

Grade 3A thumbs are generally reconstructable following the aforementioned principles outlined for grade 2 thumbs. But beware that even though the CMC joint may be present, it may be unstable or may appear stable at rest but may not be so in pinch. This is further exacerbated by MCP joint stabilization with increased force through the joint.

Grade 3B thumbs and onwards are best treated with ablation and substitution through pollicization of the index finger.^{62,96,97} Despite the advantages of a pollicization in grade 3B, some cultural and parental beliefs may preclude the use of the index



Figure 49.15 Flexor digitorum superficialis tendon combined collateral ligament reconstruction and opposition transfer. The two slips of the flexor digitorum superficialis are split and passed through drill holes in the base of the proximal phalanx and the head of the metacarpal in a 'box-like fashion'. The tendon repair is placed on the radial side of the MCP joint so that in pinch, tension is taken off the repair site. (a) Two slips of the flexor digitorum superficialis used to stabilize the MCP joint. (b) A schematic of the 'box-like' arrangement of the superficialis tendon around the MCP joint.



Figure 49.16 Abductor digiti minimi opposition transfer. Insertions of the abductor digiti minimi muscle into the base of the little finger are released. The muscle is then transferred, maintaining its origin on the pisiform and proximally based blood supply, to the thumb to provide opposition.¹⁰³

finger and demand alternative forms of reconstruction to preserve total digit number. These alternatives include staged reconstruction with both vascularized and non-vascularized tissue transfers. These approaches, despite yielding some good results, are more unpredictable and generally suboptimal when compared to index finger pollicization.^{106–110}

Pollicization

Pollicization is the transposition of the index as a vascular island into a functional thumb position. Excellent functional and aesthetic results can be achieved through careful planning and surgical execution but there will be compromises due to lack of

a true CMC joint, weaker intrinsic muscles and differences in phalangeal lengths.¹¹¹ Power is diminished to approximately 30–50% of the normal thumb. The key determinant of outcome is the quality of the index finger to be transferred. The principal stages are as follows:

- 1 Incisions – we prefer those described by Buck-Gramcko.¹¹²
- 2 Neurovascular dissection – commonly requires freeing of a neural ring, splitting the common digital nerve and division of the radial digital artery to the middle finger.
- 3 First dorsal and first palmar interossei detachment from their insertions.
- 4 Second metacarpal resection from the distal physis to the CMC joint. The physis is ablated to prevent future growth of the neotrapezium. Metaphyseal periosteum is removed to avoid new bone formation and painful bony spicules.
- 5 Fixation of the index MCP joint in maximal hyperextension to compensate for loss of the volar plate and provide stability in pinch.
- 6 Correct fixation of the neo-thumb into 100° pronation and 30° of abduction to allow for a degree of derotation during healing and rehabilitation.
- 7 Musculo-tendinous unit reattachment and balancing as follows:
 - Extensor digitorum communis becomes the new extensor pollicis brevis as it is the more radial.
 - Extensor indicis proprius becomes the new extensor pollicis longus.
 - The extensors are routinely both shortened, advanced and sutured at the level of the thumb MCP joint. The degree of shortening is proportional to that of the index metacarpal.
 - After adjustment of the extrinsic tendons, the intrinsic are inserted and tensioned appropriately. The first dorsal interosseous becomes the new abductor. The first palmar interosseous becomes the adductor.

- Balancing of the intrinsics is critical to correct posturing of the neo-thumb. The thumb is placed into maximal flexion at the interphalangeal joint and the intrinsics inserted on each side at maximal tension.
 - Long flexor shortening is only required over 7 years of age. Otherwise it tightens with growth and use.
- 8 Decompression of the tourniquet to allow for haemostasis and to check viability of the transfer.
 - 9 Skin re-draping and closure to contour the new thumb and first webspace.
- Revisional procedures are necessary in a significant proportion of patients and usually involve first web releases, extensor tenolysis and transfers to supplement opposition.

Radial longitudinal deficiency

Radial longitudinal deficiency (RLD), previously referred to as radial club hand, represents a spectrum of abnormalities along the preaxial side of the upper limb. As a result of both skeletal deficiency and soft tissue abnormalities the hand adopts a radially deviated, flexed and pronated position at the distal end of a shortened forearm³⁷ (Figure 49.17a). This may be associated with abnormalities along the whole length of the upper limb including the shoulder, elbow down to the thumb and radial digits. There may only be a mild thumb hypoplasia or, in its severest form, complete absence of the thumb and radius. The condition may be unilateral or bilateral but is rarely symmetrical. The right side is affected twice as often as the left. RLD also occurs slightly more frequently in males.¹¹³ The incidence is reported at 1:30 000–1:100 000 live births.^{114,115}

Aetiology

Environmental and genetic factors play a role though the exact aetiology is unknown. Environmental causative factors include thalidomide,¹¹⁶ valproic acid,¹¹⁷ phenobarbitol¹¹⁸ and ethanol.¹¹⁹ Bilateral cases are more likely to be linked to a syndrome. Most are due to sporadic mutations but some have a genetic basis and can be transmitted as either autosomal dominant or recessive disorders. Experimentally, radial dysplasia can be created in chick wings and the ZPA identified as responsible for anteroposterior limb patterning.¹²⁰ Expression of the sonic hedgehog gene⁶ and retinoic acid¹²¹ have been shown to be critical in regulating the function of the ZPA. This is influenced by several factors including the homeobox genes and is only induced due to signalling from the AER,² probably through fibroblast growth factor.¹²² Abnormal limb development may be related to several different interruptions within this pathway.

Associated syndromes

There are multiple associated syndromes, which confer a poorer prognosis. Syndromes that require early exclusion include those with haematological conditions, cardiac abnormalities and renal defects. It is therefore important to seek early input from a clinical geneticist.

Haematological

Fanconi's anaemia is an autosomal recessive pancytopenia, which is fatal without bone marrow transplantation. It is identified through a chromosomal breakage study which is recommended for all children with radial dysplasia. TAR syndrome is a rare autosomal recessive disorder characterized by bilateral absence of the radii in the presence of both thumbs and



(a)



(b)

Figure 49.17 (a) Image of a patient with type IV radial dysplasia showing radial deviation at the wrist and shortening of the forearm. A Blauth grade IV 'Pouce Flottant' thumb hypoplasia is also present. (b) Radiograph of the same patient demonstrating complete absence of the radius with absence of a wrist joint and bowing of the ulna.

hypomegakaryocytic thrombocytopenia.¹²³ The presence of functional thumbs distinguishes TAR syndrome from other conditions with radial aplasia which are usually associated with severe thumb hypoplasia or aplasia.^{123,124} In TAR syndrome the platelet count usually improves with age so intervention is best delayed until levels normalize.

Cardiac

Holt–Oram syndrome is autosomal dominant condition, which commonly exhibits atrial and ventricular septal defects.¹²⁴

VACTERL association

This is the largest individual syndromic group with associated radial dysplasia. Patients within this group vary in the severity of their presentation demonstrating a wide spectrum of associated anomalies. The diagnosis is made on the basis of a minimum of three of the following abnormalities – vertebral, anorectal, cardiac, tracheoesophageal, renal, limb and a single umbilical artery. Limb malformations are reported in 40–50% of cases and mostly represent radial deficiency.¹²⁵

Classification

Bayne and Klug classified RLD based on the degree of radial hypoplasia on radiographic assessment¹²⁶ (Table 49.7). This classification system does not encompass the degree of carpal or hand involvement.

Clinical findings

RLD is usually identified on a routine neonatal check. Investigations should then be instigated to exclude associated anomalies. The most profound changes in radial dysplasia are seen in the hand and wrist but it is important to examine the entire upper limb. Type I cases can be easily missed but are often heralded by the presence of associated thumb hypoplasia. Bilateral involvement is often asymmetrical in terms of severity so careful examination of both upper limbs is important.

Upper arm

The upper arm may be shorter than the opposite side with distal humeral epiphysis involvement. The humeral involvement parallels the degree of radial hypoplasia.

Table 49.7 Bayne and Klug classification of radial longitudinal deficiency¹²⁶

Grade	Description
Type I	<i>Short distal radius</i>
Type II	<i>Hypoplastic radius with distal and proximal epiphyses present but defective</i>
Type III	<i>Partial absence of the radius – most commonly of the distal one or two thirds</i>
Type IV	<i>Complete absence of the radius</i>

Elbow

The elbow may demonstrate bony abnormalities. There is usually full extension but flexion is sometimes limited initially. However this has been observed to improve over time if the wrist deformity is controlled with splintage.¹¹⁶ In addition, active pronation and supination are compromised. Patients tend to compensate for this by using shoulder rotation to turn their hands.

Forearm

The forearm is short with varying degrees of radial deficiency. The ulna is also hypoplastic and often bowed – growing to a maximum of two thirds the length of the contralateral side (see Figure 49.17). Without treatment ulna bowing can worsen. If some form of proximal radius is present its distal portion is replaced by a fibrous condensation or anlage. An additional deforming force is the presence of an abnormal combined radial muscle mass of flexor carpi radialis, extensor carpi radialis longus and brevis and brachioradialis which inserts into the anlage and on occasion the carpus. Soft tissue abnormalities, including those of musculotendinous units and neurovascular structures tend to parallel the degree of skeletal deficiency. Affected structures such as the median nerve tend to favour an aberrant radial course towards the distal forearm, hence complicating treatment and surgical correction.^{37,127}

Carpus

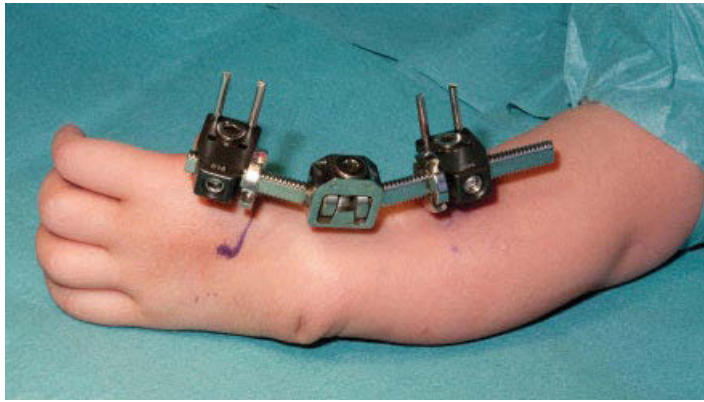
The radiocarpal articulation is often non-functional and may not even be present, with a hypoplastic or absent trapezium and scaphoid (Figure 49.17b). With ossification, increasing carpal coalitions are seen.

Hand

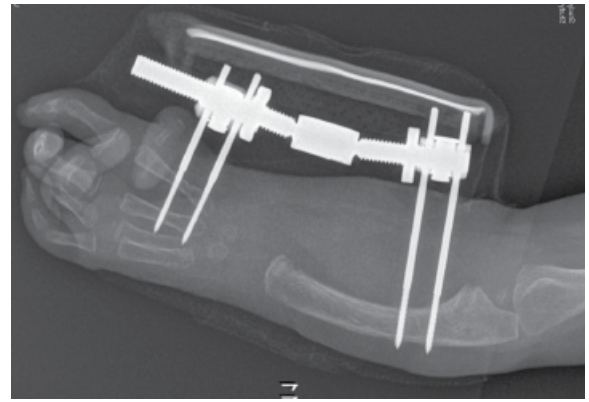
The thumb is often either hypoplastic or absent (Figure 49.17a). The ulnar side of the hand is the least affected, with worsening hypoplasia and stiffness in the radial digits. Motion at the MCP joints is restricted, although a degree of hyperextension is present. There are often flexion deformities of the PIP joints, particularly affecting central digits and in syndromic cases.¹²⁸

Surgical principles and management

The aims of treatment in radial club hand are to provide stable realignment of the carpus and hand on the distal ulna, improve function of the limb as a whole and maximize growth potential.^{36,116,126} The soft tissue structures on the radial side of the wrist are short, therefore treatment begins with stretching and splintage carried out by the parents with the guidance of a hand therapist. Physiotherapy aims to correct wrist deviation and to improve and maintain range of motion of the digits. In type I cases where the wrist is stable and easily corrected to neutral no intervention is required. But if a tendency for radial deviation remains, a tendon transfer of the tight combined dorsoradial muscle mass to the ulnar side of the carpus may be required.



(a)



(b)

Figure 49.18 Soft tissue distraction with a uniplanar mini-fixator device. (a) Image of a patient with type IV radial dysplasia with a Pennig Uni-planar Mini-fixator device with a central coaxial hinge *in situ* on the radial aspect of the limb. (b) Radiograph of the same patient demonstrating two-pin fixation across the second and third metacarpals distally and into the ulna proximally.

Type II cases are some of the most challenging to treat. Attempts have been made to distract and lengthen the radius with or without a bone graft.¹²⁹ This, however, often needs to be repeated as a child grows and results are unpredictable. In type III cases we advocate removal of the radial anlage and treatment as for type IV cases as detailed below.

Soft tissue distraction

The benefits of stretching and splinting on addressing radial deforming forces should not be underestimated. However these manoeuvres alone have proven insufficient in completely addressing radial tension prior to wrist correction particularly in severe type III and IV cases. As a result, Kessler proposed the idea of soft tissue distraction.¹³⁰ Since its conception, soft tissue distraction has revolutionized the treatment of radial dysplasia and has evolved through the use of a variety of different uniplanar and multiplanar devices. Our preference is a uniplanar device with a central coaxial hinge, which is placed on the radial side of the limb and secured by pin fixation into the ulna proximally and into the index metacarpal through to third metacarpal distally (Figure 49.18). On-table distraction yields a significant amount, after which incremental distraction is taught to parents to perform at a rate of approximately 1 mm per day. Maximizing this latter slower distraction is crucial in minimizing long-term radial tension and pressure on the ulna physis. Distraction is stopped once the third metacarpal base is aligned with the distal ulna, at which point the soft tissues are allowed to adapt to their new position prior to a wrist corrective procedure. We have noted that this 'consolidation' period of stress relaxation facilitates wrist stabilization and can produce some remodelling and straightening of the ulna.

Wrist correction

This can be achieved through a centralization with a proximal row carpal notch or a (non-notched) stabilization procedure. Centralization involves alignment of the third metacarpal with



Figure 49.19 Intraoperative image of a centralization procedure demonstrating creation of a carpal slot. The ulna is placed into the carpal notch and a tendon transfer is carried out of the dysplastic combined dorsoradial muscle mass.

the ulna through the creation of a notch in the carpus, producing a fusion or fibrous union to the proximal carpal row (Figure 49.19). This is more stable than a stabilization but inherently immobile. A stabilization procedure, also known as a radialization, involves rebalancing the carpus on the end of the ulna in line with the second metacarpal without a carpal slot. Both procedures are combined with a tendon transfer of the dorsoradial muscles mass into the base of the fifth metacarpal or

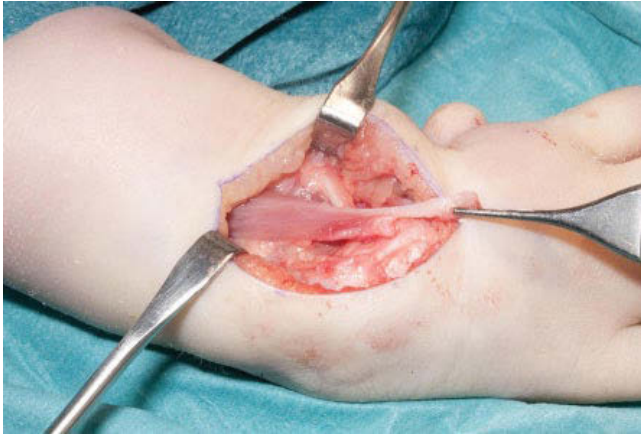


Figure 49.20 Intraoperative image of a radialization procedure showing transfer of the combined dorsoradial muscle mass. No carpal slot is made in a radialization (stabilization). Stability relies upon transfer of the combined dorsoradial muscle and congruency of the ulna and carpal joint surfaces.

extensor carpi ulnaris tendon to rebalance forces at the wrist and prevent recurrence into radial deviation (Figure 49.20). A dorsal approach via a longitudinal incision is all that is required, rendering complex incision patterns or flaps to address skin shortages obsolete. Repositioning through a radialization, although it preserves motion, is inherently more unstable than a centralization.

Review of our series shows there is a tendency for more long-term recurrence in radialization cases. As a result we would favour a stable centralization in cases where the quality of the dorsoradial muscle mass or ulna joint congruity does not lend itself to the stable equilibrium that is required for a radialization. Both types of correction are supported with longitudinal K-wire fixation for a period of six months to a year.

Alternative procedures in radial dysplasia

We consider the outcomes from soft tissue distraction followed by centralization or radialization with a tendon transfer satisfactory and have not pursued other methods. Vascularized and non-vascularized fibular head transfers have been used to replace the radius as well as vascularized metatarsophalangeal joint transfer.¹³¹ The cohorts in these series have been smaller and their take-up so far limited worldwide.

Following wrist correction, thumb hypoplasia can be addressed and later forearm lengthening. Forearm lengthening requires stability at the wrist and is fraught with potential complications. In highly selected patients it may bring functional and cosmetic gains that they consider worthwhile.^{132,133}

Constriction ring syndrome

Constriction ring syndrome is a rare condition of unknown aetiology. Numerous names have been used to describe this entity in the past, including annular band syndrome, constriction



Figure 49.21 Right hand showing constriction rings affecting the index, middle and ring fingers. Circumferential ring affecting the distal index finger and further constrictions producing acrosyndactyly of the middle and ring fingers.

band syndrome, Streeter dysplasia, Torpin dysplasia, amniotic band disruption sequence, acrosyndactyly, fenestrated syndactyly and congenital amputation syndrome.¹³⁴ The syndrome is characterized by bands of ranging severity, always present at birth, that can affect any part of the body with a particular predilection for the upper or lower extremities¹³⁵ (Figure 49.21). The reported incidence in the literature varies from 1 in 1200 to 1 in 15 000 live births depending on the population sampled and the diagnostic criteria used.^{135,136} When found in conjunction with significant limb involvement its incidence has been recorded as low as 0.19 per 10 000.¹³⁷ No genetic inheritance pattern has been identified but some environmental associations have been postulated. These have included possible links with abnormal gestational histories¹³⁸ and oligohydramnios.¹³⁹

Aetiology

Several theories have been postulated, including an intrinsic theory of localized areas of imperfectly formed tissue due to 'defective areas of germ plasm'^{135,140} and an extrinsic theory due to the formation of mesodermic strands because of rupture of or damage to the amnion during the first or early second trimester.¹⁴¹ These amniotic strands become entangled with the fetus, in particular the extremities, within the sac. In addition, resultant transient oligohydramnios caused by a loss of amniotic fluid can lead to abnormal pressure on developing limb buds, resulting in associated deformities such as talipes equinovarus – clubfoot.¹⁴² Anomalies such as cleft palate as part of Pierre Robin sequence also appear closely linked to the effects of oligohydramnios.¹⁴³ Similarly, there is a strong

association between limb ring constrictions and rare craniofacial clefts, again supporting the idea of a common causality.¹⁴⁴

Clinical presentation

No two cases of constriction ring syndrome are identical. The deformities are always present at the time of birth and prenatal ultrasonographic techniques enable early diagnosis.^{145,146} More than one limb is usually affected and associated musculoskeletal anomalies are commonly present^{135,147,148} (Table 49.8). There is a predilection for the more distal extremities – in particular the longer central three digits and longer great toe¹⁴⁹ (Figure 49.21). In line with an extrinsic aetiology, involvement of the thumb is rare due to its flexed adducted posture during intrauterine development, making entrapment more difficult. More proximal rings do occur but with much less frequency. However, it is rare for only one ring to be present as an isolated abnormality.³⁷

The severity of the deformity can vary dramatically, even between affected limbs of the same individual. The depth of the groove can range from mild indentations with minimal involvement of subcutaneous tissue to deep deficiency with associated disruption of lymphatic channels, nerves and blood vessels.^{148,150,151} Generally the rings tend to be deeper on the dorsal surface of affected extremities. Deep volar involvement can be associated with tendon involvement and joint contractures.¹⁵² Skeletal involvement is rare because in these cases auto-amputation tends to have already occurred.¹³⁴ Amputations can

occur at any level and stumps tend to be tapered.¹⁴⁷ Sensation tends to be normal and despite the presence of rings and amputations, function is generally good as proximally all soft tissue and skeletal anatomy remain unaffected.¹⁵³

Fusion of distal parts is also common in cases of constriction ring syndrome in the form of acrosyndactyly¹³⁵ (see Figure 49.2e). Distal fusions can be either simple or complex with bony fusions present between adjacent digits. Proximal fenestrations tend to lie characteristically distal to the level of the commissure.

Classification

A number of classification systems have been proposed for constriction ring syndrome. The most widely used of these is that described by Patterson,¹³⁵ a clinically useful system of four different groups which reflect a range of severity (Table 49.8).

Surgical principles and management

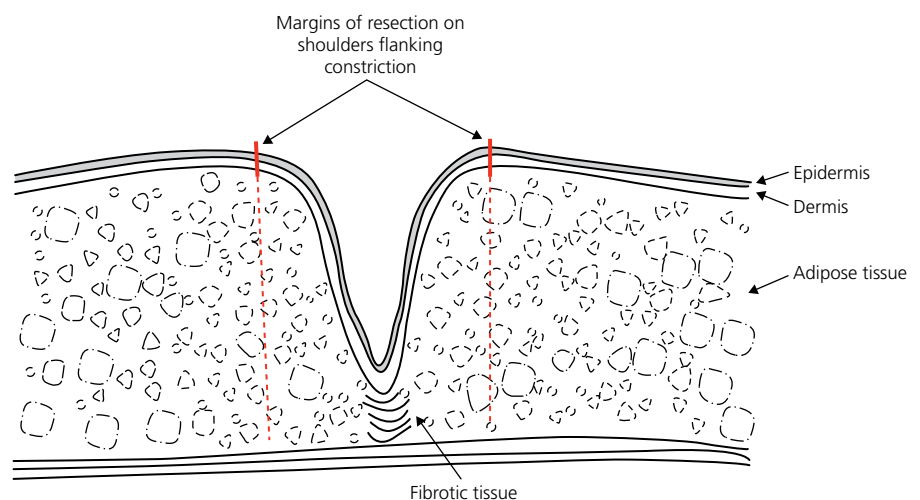
The timing of surgery is dependent on the type and severity of presentation. In the neonatal period the main treatment that may be required is the division of small soft tissue bridges between digits and the thumb to ensure unrestricted growth.¹³⁴ In some instances it may be possible to see fibrous bands encircling the fingers and toes of newborns. These can be simply removed or unwound to release the tethered digits.¹⁵⁴ Occasionally, ring constrictions can produce severe distal oedema and vascular compromise, particularly in cases of delayed healing in the postnatal period. These cases represent an emergency situation which requires expeditious decompression in the early neonatal period in order to prevent impending tissue ischaemia.¹⁵⁵ Hemi-circumferential decompression can yield a dramatic improvement in the status of the distal extremity. Once the decompression has had time to take effect, resultant redundant skin and the remaining half of the ring can be dealt with as a delayed second stage.

Simple constriction rings can be dealt with non-urgently prior to school age using principles described below. Syndactyly separation is best carried out early if it involves the border digits

Table 49.8 Classification of constriction ring syndrome¹³⁵

Grade	Description
Type 1	Simple ring constrictions
Type 2	Ring constrictions accompanied by deformity of the distal part with or without lymphoedema
Type 3	Ring constrictions accompanied by fusions of distal parts ranging from mild to severe acrosyndactyly
Type 4	Intrauterine amputations

Figure 49.22 Schematic demonstrating excision margins for correction of ring constriction. Transverse incisions are made on the shoulders flanking the constriction to ensure complete removal of all scarred tissue from within the ring.



and is tethering growth. Otherwise it can be dealt with after 1 year of age. Bilateral procedures and those treating upper and lower limbs simultaneously are possible but best performed before the child is ambulatory. More complex procedures such as toe-to-hand transfers, skeletal lengthening and digital transpositions can be delayed to optimize patient and family compliance and the size of surgical anatomy.¹³⁴

Methods of constriction ring correction

Traditional methods of correction involved excision of grooves, mobilization of adipose tissue and re-approximation of skin using Z-plasty- or W-plasty-based techniques.^{37,156} However, resultant scars have a tendency to indent with time and occasional partial loss of flaps and resultant delayed healing can result in a conspicuous depressed saw tooth appearance.¹⁵³

The ideal correction of a deep constriction ring should address the tight scar at the base of the indentation, atrophic tissue along the side walls and indurated fatty tissue within the distal swollen segment.¹³⁴ Transverse incisions are first marked on the either side of the depressed area on the limb or digit. These are marked on the shoulders flanking the constriction to ensure complete removal of all scarred tissue from within the ring (Figure 49.22). Gentle compression of one marked side against its opposing unmarked partner guides correct incision placement. All scarred tissue is then excised from within the depth of the ring, preserving large dorsal veins, volar neurovascular structures and tendons. Excess fatty tissue, usually most apparent distally, can then be excised and the remaining deeper tissue can be redraped to fill the annular contour defect. In this way final closure of the layers can be staggered to minimize the effects vertical scar contracture. If the ring is circumferential, one or two Z-plasties can be incorporated to break up the scar and are best placed in the mid-lateral axes to avoid the most socially visible areas of the limb or digit.¹⁵³

Toe-to-hand transfers

Constriction ring amputations of the thumb and digits are ideal candidates for vascularized toe-to-hand transfers as proximal anatomy including the neurovasculature is normal.^{106,157,158} These techniques can dramatically improve hand function through the reconstruction of a sensate mobile thumb or digits which will show a degree of commensurate growth with the child^{159,160} (Figure 49.23). However, this approach can occasionally be limited by a lack of usable toes in the context of constriction ring syndrome.

Distraction lengthening

An alternative approach is the use of distraction lengthening.^{161,162} This method is very effective at restoring a functional prehensile unit but can be very involved process that requires a great deal of patient and family commitment. Indications for surgery must be carefully considered.¹⁶³ This method appears to be more effective when used at the metacarpal and forearm levels as the distracted tissue is surrounded by more soft tissue



Figure 49.23 Image of a free vascularized toe-to-hand transfer to reconstruct a thumb intrauterine auto-amputation secondary to constriction ring syndrome.

with greater vascularity.¹³⁴ Distraction at the phalangeal level is possible but is extremely challenging due to problems of poor skeletal stock and soft tissue coverage.

Digital transposition

Transposition of a redundant digital tip based on its neurovascular pedicle as an 'on-top' plasty including its proximal phalanx can lengthen digits, improving length for prehension.¹⁶⁴ Transposition of a short non-functioning index finger in this way can also broaden a first webspace.

Arthrogryposis

Arthrogryposis (from the Greek *arthron*, joint, *grint*, hooking) is an umbrella term encompassing more than 300 disorders in which there are congenital contractures affecting at least two or more different areas of the body.^{165,166} The overall prevalence of arthrogryposis is around 1 in 3000 live births.¹⁶⁷ The inheritance, natural history, optimum management and prognosis varies across the disorders. Therefore ensuring a specific diagnosis and individualized treatment plan is key.^{166,168} When the results of neurological examination are normal, the most common causes are amyoplasia, one of the distal arthrogryptic syndromes, a generalized connective tissue disorder or fetal crowding.¹⁶⁸ An abnormal neurological examination points towards a CNS disorder or a neuromuscular disease (peripheral nervous system, motor endplate or muscle).¹⁶⁸

Amyoplasia, also known as classic arthrogryposis or arthrogryposis multiplex congenital (AMC), is a specific disease entity characterized by congenital joint contractures and muscle

wasting without evidence of any progressive neuromuscular disease. Distal arthrogryposis exhibits primarily hand and foot involvement but is a heterogeneous subset of autosomal dominant disorders.^{168–170} For the purposes of this chapter we will focus on amyoplasia.

Aetiology

Amyoplasia is sporadic, occurring in 1 in 10 000 live births.¹⁶⁶ Its exact aetiology remains unclear but evidence points towards a likely cause being patchy damage of the anterior horn cells of the spinal cord in the developing fetus.¹⁷¹ Genetic factors are not significant as twin cohort studies have demonstrated discordance¹⁶⁹ and no clear environmental factors have been identified.¹⁶⁷

Clinical findings

The vast majority have both upper limbs and both lower limbs affected (84%). They appear fusiform, wasted and without flexion creases (Figure 49.24). Lower limb involvement alone occurs in 11% and upper limb involvement alone in 5%.¹⁷² The joint contractures tend to be symmetrical. Characteristic features of amyoplasia include the following:^{169,173}



Figure 49.24 Image of patient demonstrating the common features of amyoplasia. The upper limb is internally rotated at the shoulder with the elbows extended, forearms pronated and the wrists flexed and ulnar deviated.

- *Upper limbs* (Figure 49.24)
 - Shoulders adducted and internally rotated
 - Elbows extended
 - Forearms pronated
 - Wrists flexed and ulnarly deviated
 - Thumbs adducted, laxity of UCL, absence of abductor pollicis brevis
 - Fingers partially flexed
- *Lower limbs*
 - Hips flexed and abducted with dislocation of one or both sides in 30%
 - Knee usually fixed in extension
 - Feet held in rigid equinovarus contractures in the majority
- *Other common features*
 - Deformities of face and jaw: asymmetry, flat nasal bridge, haemangiomas (frontal midline capillary haemangioma in 90% of those with all limbs involved), micrognathia and trismus
 - Genital deformities, umbilical or inguinal hernias are common.

Surgical principles and management

Most often affected children are of above average intelligence with the potential for independence.^{173,174} The priorities are communication, activities of daily living and mobilization.¹⁷⁵

Timing of surgery

We advocate a coordinated multidisciplinary approach parallel upper and lower limb surgery where possible to reduce the number of operations the child has to endure and ensuring greatest benefit especially as in both early surgery confers a better outcomes. A protracted course of lower limb surgery focused on ambulation alone, which may involve addressing clubfeet, knee contractions and hip dislocations for example, may result in significant delays to upper limb treatment.¹⁷⁶

Aims of treatment of the upper limb

The aims of treatment are to:

- maintain and improve joint range of motion, focusing on passive as this may improve function with passive assistance manoeuvres;
- maintain and improve muscle strength;
- preserve bimanual function; and
- optimize upper limb positioning to reach table tops.

These can be achieved initially with gentle manipulation and splintage with the help of occupational and physical therapy. Occupational therapy also teaches adaptive skills, trains caregivers to provide home therapy and tailors assistive equipment to meet individual patient needs.^{92,177} Therapy is an ongoing process which in the majority of patients extends at least into their teenage years.¹⁷⁸ Surgical intervention in the upper limb is recommended for fixed joint contractures that interfere with upper limb function.

Surgical options by region

Shoulder

This is rarely indicated unless there is significant shoulder internal rotation to preclude functional positioning of the limb in space. In these cases a humeral rotational osteotomy to around 45° will facilitate midline functioning of the hand.¹⁷⁴

Elbow

The main goal is to enable elbow flexion to allow hand-to-mouth reach. Improvement of passive range allows assistance techniques such as trunk swaying and table top propping. A posterior elbow joint release with a Z- or W-plasty to regain 90° of passive flexion with a tricepsplasty works well.¹⁶⁵ If active flexion is poor, an elbow flexorplasty transfer can be carried out at the same time. A variety of different options have been described for this purpose including the use of pectoralis major, latissimus dorsi¹⁶⁵ and triceps.¹⁷⁹ However, care must be taken to avoid development of a unwanted flexion contracture that can impair of pre-existing function. In this regard, transfer of the long head of triceps alone as a functional unit has been shown to produce reliable anti-gravity flexion while preserving active extension with minimal donor site morbidity.¹⁸⁰

Wrist

Early carpectomy prior to ossification of the carpal bones facilitates removal of a trapezoidal wedge from the mid carpus without damage to the radius or ulnar articular surfaces. This resection allows for correction of the wrist flexion contracture to around 40° of hyperextension and relaxes volar soft tissues, including the neurovascular structures and flexor tendons. This permits the fingers to adopt a more functional flexor-empowered position.¹⁸¹ The wrist position is initially maintained with K-wire fixation and recurrence is prevented through the transfer of the flexor carpi ulnaris and radial tendons to augment the dorsal pull on the metacarpals. Early surgery achieves more active flexion and allows greater bone remodelling.^{165,181}

Thumb

Patients with tight adduction contractures who do not respond to stretching and splintage may benefit from a first webspace release and either an extensor indicis transfer to improve extension or an epiphyseal sparing chondrodesis of the MCP joint. In some cases an opposition transfer is required at the same time.¹⁷⁴

Fingers

Finger position can be significantly improved with early stretching and splintage. Further improvement can be achieved through functional repositioning of the wrist contracture through wedge carpectomy, as mentioned above. In rare instances digital flexion, contractures require surgical release with full-thickness skin graft reconstruction.¹⁶⁵

Trigger thumb

Trigger thumb occurs due to obstruction of the flexor pollicis longus tendon at the A1 pulley as a result of stenosing tenovaginitis.¹⁸² Unlike the intermittent triggering seen in adult cases, children most commonly present with their thumbs locked in flexion at the IP joint. A small minority present locked in extension.

Notta's node is palpable proximal to the A1 pulley in a fixed flexion deformity. Most authors view this as secondary to bunching up of the tendon, as it appears to resolve following A1 release, rather than as a primary lesion. When the thumb is locked in extension, the failure to glide through the A1 pulley occurs distal to proximal, with Notta's node abutting the distal end.

Aetiology and presentation

Trigger thumb usually presents between 6 months and 2 years. There is debate as to whether this is in fact an acquired rather than congenital condition. Most studies reporting presence at birth depend upon a retrospective history from the parents. Prospective studies with early postnatal assessment report very few cases.^{183,184} However, diagnosis in the newborn is unreliable as they tend to clasp their thumb, and clicking or triggering is more difficult to elicit in a child than in an adult. A congenital basis is supported by the presence of trigger thumb in twins, the frequency of bilateral trigger thumb (25%) and the association with Trisomy 13.^{185–188} Trigger thumbs occur 14 times more frequently than trigger fingers. It is usually pain free, unless attempts are made to straighten the digit. A missed diagnosis of dislocation may be made with rapid recurrence following attempted reduction.

Surgical principles and management

Trigger thumbs tend not to resolve spontaneously in most cases. However there is some evidence that resolution is higher in those diagnosed prior to the age of six months.¹⁸⁸ Trigger fingers have even higher rates of spontaneous resolution. This recovery may be assisted by splinting and passive stretching programmes. However, we have found compliance difficult in this age group. Furthermore, we do not advocate treatment with steroid injections in paediatric cases.

Surgical release of the A1 pulley of the thumb is very successful.¹⁸⁹ We advise release prior to the age of 3 years,¹⁸⁸ due to a risk of permanent joint deformity. Access is via a transverse incision at the base of the thumb within a skin crease. Care must be taken to avoid damage to neurovascular bundles, which tend to sit more anteriorly when compared to the digits. However, direct visualization of the bundles is not required, only care in their protection. The radial part of the A1 pulley is divided under direct vision to allow unobstructed tendon gliding and to prevent damage to the oblique pulley. Trigger fingers, however, usually do not respond to A1 division alone, requiring sacrifice of one slip of FDS also.

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CHAPTER 50

Finger tip injuries

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Introduction

The hand is in harm's way. It is at the frontier of all work and contact with the environment. The finger tips, as a collective functional unit, bear the statistical brunt of such injuries. Hand injuries constitute the single most frequent surgical presentation in an average emergency department.¹

The hand is unique in its dual role as a considerable gatherer of information about the environment and also as an executor. Unlike the other major sensory organ, the eye, the hand is also able to act on the information gathered, constantly modifying action by feedback of sensation and proprioception. The gathering of information about the environment is especially developed in pulp and palmar digital skin, which is not to be regarded merely as skin covering, but as a specialized organ in its own right. The dense population of sensory end organs is housed in skin with specialized, mechanical properties. The skin is robust and yet deformable – the nail complex and the skeleton provide a fixed stop, against which the pulp and palmar skin complex are deformed; without such a rigid stop, one would obtain less meaningful information about the contour and texture of a held object or touched surface.

The aesthetic importance of the hand is not to be underestimated and is an important consideration in all reconstruction. Irrespective of custom and attire, in virtually all societies, the hand is always on display (the hand is exposed even in societies where the face is concealed). The back of the hand, in particular, is a significant aesthetic unit. In this regard, function and appearance are closely interlinked. The hand which behaves fluently and efficiently attracts little attention, even if it carries significant scar, or is missing anatomical parts. Smooth, healthy-looking skin cover, as well as fluid, natural behaviour form important aspects of social acceptability. Hand gestures reinforce facial expression and in some spheres, hand movement and posture constitute a very specific vocabulary, interpretable by those who converse in that language. Aside from the obvious example of signing for the deaf, the orchestra conductor's hand gestures speak a clear language to musicians and culture-specific hand

gestures convey meaning over distance beyond that possible through sound. Convention and social context convey specific messages with hand gestures – the extended index finger held vertically over the lips is universally interpreted as a request for quiet, for silence. It is also relevant that the hand is the most frequent acceptable point of contact; in most societies, the handshake is considered a permissible intrusion into personal space, and a permitted form of contact, irrespective of the difference in social standing of those shaking hands.

The hand is an integral part of one's identity for any individual but specific professions imbue the hand with an additional importance. The musician, for instance, has a special relationship with the hand since this forms an integral part of his or her identity and 'voice'. The same can be said of the craftsman or in sports that use the hand. Although these aspects form an important element of management, one must not overrate the contribution of profession to the decision-making process. Although some methods of reconstruction may entail an unacceptable period off work for, say, the manual worker, whose main concern may be for strength and durability, all will require a large measure of pain-free, sensate dexterity. Irrespective of work or leisure pursuits, all will still need to perform delicate, precise tasks, such as writing and doing up fastenings and buttons, touching spouse and offspring. Most will be disturbed by an unattractive extremity on display.

Since the hand is the part most frequently involved in work-related injuries,² the issue of personal injury claims and the cloud of possible compensation frequently hangs over the outcome of repair and reconstruction.

In the case of injuries in children, the growing hand presents special challenges on a number of fronts. Any repair must take into account the facilitation of growth of soft tissues and skeletal elements. Scarring and contracture, a perennial problem in the hand, assume an added importance. To these factors must be added the factor of parental anxiety and elements of guilt and regret centred on the occurrence of the injury in the first place. In general, the management of the child's hand involves the management of the family.

These various factors should all figure in the assessment of injury, the 'feel' of patient compliance and cooperation, the choice and planning of repair and reconstruction and the required aftercare. All too often these injuries are downgraded in the queue of surgical priorities – many still consider that 'all soft tissue injuries of the pulp heal' without regard to the quality of function of such healing. Injuries that can lead directly to significant loss of function, persistent pain and compromised appearance are still, regrettably, considered trivial and merit little surgical input. They are often misguidedly delegated to inexperienced members of the surgical team with little appreciation of their complexity and functional significance.

Assessment

The challenge of reconstruction in the hand lies primarily in the assessment and decision making, not in the technique, however immaculately executed. In assessing any injury (and the patient) one brings to bear experience and a big repertoire so that, as far as possible, questions are answered before the start of surgery. This is not to say that plans may not change when the examination reveals more detail, but it is the planning and selection of technique that is the focus and essential to a good outcome.

In the field of reconstruction, there are no recipes, only principles. In the management of reconstruction challenges of the digits, one vital principle is the appreciation that not all areas of the hand are of equal functional value. Observation of the hand in action soon reveals that all digits have leading and trailing aspects. Although one would not suggest that any part of the hand is of lesser importance, in reconstructive terms these could also be considered 'high premium' and 'low premium' territories (Figure 50.1).

The leading edge is the more commonly used in contact with a held object or contact with a surface. The trailing edge is less often in contact, and tends to have less developed discriminatory abilities or needs. This principle is most true of the thumb. The ulnar pulp 'leads' and is involved in virtually all dextrous activity and certainly all precision actions, since it is this area which faces the long digits most effectively. By contrast, the radial pulp 'trails' during most tasks, coming into play with relatively unspecialized tasks such as tapping on a horizontal surface or keyboard. The principle is least true of the index, both of whose aspects are frequently involved in common activity.

Once this principle is accepted, one can start to assess the functional significance of a specific defect and the availability of suitable methods of reconstruction:

- High premium areas require the best possible reconstruction, preferably sensate, glabrous pulp skin taken if possible from the same digit.
- Injury to a low premium area may be reconstructed acceptably and reliably with relatively non-specialized cover, such as a skin graft or an insensate skin flap.

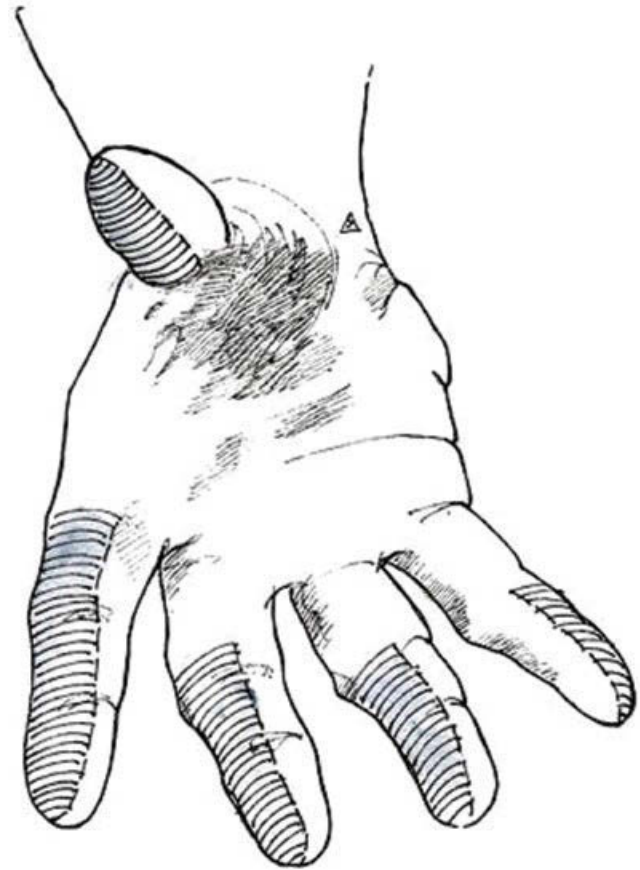


Figure 50.1 The leading aspects of the digits. Source: Donald Sammut. Reproduced with permission.

- Low premium areas can be considered for transfer to high premium areas, preferably on the same digit, as for instance the reconstruction of ulnar pulp in the thumb using radial pulp (Figure 50.2).

These observations are proposed as factors to be taken into account, and are certainly not absolute. They are among many that should be considered, including the requirements of the patient overall, likelihood of compliance, as well as special requirements for a particular patient, such as the playing of musical instruments or professional sport.

In few other areas of reconstruction is collaboration with the patient as essential. This element needs careful assessment if one is to obtain a good outcome, which, in this situation, is the maximum restoration of function (and hence appearance) and an acceptance by the patient of any limitations consequent on the injury.

Detailed informed consent also needs to take into account the time and effort investment required of the patient, particularly in the more complex reconstructions. The extension of the site of injury and/or the involvement of distant donor sites are all factors to be included in the detail of the process of informed consent.

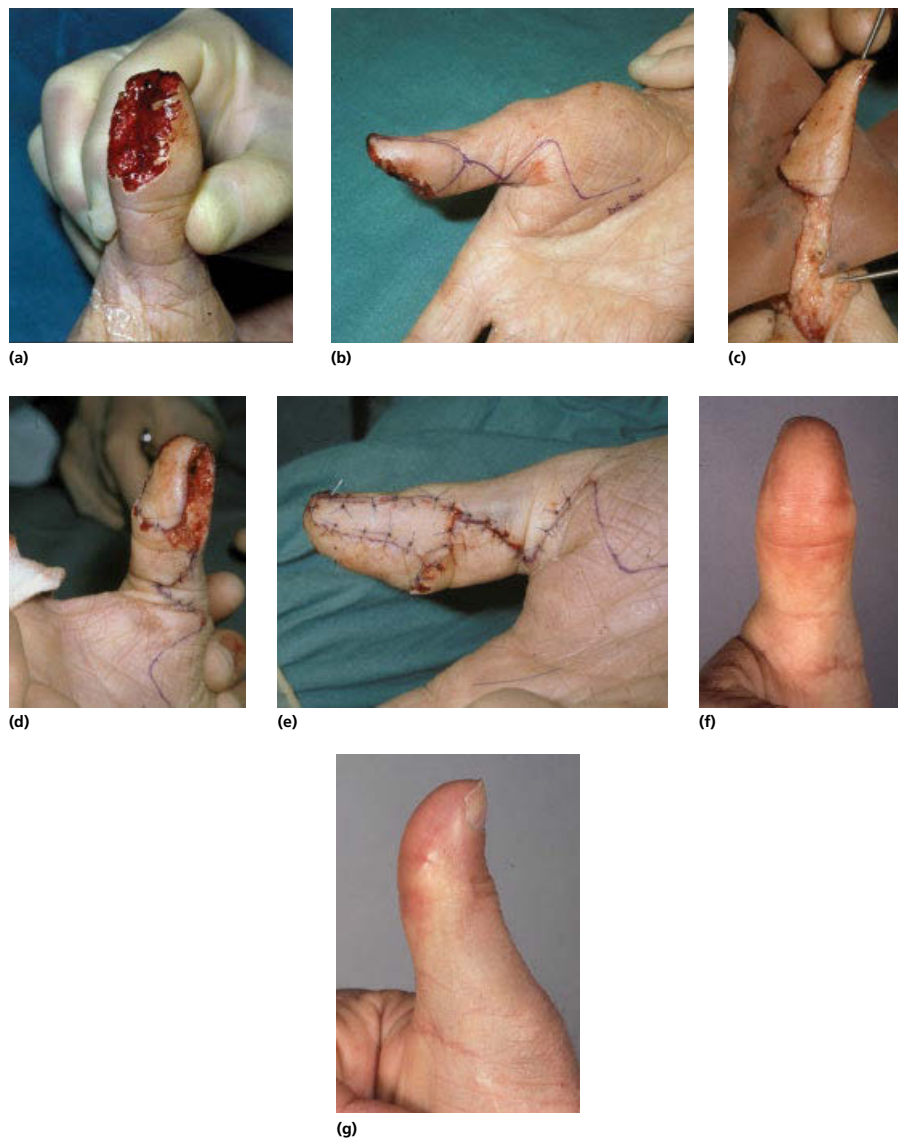


Figure 50.2 Pulp exchange flap. (a) The defect involves the entire leading (ulnar) surface of the thumb pulp. (b) The trailing (radial) aspect of pulp is intact and an island flap is designed on this pulp. (c) The flap is entirely islanded leaving ample fat around the nerve and artery. (d) The island is transposed to reconstruct the ulnar pulp. (e) The secondary defect is resurfaced with a skin graft, in this case a full-thickness graft taken from the amputated part, which was defatted. (f, g) Final result at three months. Source: Donald Sammut. Reproduced with permission.

Finally, in the process of assessment, the surgeon must also have a realistic and honest assessment of his or her capabilities. Many methods of reconstruction are attractive to the surgeon since they fulfill a natural mechanical neatness and challenge of construct. The seduction of such methods must be resisted in the absence of expertise or adequate guidance (and, preferably, also assistance) by those more experienced.

It cannot be overemphasized that the method selected must suit the defect and the level of expertise of the surgeon. In this regard one must mention that it may be entirely appropriate to select a temporary method known to be insufficient for long-term wear and restoration of function (such as a dressing or a split skin graft on pulp), leaving all options open for reconstruc-

tion with the availability of appropriate expertise. If this approach is selected, it is wise to present this to the patient as a two-stage procedure so that he or she is prepared for the second surgery and it does not come as a 'salvage', or adjustment, of the first.

Healing by secondary intention

Both pulp and palmar skin have remarkable powers of regeneration, as evidenced by the reliable healing of the open palm technique advocated by McCash.³ Large defects of pulp will heal reliably provided a number of important criteria are observed:

- The defect must be suited to this method. No vital structure, such as tendon, bone, or joint, must be exposed. Severely contaminated or crush defects must be thoroughly debrided of all contamination or necrosis.
- The hand must be elevated.
- The patient must understand that the method requires careful adherence to instruction and compliance with treatment (many patients will only remember that 'It needs no surgery' and this is rapidly translated into 'It needs no care').
- Non-adherent dressings require regular change, preferably on alternate days. Exception is made for children where it is routine to leave a dressing undisturbed for a week, after which the wound can be left exposed, in order to avoid the trauma of painful dressings.

The dressing in direct contact with the wound is a matter of personal preference. tulle gras (paraffin-impregnated gauze) is traditional. It is cheap and easily available or can be improvised, but dries rapidly and tends to adhere more than one might expect. Many superior alternatives are available, such as thin, perforated silicone sheeting (e.g. Mepitel®, Mölnlycke Healthcare), which is resistant to all adherence. Chitin (Beschitin®, Unitika) and other natural substances have their advocates. The important aspects are: non-adherence, a barrier to contamination and assiduous care and compliance.

Surgical options for cover

Amputation

In cases where a significant part of the digit is crushed, with accompanying disruption of the nail complex and/or the distal interphalangeal joint, amputation with closure of locally available skin, may be the indicated option. In this situation, it may be appropriate to elect to ablate any nail elements which, regenerating, will be unsupported, non-adherent, unattractive or likely to lead to functional problems such as snagging on clothing.

Much morbidity results from amputation performed without proper regard to indications and points of technique. There is a natural tendency to preserve length at all costs. The aim should be to achieve a non-tender digit covered by good-quality skin, irrespective of length. It is better to elect for a slightly shorter length, with good-quality, mobile skin cover, than to preserve length at all costs but with a tight and painful digit end. A digit with tense, painful skin cover may well be excluded from function, resulting in an effective loss of all useful length. This is particularly true of the index, which is readily substituted by the middle finger and regularly requires subsequent revision. It is a wise surgeon who seeks to prevent the cycle of pain, tenderness and exclusion.

There are three generally indicative levels of amputation of the finger tip (Figure 50.3):

- **Level 1** involves loss of skin and only a minimal loss of nail and distal phalanx. In this case all of the tip vital structures are preserved and skin cover is indicated.

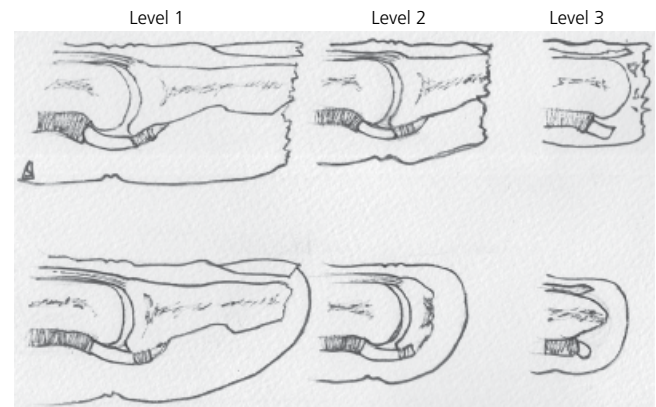


Figure 50.3 The levels of amputation. Level 1: There is a skin defect with minimal loss of nail and no significant loss of supporting distal phalanx. Indication: Dressing or, if bone exposed, advancement of skin flap. Level 2: A significant portion of nail and supporting bone is lost. Indication: Ablation of nail remnant and trimming of bone so as to preserve the next vital structures, the insertion of extensor and flexor, while achieving tension free cover (direct closure or flap). Level 3: The tendon insertions are disrupted. Indication: Trimming of bone and removal of articular cartilage to achieve skin cover with appropriate cover (direct closure or flap). Source: Donald Sammut. Reproduced with permission.

- **Level 2** involves loss of skin and most of the distal phalanx/nail complex. In this case the nail should be ablated and the next proximal structures to preserve are the tendon insertions. Just enough bone should be left to preserve these insertions which will preserve power and elements of proprioception.
- **Level 3** has lost all nail elements and the tendon insertions. The condyles of the middle phalanx can be removed to prevent an unattractive, spatulate stump shape. The phalanx is shortened to a level where tension-free, good skin cover can be achieved. One should not be tempted to anchor the tendons, or to suture them to each other. This invariably leads to an imbalance and most frequently to a flexion contracture.

All amputations require attention to the nerve ends. One should take the opportunity of any skeletal shortening to keep the nerve ends long and to transpose them dorsally, away from contact areas.

Options for skin cover

If skin cover is required, either *skin grafts* or *flap cover* can be used. Reconstruction most often involves moving tissue around from one site to another, with the added, vital, phrase 'and keeping it alive'. Blood supply is paramount. A skin graft requires a bed capable of angiogenesis, the essential process of 'take'. 'Take' is the establishment of a blood supply to support a graft. A flap requires no such bed – it brings its own blood supply and can be placed over any surface, including metalwork. Provided this principle is observed, the decision can then be taken on what form of cover will best restore function and structure. The concept of the

'reconstructive ladder,' with its implications that one chooses the simplest reconstruction available, is outdated – one chooses what is appropriate and best restores form and function.

It follows that the surgeon must have in his or her repertoire, as large a variety of reconstructions possible, in order to match the reconstruction to the defect and patient, and not the other way around. This, incidentally, is another strong reason for involvement of experienced, senior surgeons. All too often this process is left to those with one or two methods at disposal – this invariably results in the dispensation of a method which the surgeon knows, rather than that which is appropriate.

Skin grafts

Provided the bed will ensure take, a split skin graft provides excellent temporary cover or permanent cover of a 'low priority' area, including a secondary defect, from flap harvest. Many wounds involve crushing or bursting mechanisms and it is difficult, at first view, to be sure of viability unless one is wastefully radical with excision. In such situations, a flap may be the appropriate skin cover but its territory may itself lie in the area of trauma. Proceeding with immediate flap cover may lead to flap loss and failure with waste of a reconstructive option. A skin graft may provide comfort, initiate healing, resolve inflammation and will keep all other options open.

In some situations, skin graft provides good definitive cover. These include the trailing aspects of digits and the secondary defects from the raising of a flap. It need hardly be added that a flap donor source is chosen from an area that can be closed directly, can take a skin graft or can be left to heal by secondary intention. Skin grafts do not provide good definitive cover in the pulp, where accurate, pain-free sensibility and good mechanical properties are required. In such a situation, the skin graft will lead to unacceptable hyperaesthesia, poor cosmesis and troublesome long-term fissuring.

The choice between split skin graft (SSG) or full-thickness skin graft (FTSG) is a matter of balance of various factors. A full-thickness graft requires a better quality bed, since there is a larger cell population for nourish. Take of a split skin graft is more reliable on a poorer bed. Conversely, split skin grafts contract more significantly than full-thickness grafts – an important factor in cover of mobile areas crossing joint creases and anywhere in the growing hand.

Points of technique

Split skin graft

- *Donor site:* Small grafts can be taken from the hypotenar mound. The skin here is thick enough to permit harvest of the dermis while replacing the epidermis.⁴ Larger grafts can be taken from the proximal inner aspect of the forearm, where it can be unattractive and/or tender or from the thigh. With both SSG and FTSG, the opportunity to harvest skin, or indeed any other element, from a finger which is to be discarded should not be lost (the concept of the 'bank digit').⁵

- A guarded skin graft knife is used, such as the Watson, Humby, Silvers knife or, for larger areas, the electric dermatome.

Full-thickness graft

- *Donor site:* Avoid harvest from the wrist skin crease – scars here can be misinterpreted as the outcome of self-inflicted wounds and can prove tender and troublesome with the wearing of watches or bracelets. The cubital fossa provides ample skin for most needs in the hand and digits. It is usually hairless, and is in the same bloodless field, which speeds up proceedings and makes harvest more accurate. Hairy donor sites should be avoided – harvest from the groin crease in children before the distribution of pubic hair is evident provides a common pitfall.
- The graft should be taken thin, without subcutaneous fat, or thinned just before final detachment.
- The graft should be sutured tense over the recipient site (suture of a graft provides the only instance when suture under tension is permissible) and larger areas require a tie-over dressing.

Finally, one may mention the composite graft as a form of skin grafting. This is particularly applicable in the child's hand, when the section is clean and the segment uncrushed. It is more successful the fresher the injury and the younger the child. The amputated part should be applied rapidly, with minimal dissection of either surface and with as accurate alignment of landmarks, such as nail fold and even print ridges. Parents are warned that the method is not guaranteed and that reconstruction may be required if it fails. The part often appears to necrose and perish, only to reveal accurate resurfacing some three weeks later when the carapace falls off or is peeled off.

Flap cover

Flap cover is indicated when bone, joint and tendon are exposed, or when metalwork needs cover. In some situations there is no choice but to apply a skin graft to intact tendon sheath or paratenon, or even direct to cancellous bone where, somewhat surprisingly, it frequently takes. These are relative indications: in general, exposure of such structures requires flap cover.

Flap cover is also indicated when deeper structures are not exposed and the defect is capable of nourishing a graft, but sensibility, mechanical quality or cosmesis are relevant requirements.

Flaps have been classified in various ways. In the hand, the basis of the blood supply and the presence of sensibility provide the most relevant classifications:

- *The basis of the blood supply:* Preservation of continuity of blood supply is paramount. This may be either left unidentified in the flap base (e.g. the Hueston flap⁶ or the cross finger flap⁷) or formally dissected free, as in the islanded flaps. In general, the latter type of designs are the more mobile and versatile, but demand a higher level of technical expertise.
- *The presence of sensibility:* For classification purposes, one may consider 'sensate' flaps those which maintain pulp quality

Table 50.1 Classification of commonly available flaps

Relatively insensate flaps:	Kite flap Delayed flaps: cross finger, thenar, distant flaps (e.g. groin flaps, random flaps) Free venous flaps
Relatively sensate flaps:	V-Y advancements (e.g. Atasoy flap) Thenar advancement Bipedicle homodigital Homodigital islands ^{10,11,12} Heterodigital ¹³ Free flaps (e.g. hemipulp transfer from toe or from a bank digit). The recovery of sensation is inversely proportional to age in these examples

and replace lost pulp with sensate pulp. One should not consider adequately 'sensate' the cross finger flap which regains some sensibility despite the numerous reports of recovery of 'excellent' two-point discrimination.^{8,9} Nor should one consider that the transfer of an island of dorsal skin (e.g. the kite flap) replaces adequate pulp quality sensibility. On this basis, the commonly available flaps can be classified as shown in Table 50.1

This definition of sensate/non-sensate is a working classification serving to underline the importance of replacing like with like. It sorts out flaps rather too harshly on the basis of restoration of pulp quality sensation. Many studies report recovery of two-point discrimination approaching normal in flaps that cannot transfer nerve continuity, such as the cross finger flap.^{9,14} In this context, one should note that our methods of assessment of sensory discrimination remain relatively crude and subject to significant operator variability and subjectivity.

The description of flap qualities is not intended to label any flap superior or inferior to others. All are useful in context and the wise reconstructive surgeon will master all, and will amass a large repertoire on which to draw when analysing each individual defect. It cannot be repeated too often that each defect will have qualities which will be met by this or that flap, and one should have all at one's disposal so as to be able to produce a flap that matches the defect, not the other way around. In this regard, one should resist the tendency to refer to this or that flap as a 'workhorse' or to apply the same, favourite, flap to a wide variety of defects. Conversely, it is also ill advised for the inexperienced to be captivated by a clever flap and to resolve to 'try' it.

The account below cannot be exhaustive. Representative flaps in each category have been selected which illustrate the principles of selection, technique, flap attributes which form part of the analysis, planning and execution of skin cover of the digit.

The kite flap (Figure 4)

The kite flap was originally designed with a wide base pedicle by Holevitch (Figure 50.4).¹⁵ Vilain and Michon developed the design, narrowing the pedicle to about 1 cm, including the vascular axis; this design was christened the 'flag flap'. It was

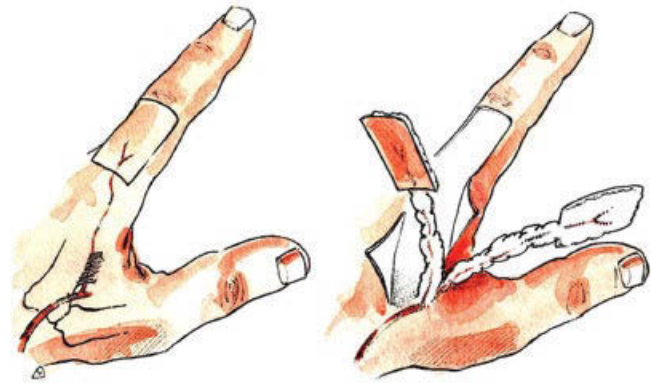


Figure 50.4 Kite flap raised on the dorsum of the index. Source: Donald Sammut. Reproduced with permission.

Foucher who islanded the skin flap, denuding the pedicle of skin and conferring a greater degree of mobility as well as the potential for tunnelling of the pedicle.¹⁶ The island of skin is raised in a plane just superficial to the extensor tendon paratenon (which must be left intact so as to receive a graft). The pedicle includes the first dorsal metacarpal artery, local veins, as may be found (usually plentiful), and any superficial branches of the radial nerve that are identifiable.

The radial nerve branches provide sensation but the density of end organs in the dorsal skin never matches the density required to provide pulp quality sensation and it is for this reason that this flap is included in the 'insensate' category.

Although it is tempting to use it for resurfacing of the leading edge of thumb pulp, its use should be reserved to areas with less demanding sensory qualities, such as the dorsum of the thumb and the radial aspect.

Important points of technique

- The first dorsal metacarpal artery should be identified early since it may follow a course part way through the substance or fascia of the first dorsal interosseous muscle.
- It is prudent to include any veins which can be gathered into the pedicle – a surfeit of veins makes for a healthier flap.
- Terminal branches of the radial cutaneous nerve are fine and must be sought and preserved.
- If the flap is tunneled, the tunnel must be generous and without sharp kinks in its course.
- The donor area must be carefully protected and the paratenon left intact so as to receive a graft.

Advantages

The technique is relatively unchallenging and the flap is robust, provided important points of technique are observed. The loss of skin from the dorsum of the index produces no functional impairment. Care with the dissection should ensure full and immediate take, so that the cosmetic defect is minimized. The pedicle is mobile and long and permits placement of the flap in a wide radius of reach.

Disadvantages

The quality of the thin, poorly sensate, transferred skin cannot replace glabrous skin with its myriad of end organs and special mechanical qualities. This limits its application. The transferred skin may be hairy, particularly in males and, in black skin, will be darker than palmar skin. The skin graft applied to the donor site may pigment.

Delayed staged flaps

Delayed flaps have a random design (i.e. they are not planned over a known vascular pedicle). They are inset into a recipient defect and their relative ischaemia (which can be encouraged by judicious clamping) encourages ingrowth of vessels from the recipient site. The flap can then be detached from the donor site.

The period required before the flap can be safely detached from its original vascular supply varies, but generally depends on the quality of blood supply in the recipient bed and the volume of flap that must be nourished. The principle of delay is discussed in more detail elsewhere.

The cross finger flap

Gurdin and Pangman⁷ are credited with the first description of this flap which uses a well-established principle. It is still one of the most commonly used (and abused) flaps used for skin cover in the digits. In principle, dorsal skin from a digit is raised and turned over like the page of a book (Figures 50.5 and 50.6). A palmar defect in an adjacent digit can be inset and receive cover. The secondary defect receives a graft. There are a number of variations, including the innervated neurovascular cross finger flap and the reverse dermis cross finger flap.

Important points of technique

- The recipient defect should be thoroughly debrided *before* planning of the flap.
- It is best, if possible, to design the flap over the dorsum of the finger between the interphalangeal joints.
- The flap must be raised carefully so as to preserve its dermal plexus but also to leave behind a graftable defect.
- The skin graft (usually full thickness) is best secured and inset into the donor defect before the flap is sutured to the recipient. It is easier to gain access to all the borders of the graft while the fingers are still unattached.

Advantages

The flap is reliable, simple to perform and versatile – the same digit can act as donor, giving a flap to a neighbour and as recipient, receiving a second flap from another neighbour (e.g. dorsum of middle finger to palmar index combined with dorsum index to palmar thumb).

Disadvantages

The enforced delay produces stiffness particularly in the proximal interphalangeal (PIP) joint of the recipient finger, which is kept flexed for some 2–3 weeks. Pigmented or hairy dorsal skin may be unsightly on the palmar surface (Figure 50.7).

A skin graft is required and may pigment. The flap is totally insensate unless modified in one of the described methods (e.g. Venkataswami), which are technically more demanding. Its lack of sensation should restrict its use to more proximal defects where the pulp has been preserved.

Distant delayed flaps

The limited indications and disadvantages of locally sourced delayed flaps hold true for distant flaps with the added problems of immobilization and difficulties with elevation of the hand. Nevertheless it is occasionally necessary to raise and use a random flap from abdomen or arm or an axial pattern, delayed flap from the groin. The groin flap has its application, delayed or



Figure 50.5 Cross finger flap. Source: Donald Sammut. Reproduced with permission.

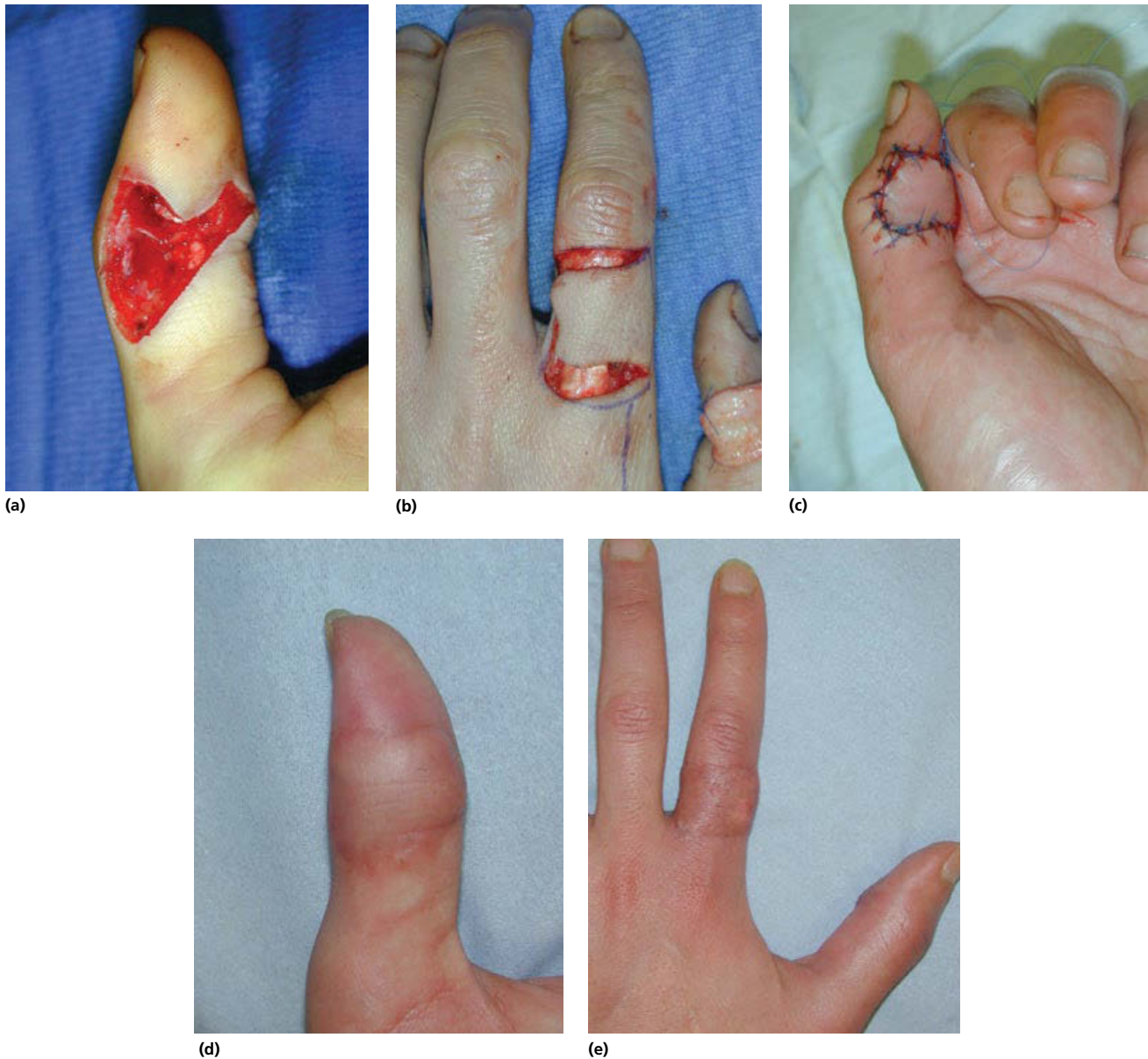


Figure 50.6 Cross finger flap. (a) The defect on the anterolateral part of the thumb. (b) The cross finger flap raised from the dorsum on the index finger, leaving behind the paratenon of the extensor tendon for skin grafting. (c) The flap is inset without tension, and the pedicle left attached for three weeks before division. (d, e) The healed flap on the thumb and the grafted donor site on the index finger. Source: Photos courtesy Peter Burge.

by immediate free transfer, in larger defects, especially in staged reconstructions such as multi-digit loss which may later receive free toe transfers, at which time it is a bonus to be able to transplant into good-quality trophic skin rather than dense scar.

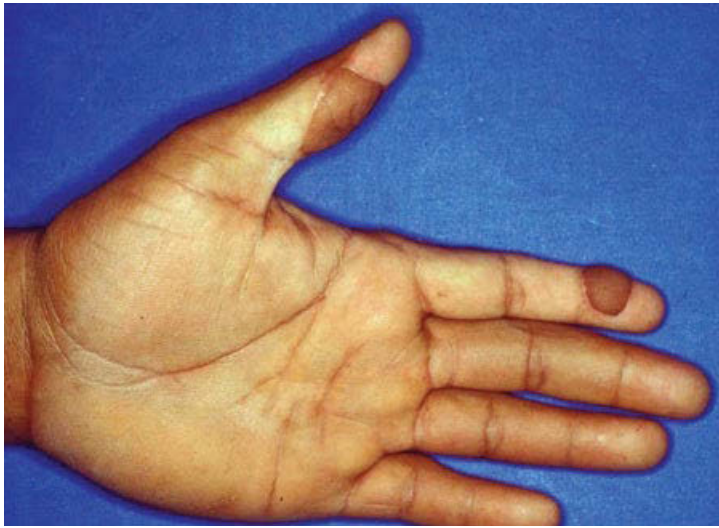
The Atasoy flap¹⁷

Some elements of the literature have likened this V-Y advancement of pulp flap described by Atasoy in 1970 to the Tranquili-Leali flap¹⁸ described in 1935 to which some even attribute precedence. There is no connection between the two other than the original triangular shape of the flap. The Tranquili-Leali flap involved division of both vascular pedicles and flap survival

depended on direct supply from the vertical connections between skin and deeper surfaces. This was sheared forward rather like a jelly and its advancement was very limited. In contrast, the Atasoy flap is a bipedicle island flap, raised on both, intact, vascular pedicles and nerves (Figure 50.8).

Important points of technique

- Selection is crucial. Only transverse or dorsally inclined defects should be considered. At least half of the distal phalanx should be preserved to provide adequate nail support.
- The width of the advancing edge must correspond precisely to the width of the nail plate. Too narrow a flap produces



(a)



(b)

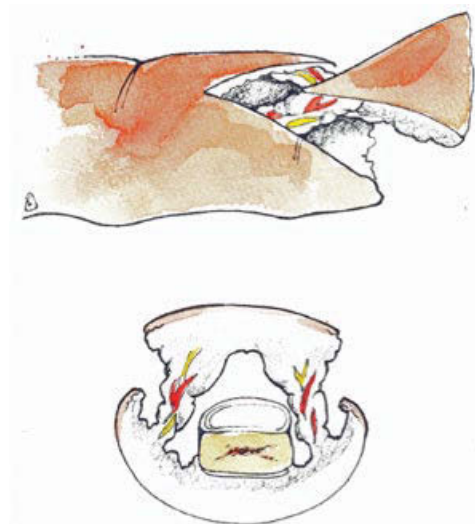
Figure 50.7 Pigmented cross finger flap. Dorsal, hairy, pigmented skin transposed to pale palmar skin. Source: Peter Burge. Reproduced with permission.



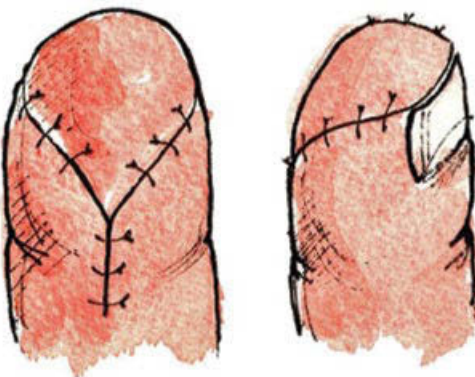
(a)



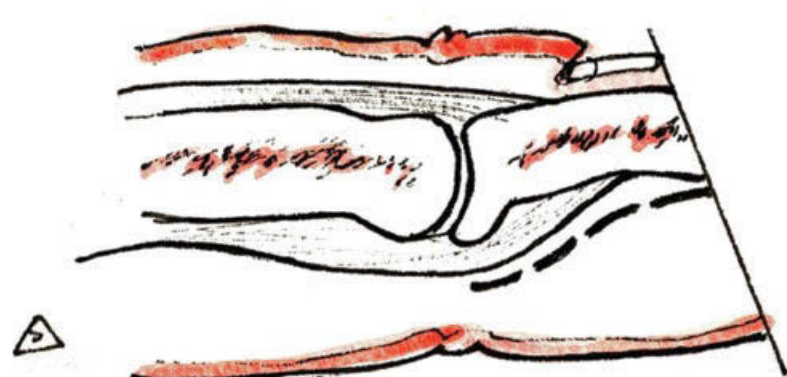
(b)



(c)



(d)



(e)

Figure 50.8 Atasoy flap. (a) The flap is raised as a V on palmar skin. The distal width of the flap must equal the width of the nail plate. (b) The digital bundles on either side are mobilized. (c) It is important that the flap be entirely islanded free from its bed and attached only by the pedicles with a generous sheath of fat. (d) The flap is advanced and inset. The secondary defect can be closed as a V-Y or left to heal. (e) In selecting an appropriate defect it is important that the amputation be angled transversely or, preferably, with a dorsal angulation. Source: Donald Sammut. Reproduced with permission.

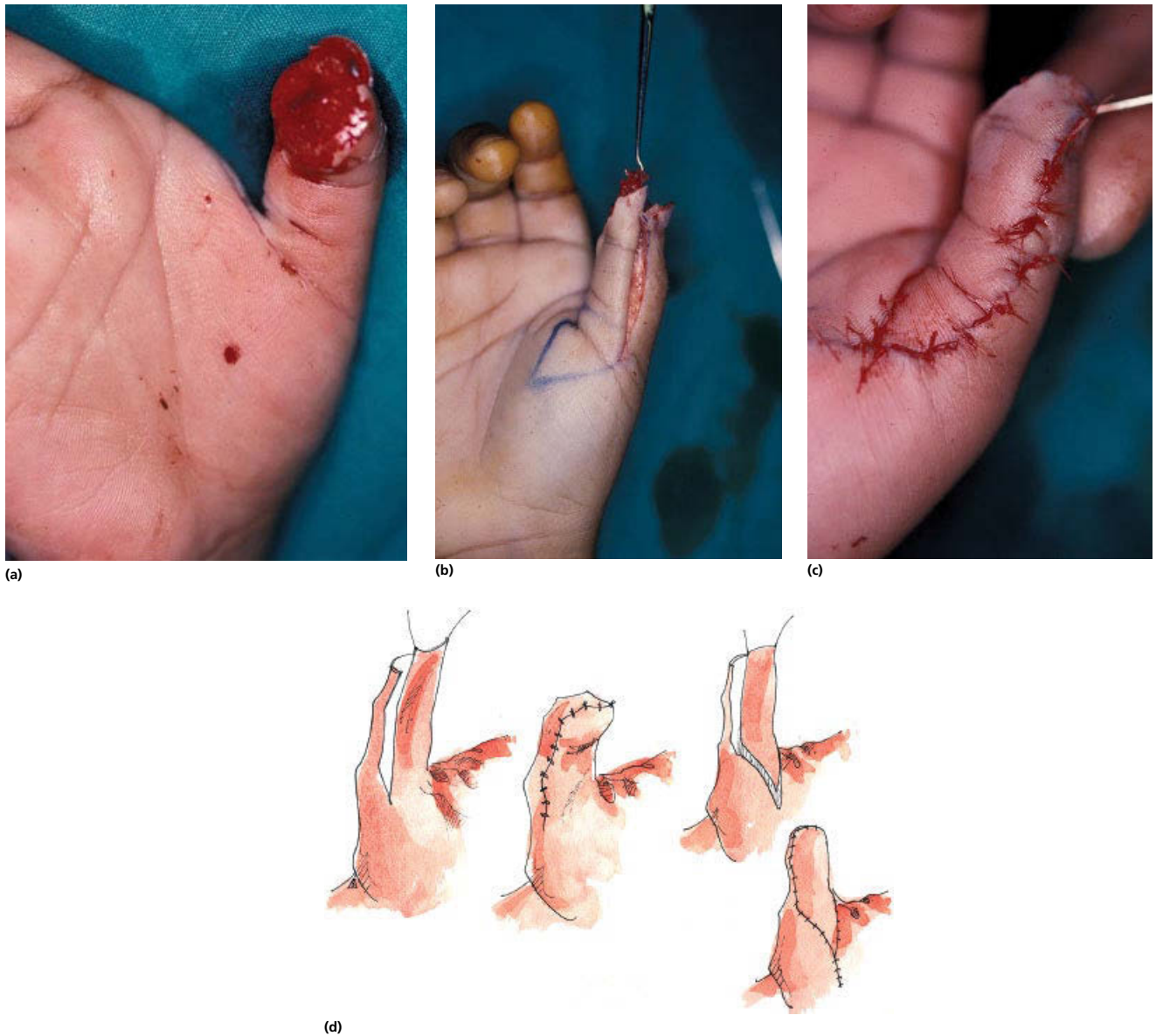


Figure 50.9 The Moberg flap. The flap is raised in the plane of the flexor pollicis longus sheath and includes both digital bundles. The original flap simply advanced by 'creep', aided by some flexion of the thumb, and with no secondary defect. Further advancement can be obtained by transecting (and islanding) the base (O'Brien), and grafting or closing as a V-Y (Carroll). (a) The defect. (b) The flap raised in classical design. (c) The Carroll V-Y modification to close. (d) Design of the classical Moberg flap and the Carroll modification (islanding the flap and closing the base as a V-Y). Source: Donald Sammut. Reproduced with permission.

unattractive 'shoulder pads' either side. Too wide a flap produces an unattractive spatulate result.

- The flap must be completely islanded (i.e. it must remain attached only by the neurovascular pedicle).
- The proximal V apex need not be limited to the distal interphalangeal (DIP) joint crease. The flap should be designed of the dimensions required and the crease line is an irrelevant landmark.
- If, postoperatively, there seems to be tension, or congestion, only the side sutures should be considered for release. The leading edge sutures must be left intact; their division will

result in retraction of the flap proximally, exposure of bone and healing with a tender, dimpled scar.

The Kutler flaps,¹⁹ double VY advancements with each flap on either side of the digit tip, are mentioned because they bear a superficial resemblance to the Atasoy V-Y but, once again, the main point in common is the shape. They are technically challenging and generally unreliable, not least because the anatomy of the vasculature on this area is variable. The vascular arcade in the pulp may be U-shaped, Y-shaped or H-shaped²⁰ and one cannot rely on an axial vessel on either side in the flap triangle. In practice, Kutler flaps are raised without islanding, left attached to

their bed, and teased forward to obtain modest advancement, a process made more difficult by the presence in the flap of Cleland's ligament, which must be teased apart or divided.

The thenar advancement flap

This flap consists of the entire palmar surface of the thumb, raised off the flexor sheath and skeleton, and stretched distally to cover a distal transverse defect (Figure 50.9). It enjoys the advantage of perfectly sensate, homodigital cover, which requires no re-education.

In essence, this flap 'creeps' distally, the division of septa and tethering fibres enabling the skin to cover a larger area by stretching. In practice, there is also an element of flexion of the interphalangeal joint in most instances, so that the defect comes to meet the flap as much as the flap advances to cover the defect. Flexion contracture of the interphalangeal joint is a common sequel to this flap and is, fortunately, not a significant functional impairment.

The flap, originally described by Moberg,²¹ is applicable only to the thumb, which enjoys a dual vasculature, dorsal and palmar, so that the two can be separated. Application in other, long digits, as advocated by Snow, runs a significant risk of necrosis of the dorsal skin.

Important elements of technique

- The neurovascular bundles in the thumb lie apposed to the flexor pollicis longus (FPL) tendon sheath either side and not down the midlateral lines.
- The parallel incisions must be strictly in the midlateral axis if one is to avoid subsequent flexion contracture.
- If the flap does not advance enough and/or is too tense, one can join the two parallel incisions on the palmar aspect, effectively islanding the flap on its bundles as advocated by O'Brien²² (Figure 50.10) or close the base as a V-Y (Carroll) (Figure 50.9c).

The advancement rotation flap

This flap is based on one digital bundle, without islanding, rotating to cover a terminal defect. It was described by Hueston⁶ (Figure 50.11) as a square, or rectangular flap, raised

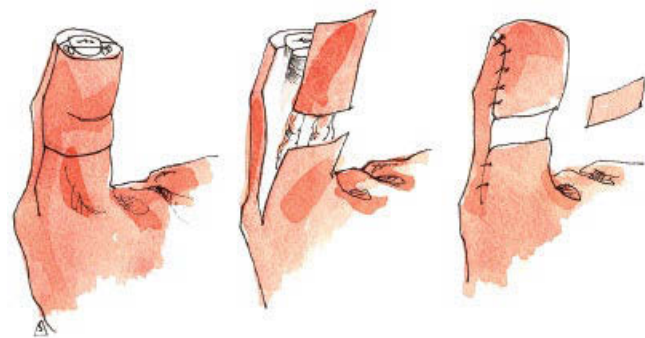


Figure 50.10 The O'Brien islanded flap. O'Brien modified the Moberg flap by transection of the base and islanding. Source: Donald Sammut. Reproduced with permission.

by an L-shaped incision. It excludes the neurovascular bundle from the leading edge and is nourished by the neurovascular bundle in the base. This means that the leading half of the flap is insensate.

Important points of technique

- Selection is best reserved for transverse amputations or for oblique loss slanted towards the base. This minimizes the required arc of travel.
- Cleland's ligament should be divided in the static base of the flap, since this base also is involved in the stretch and rotation required.
- The distal end of the base will produce a dog-ear and this should be dealt with in the manner described by Foucher, by excision of a square of skin *on the dorsum* (Figure 50.12) without reducing the width of the base pedicle.
- The secondary defect, a raw triangle at the proximal border, can be left to heal by secondary intention or may accept a split skin graft.
- Provided the underlying bone is not wide (for instance including phalangeal condyles) one should not trim the skin to coat the skeleton too closely. A looser skin pocket will contract to conform to the skeleton without tension.

The issue of loss of sensation of the leading half has been addressed in various ways:

- Souquet described a modification which included the leading neurovascular bundle in the flap and teasing it so as to permit some stretch. This maintains sensation but greatly limits the ease of rotation and movement (Figure 50.13).
- The flap can be based on the leading edge of a digit, so that the insensate half comes to be placed on the trailing edge and is of lesser functional consequence.
- The two techniques, Hueston and Souquet, can be combined in the manner described by Lloyd and Sammut (Figure 50.14),²³ which includes both bundles in the flap, but mobilizes the leading neurovascular bundle to obtain better movement.

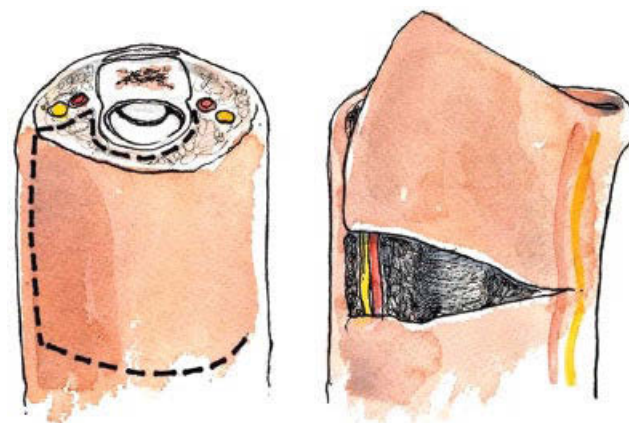


Figure 50.11 The Hueston flap. The flap is square, raised palmar to the leading edge neurovascular bundle, in the plane of the flexor sheath and nourished by the neurovascular bundle in its base. The leading half of the flap is insensate since the leading edge bundle is not included. Source: Donald Sammut. Reproduced with permission.

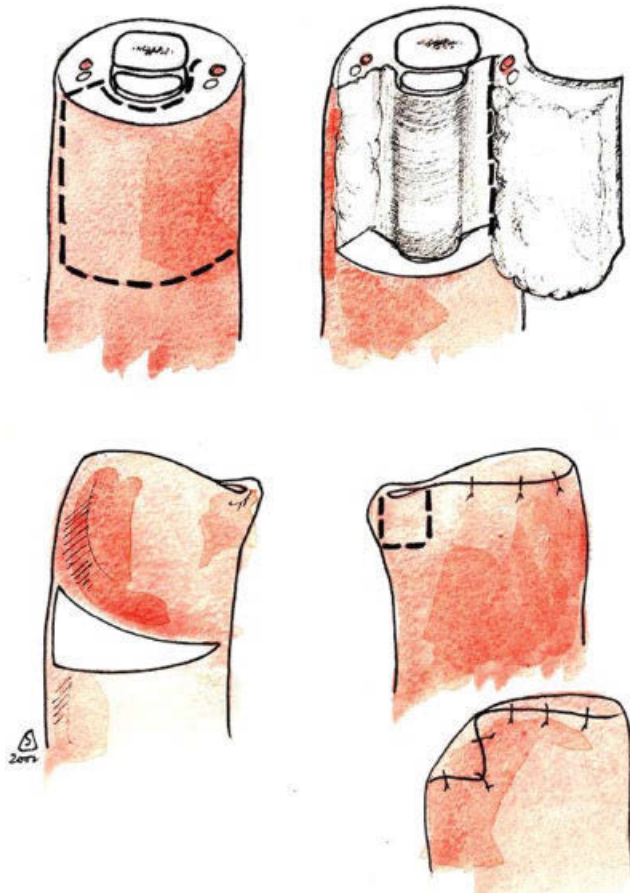


Figure 50.12 Foucher elimination of Hueston dog-ear. Source: Donald Sammut. Reproduced with permission.



Figure 50.13 The Souquet flap. Like the Hueston flap, the design is square but includes the neurovascular bundle in the leading edge. The flap will rotate less but remains sensate in all areas. Source: Donald Sammut. Reproduced with permission.

The island flaps, homodigital and heterodigital

Numerous variations of islanded flaps have been described^{10–12} but the principle is the same as that originally designed by Littler, who, in 1953, raised an island of skin from the ring finger

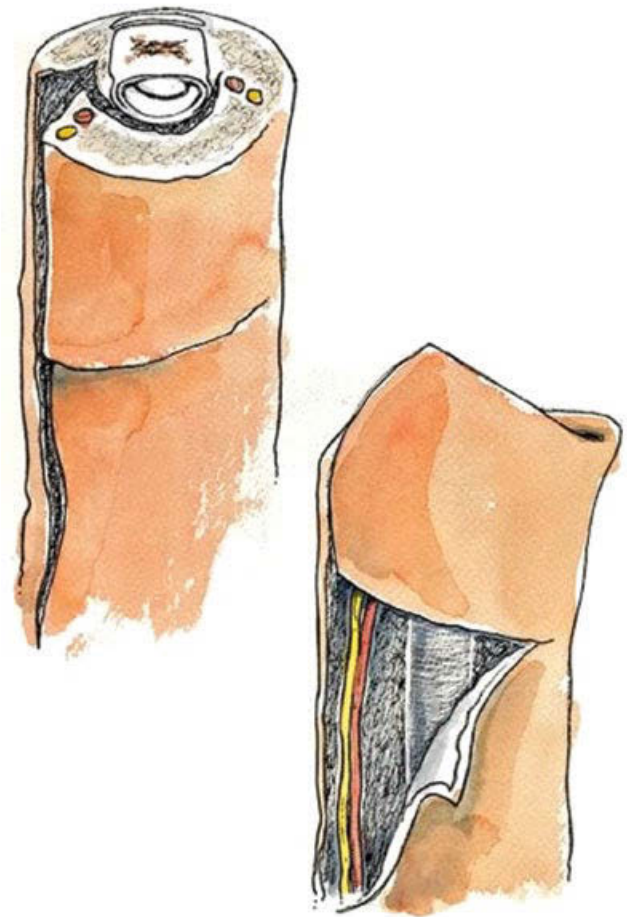


Figure 50.14 Sammut modification of Souquet flap. The Hueston flap has a wide arc of rotation, but the leading edge is insensate. The Souquet flap moves far less but is entirely sensate. The Sammut achieves the best qualities of both flaps by including the leading pedicle (retaining sensibility) and mobilizing it so that it permits flap advancement. Source: Donald Sammut. Reproduced with permission.

on its neurovascular bundle and transferred this to the ulnar aspect of a reconstructed thumb to confer sensation¹³ (Figures 50.15 and 50.16).

These are among the most versatile flaps for reconstruction, since the pedicle permits so much movement. They are technically challenging but provided important points of technique are observed, they have a safe ratio of blood supply to volume of flap. In most cases they are raised on a digital artery which could have supplied the entire digit and, in supplying an island, is now providing generous vascular input.

The homodigital flaps (Segmuller,¹⁰ Venkataswami,¹¹ Joshi,¹² Evans²⁴) vary in their design and orientation – the Segmuller flap is lateral, the Joshi design is predominantly dorsolateral and the Venkataswami modification was designed obliquely lateral (Figure 50.16) Evans designed an ingenious version, the *step advancement island flap*, which utilizes a series of triangular flaps, each of which advances distally into the next defect, such that the most proximal secondary defect is closed directly (Figure 50.17).

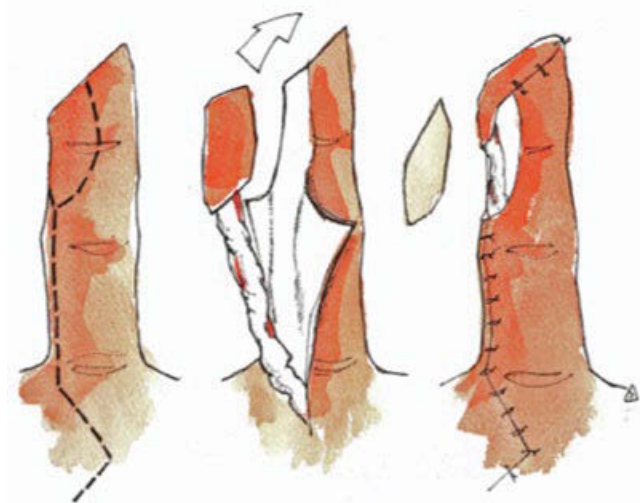


Figure 50.15 Design of homodigital island flap. Source: Donald Sammut. Reproduced with permission.

Many modifications of the island flap have been described, including the *exchange island flap* (Foucher) (Figure 50.2), the technique of dividing the flap nerve and anastomosing it to the nerve in the defect in order to minimize reorientation.²⁵ Glicenstein proposed a retrograde perfusion flap similar in principle to the Chinese radial forearm flap, but based in the digit and rotating on the transverse arcades at the level of the joints.²⁶

The vessel and nerve, with accompanying fat which contains essential venous drainage, are dissected free, as far proximally as is required. Additional advancement can be obtained by supplementary techniques such as ligation and section of the vessel to the adjacent digit at the Y bifurcation and by careful longitudinal separation of the relevant elements of the digital nerve from the common digital nerve. On the radial side of the index the digital bundle is tethered at the base; additional advancement can be obtained by releasing the bundle from this tethering (Figure 50.18).

Important points of technique

- The digital artery is not accompanied by an identifiable digital vein, since most venous drainage in the digit travels via the dorsum. It is important to include the fatty investiture of the neurovascular pedicle since this contains important venules which later dilate and drain the flap (Figure 50.19).
- One must produce adequate advancement and not be tempted to flex the finger to accommodate the flap. Flexion contracture inevitably follows. The finger must leave the operating room with the flap inset and without resort to flexing splintage.
- Any tunnelling must be relaxed, ample and there should be no points of kinking. The process of tunnelling must also ensure that no twisting movement of the pedicle occurs.

Free pulp transfer

First described by Buncke and Rose in 1979,²⁷ free pulp transfer gives arguably the most 'like for like' reconstruction, the subcutaneous fat, fibrous septa and glabrous skin of the toe pulp providing an excellent pad for the finger tip. In the young, where a useful return of sensation can be expected from nerve anastomosis, free transfer provides the opportunity to customize the transferred tissue for reconstruction. Options include the contiguous hemipulps of first and second toes, toenails including germinal matrix, wraparound flaps from first or second toes and partial lesser toes.²⁸

Pulp flaps can consist of simple skin islands or may extend to inclusion of the nail as a wraparound flap. In this case, it is useful to include the distal phalanx of the toe to which the nail matrix adheres, to provide stability and to prevent resorption of the otherwise 'open' end of the recipient skeleton.

Important points of technique

- Most microsurgeons will favour distal-to-proximal dissection in this situation. This ensures that the island of tissue is in continuity with the vessel to be harvested and also minimizes the incursion into the donor toe.
- The design and harvest of the flap and the preparation of the recipient should ensure that the transferred tissue is not excessively bulky to avoid the well-recognized 'cobra' appearance of the recipient digit.

The bank digit

The concept of the bank digit was introduced by Foucher.⁵ In multi-digit trauma a damaged finger might be beyond salvage but may still be capable of providing elements useful in the repair or reconstruction of other digits. These include any element ranging from split skin graft and nail plate, to arterial grafts or a complete island (or free) flap. One should not miss the opportunity to utilize such elements.

Venous flaps

The concept of establishing an arterial inflow to a venous flap ('arterialized' venous flap) was introduced in an experimental model by Nakayama *et al.* in 1981,²⁹ and then used clinically both by Yoshimura *et al.*³⁰ and by Mimoun *et al.*,³¹ the former to reconstruct a traumatic finger defect.

Thatte and Thatte³² subsequently classified venous flaps into three types:

- venous flow through (venous inflow, venous outflow),
- single venous pedicle, and
- arterialized (arterial inflow, venous outflow).

This classification is generally accepted, although it is only the arterialized venous flap that has a role in finger tip reconstruction.

Advantages

Ease of harvest and a thin versatile flap with minimal donor site morbidity are the chief advantages of the free venous flap. Several veins are usually visible through the thin skin of the volar wrist/forearm, which allows a choice of pedicle whose length can be

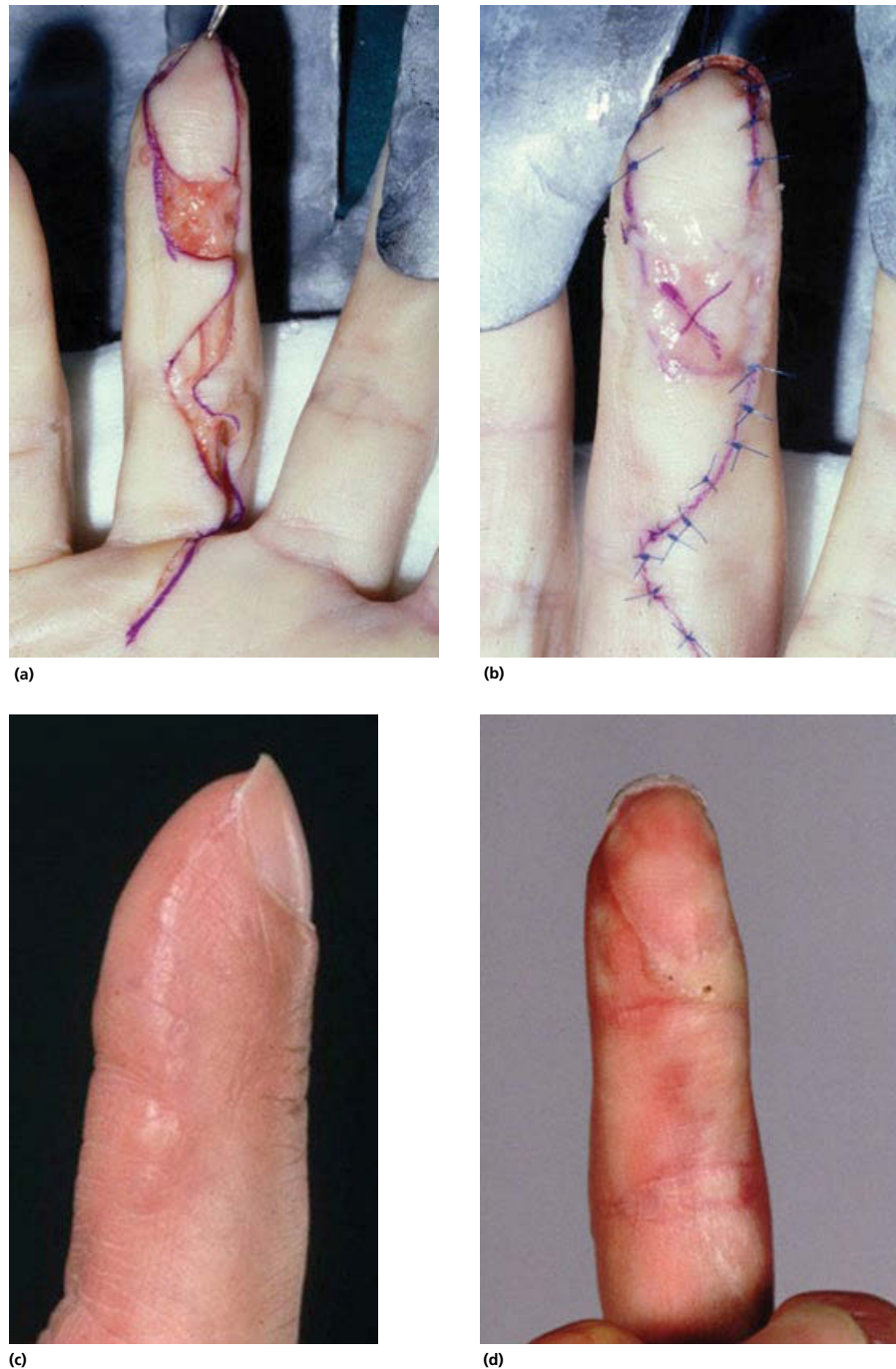


Figure 50.16 The homodigital island flap. The island is designed over the neurovascular bundle which is dissected to the base and advanced. The secondary defect receives a split skin graft. Source: Donald Sammut. Reproduced with permission.

tailored and the flap orientated to suit the finger tip defect. Small cutaneous nerves invariably run with the veins and these can be anastomosed to terminal branches of the digital nerves.

Disadvantages

The veins supplying these flaps are <1 mm diameter and the anastomoses are technically challenging. Venous flaps are by reputation difficult to monitor, as their initial blueish/purple hue and gradual darkening is easily mistaken for vascular congestion.

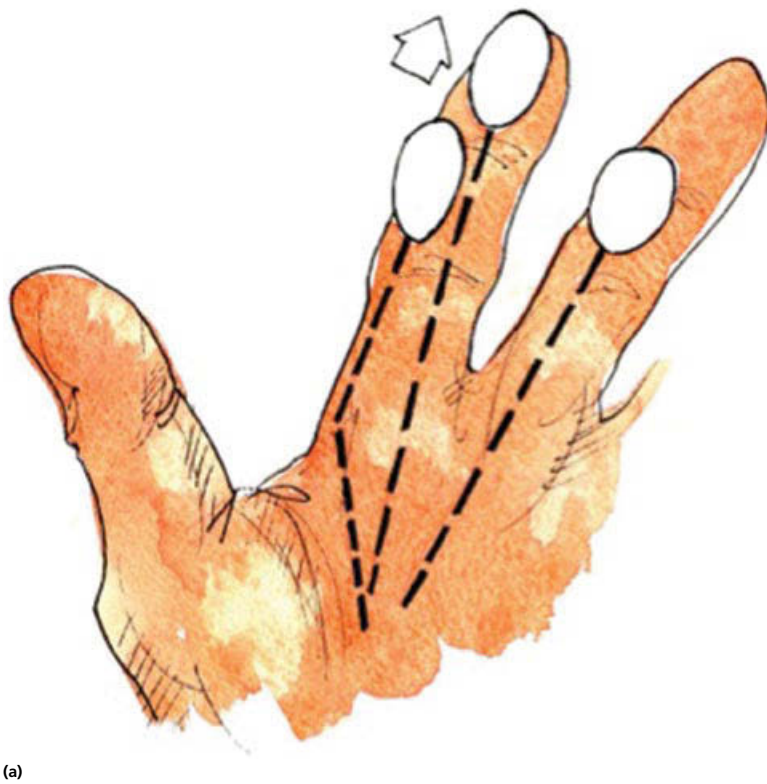
Capillary refill is always more brisk than the norm for flaps but is a reliable indicator.

Points of technique

The afferent and efferent veins of the flap should not be in direct continuity, since blood flow may choose this low resistance route and shunt from one to the other without perfusing the capillary bed. Longer pedicle length will aid the microsurgery. Some operators prefer shorter pedicles to try and limit the number of valves



Figure 50.17 The Evans step advancement island flap. (a) An island is designed with triangulated edges and the flap is advanced such that the leading portion surfaces the defect and successive triangles advance by one place. (b, c) Design of the flap. Note that the dorsal border is a straight midlateral line and that the triangles diminish in size from distal to proximal. (d) The flap is completely islanded. (e) The flap is advanced so that each triangle takes the place of the defect immediately distal. The final, proximal, defect is closed. (f, g) Result. Source: David Evans. Reproduced with permission.



(a)



(b)

Figure 50.18 Release of base of radial pedicle in the index. The radial neurovascular bundle in the index executes a sharp angle at the base. Useful extra advancement can be gained if these tethering bands are released, and the neurovascular bundle straightens out. Source: Donald Sammut. Reproduced with permission.



Figure 50.19 Fatty pedicle. The neurovascular bundle contains no formal vein since venous drainage is mainly on the dorsum of the digit. It is important to include generous fat with the pedicle since this contains important venules and drainage. Source: Donald Sammut. Reproduced with permission.

within the afferent veins.³³ Either way, the venous flaps should be reversed so any valves will not impede blood flow.

The nail

The nail deserves special mention since it is often neglected in crush and amputation injuries. Deformities of nail shape or growth can compromise otherwise very successful and inge-

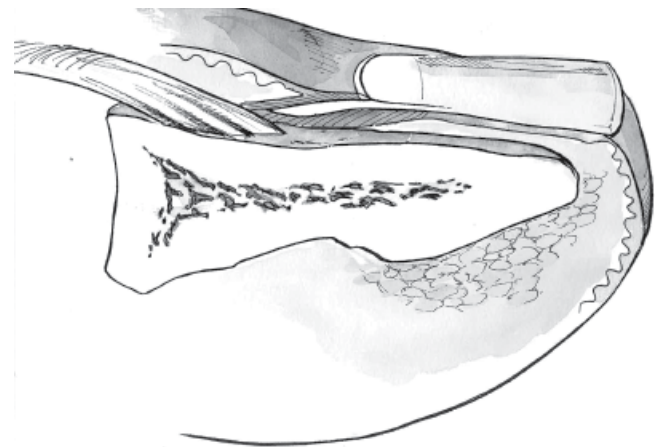


Figure 50.20 Cross section of nail complex. The germinal matrix, the nail fold, the sterile matrix and the support provided by the phalanx are all essential elements in producing the nail complex shape and structure. Source: Donald Sammut. Reproduced with permission.

nious soft tissue reconstruction. Many such unfavourable outcomes can be avoided if basic features of nail support and growth are borne in mind (Figure 50.20).

- The germinal matrix must be unscarred. If it is involved in laceration, it should be repaired as accurately as possible, preferably with microscope assistance.
- The nail fold must be kept open – the nail plate is only plate shaped because the nail fold permits it out of the germinal

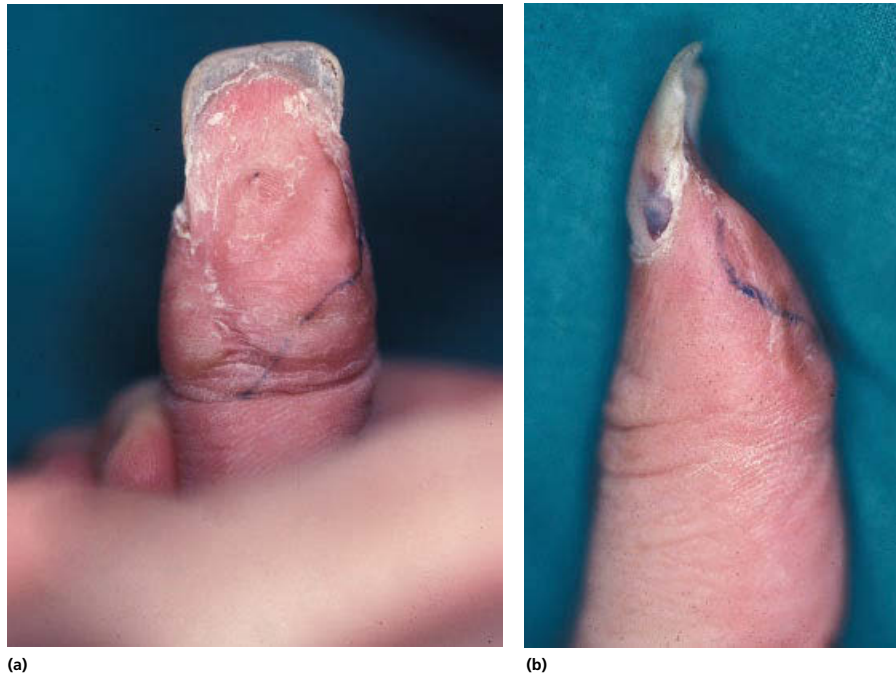


Figure 50.21 Nail support. The presence of the phalanx provides full support for the nail despite loss of all soft tissue. Source: Donald Sammut. Reproduced with permission.



Figure 50.22 Hang nail. The loss of distal phalanx length removes support for the nail. Source: Donald Sammut. Reproduced with permission.

matrix in this shape, much as the outlet of a toothpaste tube dictates the round shape of the extrusion. If the nail plate is removed, detached or lost, it should be replaced and kept in the fold while a new nail plate is advancing.

- The sterile matrix has special qualities – it allows the nail to adhere, while permitting slow progression of the plate as it grows. It is also smooth and flat, as is the plate. Any laceration of the sterile matrix must be repaired as accurately as possible, preferably with microscope assistance, in order to reduce scarring and to preserve these qualities.
- The distal phalanx is essential for support, whereas the pulp provides no structural support. A digit without pulp will support the nail (Figure 50.21) whereas one with soft tissue

but no bony support, will allow the nail to curve over the end (Figure 50.22). Even crushed elements of distal phalanx should be preserved if possible and allowed to unite – it is often surprising how well these remould or require just minor secondary surgery while the support is maintained.

- All surgery to an injured nail structure is best performed in the acute situation while these structures are relatively supple and, indeed, have some distinctive features which aid identification. Elective secondary surgery on germinal matrix or sterile matrix is especially difficult if it has been allowed to scar.

Psychological aspects of hand injuries

All injuries and surgery to the hand carry a psychological dimension. Leaving aside the special situation of the professional sportsman, the musician, the creative artist or the child, most will require their hands to earn their living and for the performance of the most elemental functions. It is part of the initial assessment of any hand injury quickly to sense these dimensions and to incorporate them into the analysis and plans.

The manual worker who suffers a major hand injury in a repetitive, uninspiring and low paid job also merits particular attention to this aspect. The initial attention by many professionals and the sudden thrust into the centre of attention is often unfamiliar and curiously welcome. Surgery and assiduous care follow, then discharge and increasingly spaced visits, until the patient comes out at the other end of the process, back in a humdrum, low-paid job (or unemployed), now with even worse

employment prospects and skills. The attention disappears and the specialized team moves on, while life is now even less inspiring and lower earning.

Conclusions

- Assessment of the defect, overall injury, psychological aspects is crucial as is the formulation of a plan based on restoration of function, not mere anatomy.
- The managing surgeon should master as many techniques as possible even those which seem unspecialized or unappealing. The larger the toolbox available the more likely is there to be found just the right tool for the task.
- The patient must be as informed as possible of the issues at stake, the plan of action, the realistic outcome and the possible complications. The need for secondary procedures should be set at the outset so that it becomes part of an anticipated plan not a remedy for initial failure.
- If tissue is to be discarded, one should always be aware of the possibility of harvest of skin, nail, bone, etc. for use in reconstruction in other digits.
- Points of technique for the various methods must be respected since there is usually little room for error or revision. It need hardly be added that a method should be used only if technical competence has been achieved and not merely because it is an enticing and attractive technique.

In the field of skin cover of the finger tips, restoration of function is the goal. Despite ingenious and constantly evolving methods of tissue movement and reconstruction, this field is dogged by perennial limitations, including: the relatively limited outcome of nerve repair; the incidence of neuroma and cold intolerance; the problem of scar contracture. Advances in the understanding of nerve regeneration and methods of influencing nerve recovery may one day transform our approach to these common and debilitating injuries.

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CHAPTER 51

Flexor tendon injuries

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Anatomy

An in-depth knowledge of the anatomy and biomechanics of the flexor tendon system is essential to every stage of the management of flexor tendon injuries from diagnosis, surgery, correct assessment of complications through to secondary surgery. It must be remembered that this is a carefully balanced system with the function of any particular muscle not depending only on its origin and insertion, but the complex interplay between the system of pulleys in the hand and the balancing forces of all the muscles acting across the intervening joints. For example the effect of flexor digitorum profundus (FDP) acting alone would be to act purely at the distal interphalangeal (DIP) joint until this was fully flexed before acting at the proximal interphalangeal (PIP) joint in a similar manner, followed by the metacarpophalangeal (MCP) joint and lastly the wrist. However in normal function, such as grasping an object, the action of FDP will always be accompanied by balancing tensions in other muscles, and moderated by the pulley system to produce the desired functional outcome. For example, in grasping an object the wrist is stabilized and the joints of the finger are simultaneously flexed around the object.

Figure 51.1 shows the structure of flexor digitorum superficialis (FDS) as it divides and rejoins to become deep to FDP just before its insertion. This illustrates the complexity of just one element of the interplay between anatomy and function.

The wrist flexors and FDS are part of the superficial flexor muscle mass in the forearm arising from the common flexor origin. Both of the wrist flexors have pulley systems close to their insertions that redirect the vector of force and thereby increase the efficiency of their action. The flexor carpi radialis (FCR) is redirected by the scaphoid before being inserted into the bases of the second and third metacarpals and the flexor carpi ulnaris (FCU) is redirected by the pisiform before its insertion into the base of the fifth metacarpal. The FDS muscle also has an origin from the radius, and provides

independent control of pull for the FDS tendon to each digit, with the exception that the action on the little and ring is sometimes conjoined (see under Clinical examination below). The function of FDS is partly to assist with power grip, but more importantly provides individual finger flexion as required in manipulation of objects in the hand and in fine motor skills. As the FDS is inserted into the base of the middle phalanx, it acts mainly at the PIP joint, but also crosses the wrist and MCP joint, where the magnitude of its effect is determined by the balance of tension in the other muscles acting at these sites, such as the wrist flexors and extensors, the long finger extensors, and, importantly, the interossei and lumbricals.

The deep flexor muscle mass consists of flexor pollicis longus (FPL) arising from the palmar surface of the body of the radius, and the FDP arising from the ulna. FPL is the only flexor of the thumb and provides the strongest pull of all the flexors to the hand,¹ and therefore repair of the tendon to FPL must always be very resilient. FDP is a mass action muscle that provides a flexion force to all the digits through its insertion into the distal phalanx of each finger and its main function is in power grip. There is sometimes an individual muscle belly providing independent action of FDP to the index, but this is rarely the case for the other digits as the muscle belly is a single mass for them and the tendons are interconnected in the distal forearm and wrist.² In some patients there is a tendinous slip from FDP to the index to FPL.³

Pulley system

The pulleys acting on the wrist flexors have already been described above. The FDS and FDP enter a system of pulleys at the level of the MCP joint. These are thickenings in the flexor sheath that are recognizable and follow a set pattern of five annular pulleys and three cruciate pulleys⁴ (Figures 51.2, 51.3 and 51.4).

The most significant of them are the second and fourth annular pulleys known as A2 and A4. A2 is 20 mm long, arises from the proximal phalanx and contains both FDP and FDS. A4 is 12 mm long, arises from the middle phalanx and contains FDP alone distal to the FDS insertion.

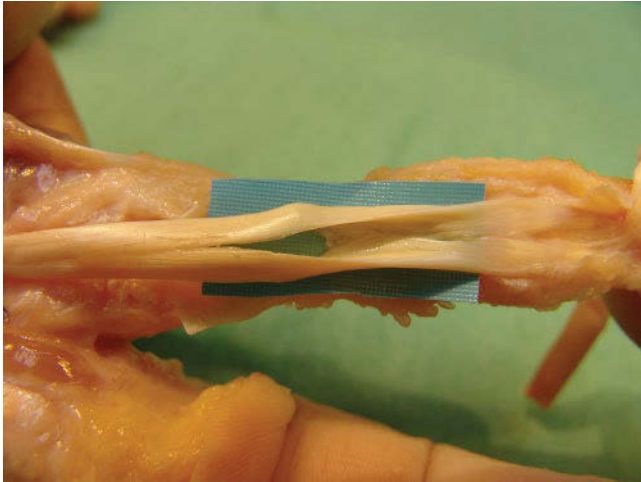


Figure 51.1 Flexor digitorum superficialis (FDS) chiasma. In this cadaver specimen the flexor digitorum profundus (FDP) tendon has been removed and the complex structure of the FDS chiasma can be clearly seen. FDS tendon divides into two slips that spiral around the FDP tendon to meet each other again deep to FDP. Here the central half of each slip decussates to form a chiasma with the central half of the opposite slip, while the outer half of each slip continues to form the outer edge. Distal to the chiasma two slips are again formed, each of which contains the outer half of the original slip plus the central half of the contralateral slip. The FDS tendon has thereby formed a cradle around the FDP which will tighten on FDP when FDS is activated and may therefore play a part in the pulley system for FDP and the rebalance of the pull of FDP on the joints that it crosses on the way to its insertion. Source: Reproduced with permission of Donald Sammut.

Zones

The regions that the tendons pass through are divided into zones from distal to proximal. Zone 1 is beyond the insertion of FDS where only one tendon is present – FDP. Zone 2 is the rest of the flexor sheath. Zone 3 is from the mouth of A1 to the distal limit of the transverse carpal ligament. Zone 4 is the area deep to the transverse carpal ligament (i.e. the carpal tunnel). Zone 5 is from the proximal edge of the transverse carpal ligament to the musculotendinous junction.

Blood supply

The blood supply to the tendon varies along its length. At the musculotendinous origin a blood supply will be originating from the muscle itself, then as the tendon is further away from this the blood supply from the epitenon will become more important with some contribution to nutrition also coming from the synovium bathing the tendons in the various spaces of the hand, such as the carpal tunnel. For the finger flexors, within the flexor sheaths the blood supply is derived from the vincula (Figure 51.5) that enter the dorsal surface of the tendon, as well as nutrition arising from the synovial fluid produced by the inner surface lining of the flexor sheath.

Tendon healing

As with the healing of any tissue, the process of tendon healing consists of three overlapping phases starting with *inflammation*, followed by *proliferation* of cells and progressing on to *remodelling*.



(a)



(b)

Figure 51.2 (a) The pulleys exposed on a cadaver dissection. The filmy appearance of these structures belies the strength with which they are able to resist bowstringing of the tendon. The distinct distal edge of A2 is seen clearly, followed by C1, A3 and C2 overlying the proximal interphalangeal joint. (b) In this cadaver specimen of an index finger the filmy elements have been removed leaving the more clearly defined A1, A2 and A4 annular pulleys. Source: Reproduced with permission of Donald Sammut.

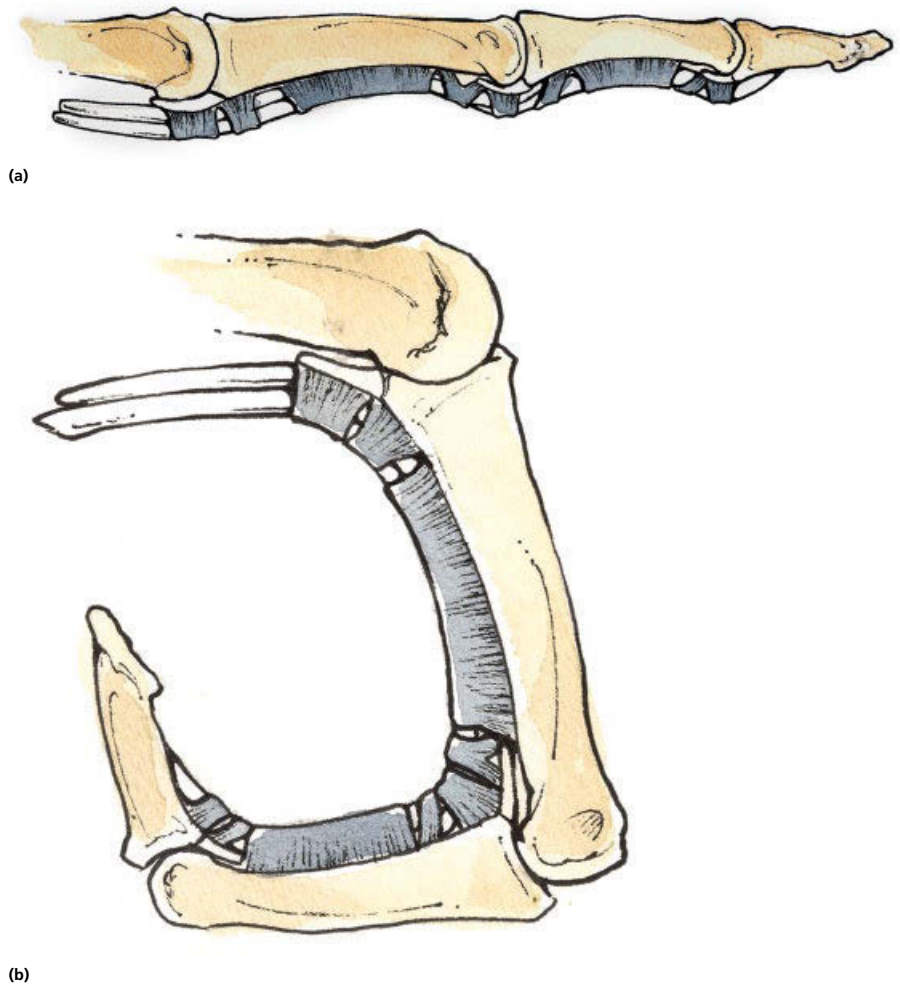


Figure 51.3 (a) This drawing shows the anatomical arrangement of the pulleys – from proximal to distal: A1, A2, C1, A3, C2, A4, C3 and A5. (b) On full flexion, when the force against the pulleys is at its strongest, the effect of the anatomical arrangement is such that the thickened parts of the flexor sheath lie in continuity without any bulkiness between them to impede flexion. Source: Reproduced with permission of Donald Sammut.



(a)



(b)

Figure 51.4 (a) This cadaver specimen demonstrates that when tension is applied to the flexor tendons they are held close to the bony skeleton by the intact pulley system. (b) The same specimen has had the pulleys excised. Now when the tendon is pulled in the same way the tendons bowstring away from the metacarpophalangeal and proximal interphalangeal joints, reducing the mechanical advantage obtained from the same excursion. Source: Reproduced with permission of Donald Sammut.

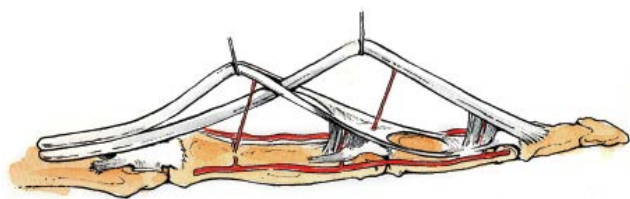


Figure 51.5 Illustration of the vincula to flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP). Each tendon has a long vinculum proximally, where they need to accommodate more excursion, and a short vinculum more distally. Both vincula of the FDS arise along the length of the proximal phalanx. The long vinculum of the FDP also arises from the proximal phalanx, close to the short vinculum of the FDS, whereas the short vinculum of the FDP arises at the middle phalanx. Source: Reproduced with permission of Donald Sammut.

Traditionally, the modes of healing are divided into extrinsic and intrinsic. In the extrinsic pathway the tendon becomes adherent to the flexor sheath during the inflammatory phase. This occurs because the epitenon is in effect an epithelium and once breached allows the egress of tenocytes into the surrounding tissue with subsequent adhesion formation. In reality, the distinction is not so clear cut and cells have been shown to repopulate the tendon from within the tendon, the circulation and the bone marrow, whether adhesions form or not. What we do know is the tendon will heal even when the factors that are dependent on adhesion formation are prevented by early motion and in fact early loading increases the cellularity of the tendon with increased strength and a more orderly matrix being formed.⁵

Patterns of injury

The commonest cause of injury to a flexor tendon is a sharp laceration, for example from a knife or broken glass. A multitude of scenarios present themselves in this manner, but it is worth noting that a knife used in an assault may cause injury to either the victim if they grab the knife in defence or the assailant as they stab something that is more resistant than they expected such that the blade of the knife cuts the hand that was gripping it.

When describing the zone for an injury or repair it is usually referred to by the zone that it would lie in with the hand in the resting posture, as this is where the repair will lie for the majority of the time. However, repairs that are near the end of a zone will clearly move through more than one zone on activity.

Blunt injuries rarely divide the tendon, but they can cause significant crushing to the surrounding tissue and abrasions to the tendon that will cause adhesions if not managed well.

The FDP tendon is also prone to avulsing from its insertion into the distal phalanx. This is commonly known as ‘jersey injury’. The avulsion may also involve a fragment of bone, and this, along with whether the ongoing force of pull also

Table 51.1 Modified Leddy and Packer classification of flexor digitorum profundus (FDP) avulsion⁶

Type I	FDP and both vinculae rupture with no fractures Tendon retracts into palm, presenting as a tender lump <i>Early operative repair necessary</i>
Type II	FDP ruptures but long vincula remains intact Avulsed FDP held at proximal interphalangeal joint There may be a small avulsed bony fragment <i>Repair required within 3 months</i>
Type III	FDP avulsion with a large bony fragment that gets caught at A4 Both vinculae are preserved <i>No time limit for repair</i>
Type IV ^a	FDP and bony fragment avulsion, with tendon avulsion from the bony fragment
Type Va ^b	FDP and bony fragment avulsion, in association with fracture of the distal phalanx (extra-articular)
Type Vb ^c	FDP and bony fragment avulsion, in association with fracture of the distal phalanx (intra-articular)

^aSmith’s modification.⁷

^bAl-Qattan modification.⁸

ruptured the vinculae, will determine how far proximally the avulsed end of tendon retracts. This has been classified into type I–III injuries by Leddy and Packer.⁶ Type IV was added by Smith⁷ and types Va and Vb described by Al-Qattan⁸ (Table 51.1).

History and examination

Eliciting a careful history will raise the suspicion of a tendon injury. The commonest injury that is missed at this stage is the FDP avulsion, as the assessment often takes place in a setting where there is no hand surgery expertise. Here, in the absence of a laceration and significant swelling it is assumed that the tendon is unlikely to be injured and the lack of movement is due to pain. Examination is completed with assessment of the vascular supply and nerve function.

On examining a patient with a laceration, the integrity of every tendon that could have been affected must be assessed. The wrist flexors are tested by getting the patient to flex the wrist against resistance while simultaneously palpating the muscle bellies and tendons to ascertain whether there is continuity. FPL function is assessed using the ‘O’ sign where the patient is asked to make an O shape between their thumb and index finger. An O shape is only possible with active FPL function at the interphalangeal (IP) joint, and resistance is tested by asking the patient to try to prevent the examiner from breaking through the ring of the O. This test is more reliable than asking the patient to flex the IP joint as there are trick movements that can cause a flicker of movement at the IP joint, causing diagnostic confusion.

In the digits the resting cascade will often provide some information even before the patient is asked to do any

movements and then a composite fist formation will add to this before individual tendons to individual digits are assessed, for example the DIP joint of a digit with a divided FDP will fail to curl into the fist. FDP function is assessed by its action on the DIP joint of the affected digit.

In order to assess FDS function the FDP has to be blocked from acting on the PIP joint. This is done by holding the other digits out in full extension, releasing only the affected digit and asking the patient to bend it down. However, if there is no active flexion at the PIP joint of the little finger in this examination it is necessary to release the ring finger as well and retest for FDS function. This may then allow flexion of the little finger PIP joint, demonstrating that the little finger FDS is not independent of the ring finger FDS. This is usually due to an anomalous muscle or tendon slip from the FDS of the ring finger to the FDS of the little finger.⁹ FDS may also be completely absent in the little finger in up to 13.7% of the population, 64% of whom have this variation bilaterally.^{10,11} Therefore the presence or absence of FDS to the little finger on the other side adds little to the assessment.

In all of these examinations, significant pain on flexion in the presence of tendon continuity may represent a partial division of the tendon. In any case, where there is a deep laceration over a tendon the patient will require formal exploration in theatre under tourniquet.

Surgical management

The historical perspective and evolution of current concepts are outlined in Box 51.1.

Box 51.1 Historical perspective and evolution of current concepts: surgical techniques

At the turn of the century a laceration to the finger that had divided the flexor tendon(s) in zone 2 was usually allowed to heal and then reconstructed with a secondary tendon graft because the results of primary repair were considered to be too poor.¹² It was not until half a century later that publications began to emerge describing successful tendon repairs in zone 2.¹³ In the years following this there have been many new suture techniques tried with varying success.^{14–16} Despite the advances over the last century there is still no method of tendon repair that is strong enough to withstand the force across the repair site in normal use. Attention is now directed at a method of repair that will maximize the strength of the repair but also the stiffness of the repaired section, its tendency to gap and the force of work required to move the tendon, all of which will affect the glide of the tendon through the pulley system. Many surgeons now perform repairs with a four-strand core suture technique with a locking configuration which, combined with a strong peripheral suture can adequately bear the stresses of early active motion.^{17–19}

Exploration and tendon retrieval

Approaches to the tendon need to respect the usual principles for incisions in the hand (i.e. not crossing joint creases longitudinally). There are points of technique specific to each of the zones as described below.

Zone 5

In the forearm with multiple tendon injuries the function of each distal end can be assessed by simply pulling on it, but it can be difficult to match it to the correct proximal end. This is assisted by:

- working methodically (e.g. from the radial side to the ulnar side);
- having an in-depth knowledge of the anatomy at the various levels (e.g. the fact that the tendons of FDS to the middle and ring fingers lie superficial to those to the index and little fingers);
- a close inspection of the ends of the tendons to show their comparative cross-sectional shape and size as well as the configuration of the laceration.

To prevent errors it is wise to have completed the exploration and identified all the tendon ends on both sides of the wound and matching them before proceeding to repair any of them, working from deep to superficial.

Zone 4

This is unusual due to the depth at which the tendons lie in zone 4, but when there is a laceration to the distal forearm that occurred when the wrist and fingers were flexed the distal end will come to lie in the carpal tunnel. Although it can often be delivered into the forearm by reproducing this posture the repair site will then also be within the carpal tunnel. In this case, particularly if more than one tendon is affected, it is prudent to open the carpal tunnel to prevent an acute carpal tunnel syndrome, so this should be done as part of the exploration stage as it makes the repair much easier to do. In this situation the carpal tunnel should be opened with a step configuration so that if there is concern about bowstringing of the repaired tendons at the end it can be repaired with step lengthening.

Zone 3

When exploring lacerations over FPL and all lacerations in the palm of the hand, the proximal ends may have retracted back proximal into the carpal tunnel. In this case a separate incision is made in the distal forearm to retrieve the tendon, which is then fed through the carpal tunnel. This still requires a separate incision to allow direct vision of the grasping end as blind grasping for the tendon in the carpal tunnel carries a risk of damage to the median nerve. One method of retrieval is to feed an umbilical catheter or infant feeding tube retrograde through the tunnel then either suture it side to side to the tendon and pull both through together, or put the first part of the core suture into the tendon and feed this through the tube (Figure 51.6a–k). In this second technique the tube is used to deliver the suture only, then

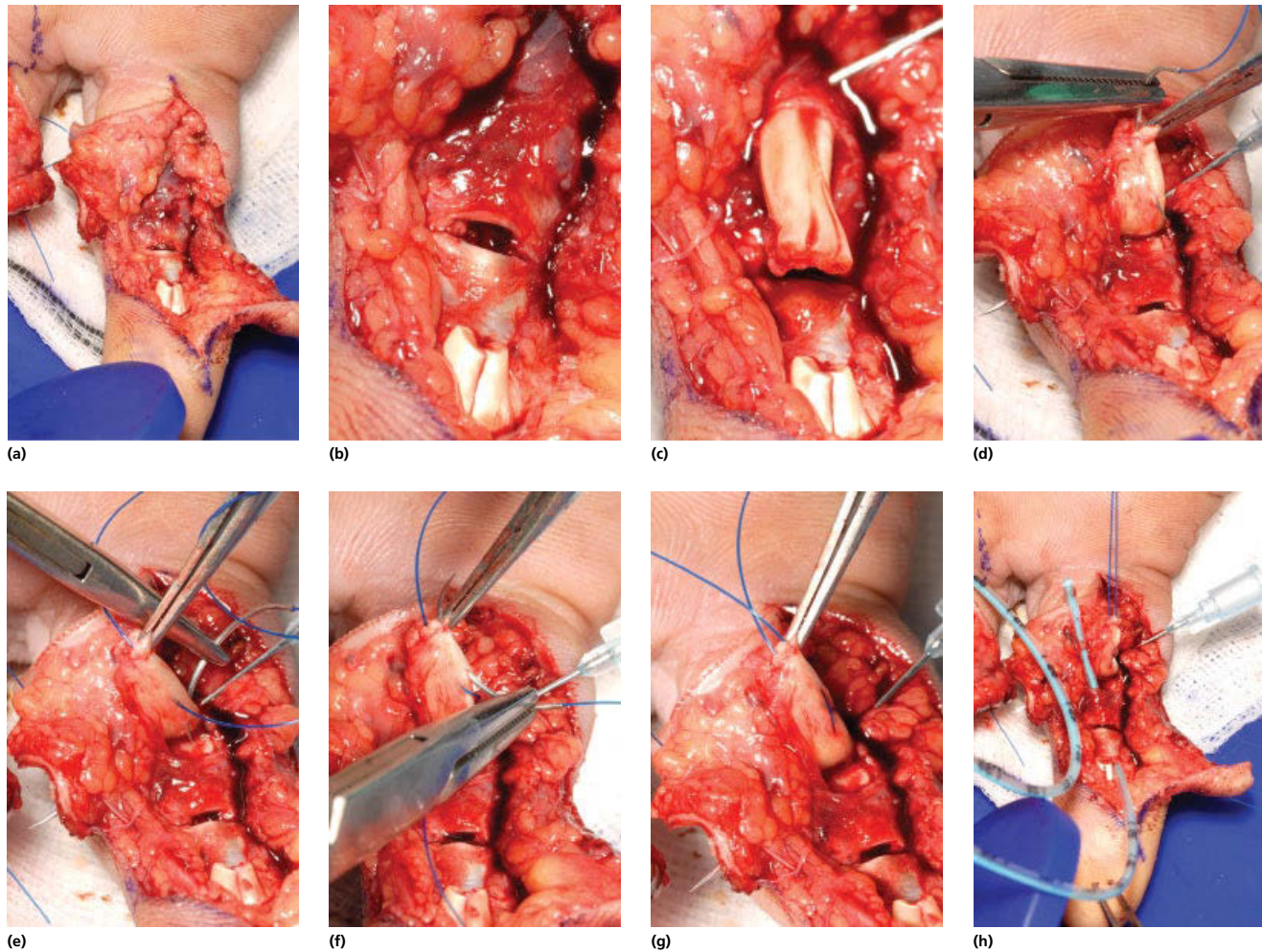


Figure 51.6 This operative series shows the retrieval and repair of a flexor digitorum profundus (FDP) tendon using a four-strand Adelaide core suture and a Silfverskiöld epitendinous suture. (a) Overview after exploration of a laceration to the little finger – a clean transverse cut at the proximal end of the A4 pulley is seen. (b) Close up view shows that the distal end of FDP has been flipped out of the flexor sheath through a transverse incision at the distal end of A4 to allow exposure of enough tendon for a good repair. The proximal sheath is seen to be full of blood. (c) Milking the finger has brought the proximal end of FDP out through another transverse incision in the region of the C1 pulley. It has been prevented from retracting with a hypodermic needle. (d) The proximal end of the tendon is picked up and reflected back to allow easy access for the first bite of the core suture. The vincula is clearly seen on the dorsum of the tendon and sutures are not placed within this vascular complex. (e) The transverse bite of the cross locked suture is placed across the side of the tendon. (f) The subsequent longitudinal bite is placed. The forceps holding the tendon have not been moved throughout these manoeuvres, ensuring that minimal handling of the epitendon has occurred. (g) The suture cross thus formed can be seen on the side of the tendon. (h) An infant feeding tube is fed retrograde through all the pulleys that the proximal end needs to be delivered through.

tension on the suture is applied while easing the tendon into the tunnel opening until it delivers through and out the other end.

Zone 2

In zone 2 repairs it may also be necessary to retrieve the tendon from the palm up through the tendon sheath as described above. The distal end can usually be delivered into the wound by flexing all the DIP joints and the PIP joints as necessary; most lacerations occur with the finger flexed. It may be necessary to open some of the pulleys to gain enough access to retrieve the ends and carry out the repair. Although the pulley system should be respected and not incised without consideration, it is more important to have a repair that glides at the end of the operation

than to have intact pulleys, even at A2 and A4. If they have been opened it is not necessary to attempt to repair them at the end of the operation and doing so may actually increase the risk of adhesions or rupture due to the increased work required to move the tendon repair site through the constriction caused by a pulley repair.^{20–22} In most cases where the pulleys are left open no functional deficit is found, and in the minority who do find that the tendon bowstrings and reduces the power of flexion a secondary reconstruction of the pulleys is likely to be more effective.

It is not always necessary, or advisable, to repair the FDS as well as the FDP in zone 2²³ and repairing one slip and excising the other may provide the best compromise between ensuring

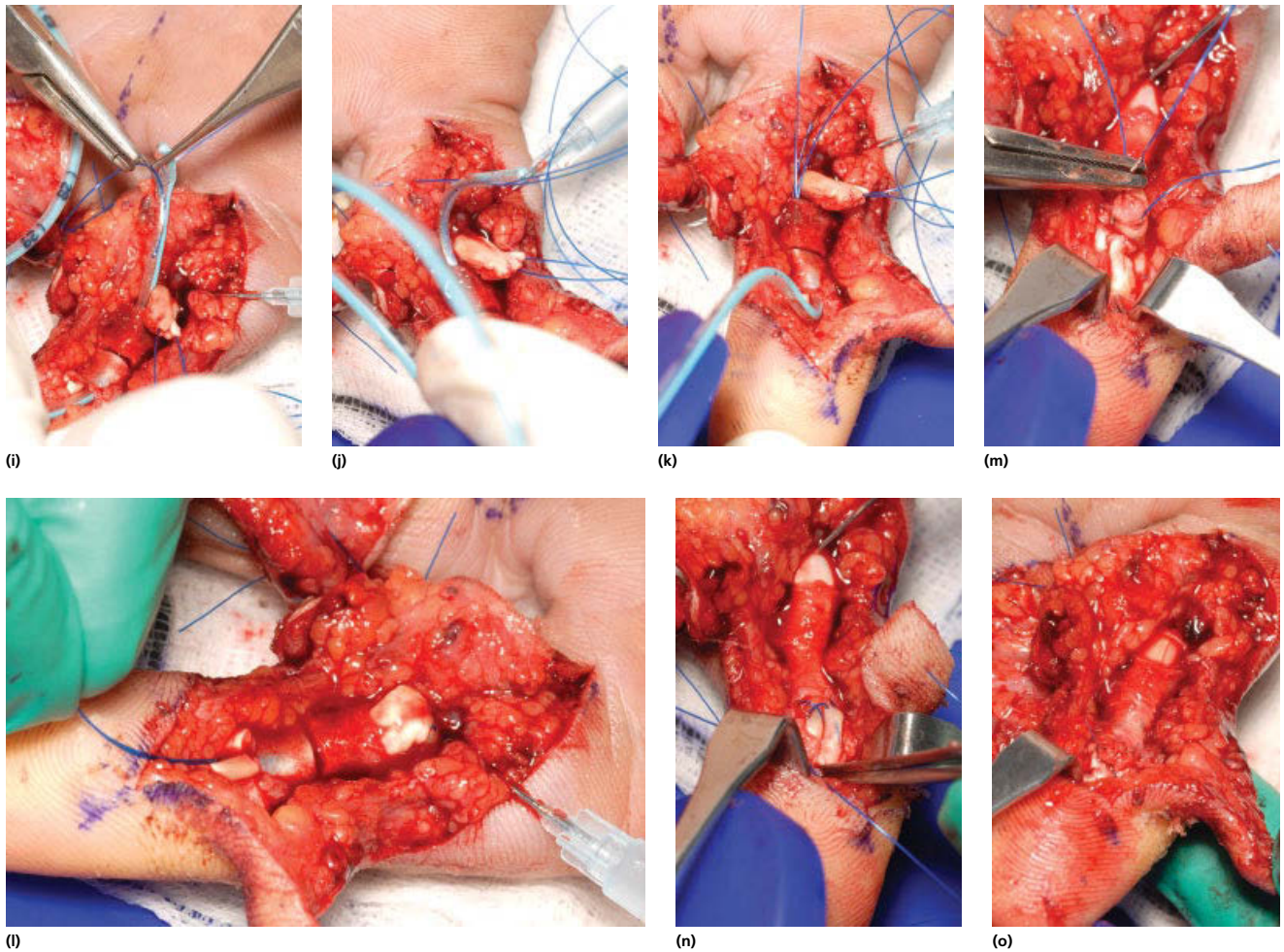


Figure 51.6 (continued) (i) The suture needle is fed into a side hole on the feeding tube, blunt end first. (j) The sharp end of the needle is then tucked into the blind end of the feeding tube, and the other end of the suture is also fed into the feeding tube. (k) The feeding tube is now withdrawn through the flexor sheath so that the suture and needle are delivered through without the sharp end of the needle coming into contact with the lining of the flexor sheath. (l) Tension can then be applied to the suture while feeding the tendon end into the opening in the pulleys. In this way the tendon is delivered through; the hypodermic needle is withdrawn as the tendon is pulled through and replaced to secure it in its new position. (m) The placement of the core suture then continues with a cross lock on the distal end, followed by a further cross lock on each end to give a total of four cross locks and four strands as described.¹⁹ (n) Here the third cross lock has just been placed (i.e. the second one to the proximal end), and the proximal end is held under tension to show the suture configuration. (o) The core suture has been tied and a Silfverskiöld epitendinous repair added. The hypodermic needle has then been removed and the tendon has slid back in so that the repair now sits deep to A4.

enough space for glide while preserving independent PIP joint flexion, particularly in the little finger.

Zone 1

If there is more than 5 mm of distal stump left on the FDP the tendon can be repaired as normal with equivalent strength when compared to reattachment.²⁴ However, if it has been avulsed, or if there is less than 5 mm, it will need to be reattached to the distal phalanx. If there is a sizeable bony fragment associated with an avulsion this can be fixed with a lag screw.

Methods for attachment where there is not a significant bony element include bone fixation devices commonly referred to as bone anchors, or drilling through the distal phalanx, either

through to the dorsum or transversely. In all cases any distal stump remnant should be elevated to allow the new insertion to tuck underneath, and the bone should be decorticated to encourage adherence of the tendon.

When bone anchors are used there are several important points of technique:

- There is no strong evidence that the angle of insertion of the bone anchor significantly affects breaking strength on cyclical loading²⁵ but care must be taken not to place a bone anchor so that it impinges on the germinal matrix of the thumb or the nail bed as this will cause ridging of the nail. For this reason it is preferable to use a screw-type bone anchor rather than a traditional anchor type, as removal of the latter once it has been detonated is extremely difficult. A check X-ray should

Box 51.2 Reattachment of flexor digitorum profundus (FDP) to the distal phalanx: author's preferred method

- 1 Transverse hole with Keith needle or hypodermic needle.
- 2 Two sutures of braided nylon in cross locked formation as per Adelaide repair.

be taken to ensure the bone anchor is in an acceptable position before utilizing the sutures.

- The distal phalanx can be drilled through with a green needle either volar to dorsal or transversely and the suture threaded down the needle to deliver it through the hole before pulling the needle on through and out the other side. When the distal phalanx is drilled through to allow the sutures to be passed from volar to dorsal, the suture may be tied directly over the nail, or a buttress can be used. The traditional buttress was a button, however there have been cases reported of the button causing necrosis of the nail bed,²⁶ particularly if it has a prominence on it at the circumference. A better buttress is the bung from a 2 mL or 5 mL syringe. In this case the suture used for the core stitch will be removed by cutting it and pulling it through from the nail at least six weeks after the repair. The suture therefore needs to be of a type and configuration that will allow it to come out easily, such as a 3.0 Prolene in a Kessler configuration.
- A Keith needle is specifically designed for the purpose of making a hole in a bone and passing suture through the hole. It is similar to a K-wire, but has a needle eye in it at one end without the diameter of the needle getting any wider. It can therefore be drilled through the bone with a K-wire driver with the suture threaded through it so that it is taken through the bone.

In the methods where the suture is passed through the bone, a less brittle suture such as braided nylon should be used to prevent the suture failing where it turns an acute corner on emerging from the bone. The author's preferred method for reattachment of FDP to the distal phalanx is described in Box 51.2.

Repairing the tendon

Where there is a partial division of the tendon this does not require repair if there is 50% or more of the tendon still intact. Any edge that may catch on the pulley should be excised while also trying to minimize the extent of exposed cut surface of tendon that is left.²⁷

There are many techniques available for repairing the tendon and these have been eloquently described elsewhere.²⁸ It is important that the surgeon masters one of them reliably before carrying out this surgery unsupervised. Four-strand techniques are generally recognized to be superior to two-strand techniques. The 'Adelaide repair'¹⁹ has increased in popularity and been shown to be easily mastered.²⁹ The choice of core suture can be made independent of the choice of epitendinous repair. There is little evidence to recommend one suture material over another. Steel and fibrewire have been shown to be stronger than nylon, Prolene and braided polyester, with no significant difference between the latter three.³⁰ In the author's experience the use of 3.0

Box 51.3 Repair of zone 2 flexor tendon injuries: author's preferred method (Figure 51.6a–o)

- 1 Midlateral incision with oblique extension at either end.
- 2 Flap sutured back.
- 3 If proximal end has pulled back and simple milking techniques fail to deliver it into the wound:
 - Incision over proximal end.
 - Insert one limb of an Adelaide repair.
 - Retrieve tendon ends using feeding tube to deliver suture only, then pull tendon through (Figure 51.6a–k).
- 4 Use hypodermic needle to hold tendon.
- 5 Incise pulleys if needed

Adelaide repair using 3.0 Prolene on a round-bodied needle.¹⁹

The benefits of an Adelaide repair over a Kessler-type suture:

- Transverse bites are placed further along tendon than longitudinal lengths of core stitch so less risk of damaging core stitch on passing needle through for transverse bites.
- More tendon length between damaged area and transverse bites is possible when access to tendon is limited.
- Tendon ends dock into place rather than buckling.

Silfverskiöld epitendinous using 6.0 Prolene on a round-bodied needle³¹ (Figure 51.6l–o)

- Check glide using tenodesis effect (Figure 51.7a,b).
- No repair to pulleys.
- Close skin.
- Apply minimal dressings to wound.
- Plaster of Paris splint made to provide a dorsal hood holding the wrist in neutral, the MCP joints at 80° flexion and the IP joints in full extension. If the FPL alone has been repaired, a dorsal hood to the thumb and fingers should be supplied – protecting the thumb alone may result in rupture due to the Linburg–Comstock³ anomaly.³²

Prolene in an Adelaide core suture configuration minimizes the trauma to the tendon and allows the two ends to be brought together in a very controlled 'docking' manoeuvre that minimizes the bunching up of the tendon at the repair site. This allows the repair site to still glide unimpeded through the pulleys.

The author's preferred technique for repair of zone 2 flexor tendon injuries is outlined in Box 51.3.

Occasionally there is segmental loss of the tendon. If this is less than 1 cm the two ends can be directly repaired, but any more than this and a graft is needed. Short segments that do not involve zone 2 can be treated with an interposition graft (Figure 51.8); longer segments and those involving zone 2 will require standard tendon grafting techniques as described below.

Rehabilitation following flexor tendon injury

Unfortunately a repair technique that allows the patient to immediately return to full normal activity has not yet been developed. The rehabilitation of flexor tendon repairs is



(a)



(b)

Figure 51.7 At the end of a repair the finger should be seen to flex with the tenodesis effect without resistance. As seen here the finger flexes fully (a) and extends with the normal cascade (b).

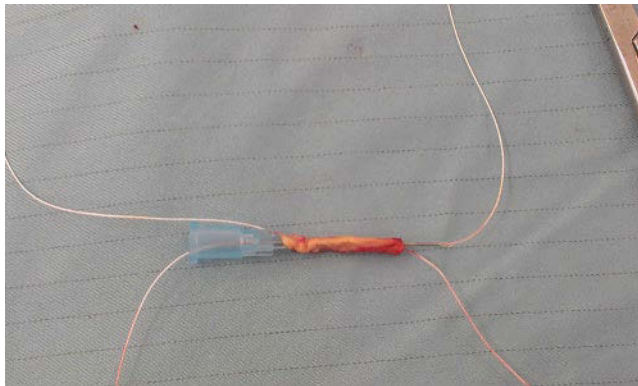


Figure 51.8 When a small section of graft is missing an interposition graft can be used for up to 2 cm outside of zone 2. This is facilitated by threading the length of tendon graft onto a hypodermic needle and then the loose end of a suture can be threaded through the needle before it is removed. If this is done twice, once in each direction with a separate suture each time, it provides a length of tendon with a needle and free end at each end of the graft ready for repair into the tendon defect.

prolonged and complex, and should always be explained to the patient before embarking on surgery. Therefore, although it is essential to have a physiotherapist providing the rehabilitation programme, the surgeon needs to be aware of the principles involved and the general timescale in order to provide the patient with the information they need in consenting to a repair.

There are many different protocols for rehabilitation of the healing flexor tendon. They can be divided into immobilization, early passive mobilization (EPM) and early active mobilization, also known as controlled active mobilization (CAM).

Immobilization

These techniques involve complete immobilization of the hand, wrist and fingers. Typically, the wrist and MCP joints are immobilized in flexion, and the PIP joint and DIP joints in extension; this takes tension off the finger flexors while preventing contractures at the PIP joints. The hand is usually immobilized

for 3–4 weeks before commencing active and passive flexion and extension at home. Often, the period of immobilization is interrupted by visits to hand therapy where protected passive motion may be undertaken to prevent stiffness. This method is seldom used, with the exception of children and patients unable to be compliant with the preferred regimen.

Early mobilization

EPM means passive flexion with either passive or active extension. Early active mobilization (EAM) involves active flexion and extension of the involved digit or digits. Place-hold flexion is another type of active mobilization, involving passive flexion of the finger. The patient then actively maintains that position.

The historical perspective and evolution of current concepts of rehabilitation regimes are outlined in Box 51.4 and the author's preferred rehabilitation regime in Box 51.5.

Box 51.4 Historical perspective and evolution of current concepts: rehabilitation regimes

In 1917, Harmer³³ described flexor tendon repairs coupled with early active mobilization. Just one year later, Bunnell¹² described grafting of zone 2 flexor tendon lacerations, followed by a period of immobilization.

Primary repair of zone II flexor tendons regained popularity in the 1960s.^{13,34} However, immobilization remained in vogue, in the belief that this predisposed to adhesions, which in turn improved tendon nutrition and healing. However, in some units early passive mobilization was being pioneered, and started to show promising results.^{35,36}

It is now known that adhesions are not required to aid healing and actually limit tendon excursion. In fact, the application of early post-repair controlled motion stress increases the rate of revascularization and healing of the laceration, in addition to improving the strength and remodelling of the repair.^{37–39} Two main systems are in use today: *early passive motion* (EPM) using elastic bands to provide a passive flexion force against which active extension is done (e.g. Duran and Houser⁴⁰), and *controlled active motion* (CAM) in which both flexion and extension are carried out actively in a strictly controlled programme.⁴¹

Box 51.5 Modified Belfast Protocol for Early Active Mobilization following flexor tendon repair (zones I–V): author's preferred rehabilitation regime, as used at Oxford University Hospital^a

Days 1–3

- 1 Manufacture a thermoplastic dorsal 'hood' splint with:
 - Wrist 0–20° of flexion
 - MCP joints 60–80° of flexion
 - IP joints full extension.
- 2 Explanation of the early active movement (EAM) protocol:
 - Passive flexion of fingers using contralateral hand to enable nail(s) to touch distal palmar crease (DPC)
 - Repeat exercise but maintain finger flexion using muscle power only
 - Active flexion, nail-to-distal palmar crease then return nails to fully touch the splint
 - Assess active range of movement using goniometry or tip-to-palm measurements

Concerning features include:

- Inability to flex more than two thirds of distance from nails to distal palmar crease
- Inability to fully extend the IP joints
- Full active range of movement at first appointment – therapy may need to be modified to a slower pace to allow adequate tendon scarring and healing to take place.

Week 2 and week 3 appointment

- 1 Check EAM exercises, assessing for differential gliding, quality of movement and tendon integrity.
- 2 Check pain, oedema and range of movement.

Week 4 appointment

- 1 Check EAM exercises, assessing for differential gliding, quality of movement and tendon integrity.
- 2 Progress to full active range of movement (FAROM) exercises. Focus on tenodesis, differential glide: flat fist and hook and full extension.
- 3 Removal of splint for hourly exercises.
- 4 Graded return to activity using chart in booklet.
- 5 Encourage full active extension.
- 6 Maintain passive IP joint range of movement while in a safe position.

The aim is to achieve full active range of movement within 1 week of this appointment (not including resistance/passive extension stretch).

Week 5 appointment (as required)

- 1 Review FAROM exercises and splint.

Week 6 appointment

- 1 Review FAROM exercises.
- 2 Removal of splint for light activities of daily living, continuing to wear it at night and other times of risk.
- 3 Consider night extension splint if there is ongoing limited passive extension.
- 4 Commence light resisted exercises.

Week 8 appointment

- 1 Discard dorsal hood splint.
- 2 Return to unrestricted normal activity, educating patient not to partake in contact sports until 12 weeks.
- 3 Commence full passive stretches as required.
- 4 Serial night extension splinting as required.
- 5 Assess patient for functional limitation and address as required (e.g. driving).

Week 12 appointment

- 1 Return to contact sport and/or manual lifting.

^aDuring each appointment, hand therapists provide patient education, wound and scar management, and assess splint suitability.

Complications of primary repair

The main complications of flexor tendon repairs are rupture, adhesions and joint contracture at the PIP joint.

Rupture

Every case of rupture should be reviewed, and trends in rupture rates need to be monitored. Risk factors can be divided into technical and patient related. Technical errors that lead to rupture are poor knot technique, insufficient size of bite leading to pull-out, damage to the core suture from the needle used for successive passes with the core suture or the epitendinous suture and bulky repair that then catches on the pulley system. When a ruptured tendon repair is re-explored it is important to carefully examine the suture to evaluate *how* the repair failed, although this does not necessarily answer the question of *why* the repair failed (Figure 51.9).

Patient factors are related to failure to follow the rehabilitation programme instructions, resulting in excessive strain at the repair

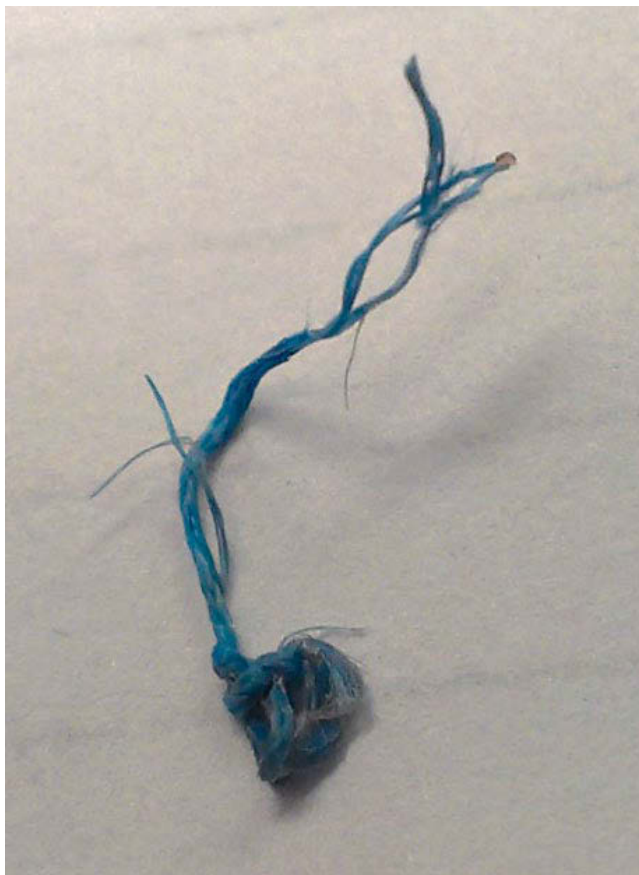


Figure 51.9 Inspection of the suture after a repair has failed should always be carried out as part of the learning process. Here the braided nylon suture appears to have been split just before the point where it gave way, and may represent a place where the suture went back through itself with damage being caused by the needle as it passed. The configuration that had been used in this case was a modified Kessler.

site and subsequent rupture. Unintentional excessive strain can also occur during sleep or on a sudden startle. Treatment of rupture is usually re-exploration and re-repair, unless there is a delay to presentation such that the repair is no longer possible or the pulleys have collapsed. In this case re-exploration is still needed but the patient should be consented for proceeding to a tendon graft or the first stage of a two-stage tendon graft.

Adhesions

Adhesions may also be due to technical factors, patient factors or a combination of the two. Technical factors include excessive handling of the tendon or instrumentation within the flexor sheath. Patient-related factors include pattern of injury and extent of crush, development of infection, delay to starting physiotherapy or failure to carry out sufficient mobilization during rehabilitation.

Treatment of adhesions is initially physiotherapy with two aims in mind: first, to ensure that full passive range of motion is restored and maintained, and second, to soften and break the adhesions that have been formed. Only when physiotherapy has been given a sufficient chance of success should surgery be considered, and in any case should not be embarked upon unless there is a full passive range of motion. Timing of surgical intervention is also important as tenolysis carried out too soon after the repair carries an increased risk of rupture.⁴² In all cases the patient should be warned of the risk of intraoperative tendon rupture and preparations made to proceed to the first stage of a two-stage tendon graft (see below) plus pulley reconstruction if the patient so wishes.

Proximal interphalangeal joint contracture

The PIP joint rapidly develops a contracture if full extension is not achieved during the rehabilitation exercises. If caught early, an adjustment to the splintage, with dorsal blocking to limit extension of the MCP joint, facilitates full PIP joint extension and may prevent the PIP joint contracture from becoming fully established.

Outcome measures

Audit and reporting of results should include rupture rates and some measure of the final motion achieved, such as the total active motion (TAM).⁴³ Added to this should be a patient-reported outcome to take account of overall satisfaction with the process as well as the final outcome.

Treatment of late presentation or failed repair

When a patient presents late with a divided or avulsed tendon, when the tendon has been damaged over a significant distance within zone 2, or when a tendon repair fails, the options to consider are as follows:

- *No treatment:* This may be appropriate for example where there is an isolated FDP injury and the DIP joint is stable.

- *Joint fusion* (e.g. in an isolated FDP rupture fusion of the DIP joint where it would otherwise hyperextend if used in pincer grip).
- *Direct repair*: This may still be possible in certain cases. However this should not be attempted without having a discussion with the patient as to which alternative will then be used if it is not possible. Direct repair should not be carried out if it would result in the finger being held too flexed, as will occur if the muscle has become fibrosed or fixed at a shorter length or more than 1 cm of tendon needs to be resected. The result of this would be a quadriga effect similar to when a tendon graft is set too tight (see below).
- *Single-stage tendon graft*: This may be possible in an early presentation of a rupture where the ends have retracted back preventing repair but the pulleys have not yet collapsed. This can only be assessed at the time of operation and every patient should therefore also be consented and prepared for the two-stage option.
- *Two-stage tendon graft*. This involves insertion of a silicone rod to reform the tendon sheath, followed at least six weeks later by removal of the rod and replacement with a tendon graft.
- In the thumb there is also the option of carrying out a *tendon transfer* using the FDS tendon of either the middle or ring fingers.

The choice between these options has to be made following an in-depth discussion with the patient about what is involved in the various surgical procedures, what rehabilitation is needed, and a realistic appraisal of the likely outcomes. Then, taking into account the patient's own priorities (e.g. self-employed versus retired) and preferences, it is possible to make an individualized decision as to what is the most appropriate course of action for them.

Tendon grafting

Principles

A graft of smaller calibre than the original tendon should always be used. The junctions at the ends of the graft must be outside the flexor sheath, and the tension needs careful adjustment to ensure that it is correct.

Prerequisites

The patient's hand must be quiescent with soft, supple and well-healed scars.

The patient needs to have a full understanding of the rehabilitation requirements and length of time needed before returning to normal activities.

Technique

In a two-stage repair the first stage can be divided into the following steps:

- 1 Exposure
- 2 Preparation of the graft bed
- 3 Insertion of the rod

- 4 Reconstruction of pulleys (if required).

The second stage can be divided into

- 1 Exposure
- 2 Harvesting the tendon graft
- 3 Passing the graft through the flexor sheath
- 4 Distal and proximal juncture repair.

A single-stage repair can be carried out if the assessment at the end of the exposure and graft bed preparation indicates that it is appropriate, and the operation then proceeds with harvesting the graft etc.

First stage

Exposure

The flexor sheath should be exposed for the entirety of its length from the start of A1 to the insertion of FDP. In addition the exposure needs to extend proximally enough to allow manipulation of the tendons for the proximal repair in the palm. The author prefers a mid-axial incision from the DIP to non-leading edge of the finger base. The neurovascular bundle is excluded from the raised skin flap. Extension across the pulp as well as into the palm is oblique.

Preparation of the graft bed

A 1 cm distal stump of FDP is left, and the section between this and a point just distal to the lumbrical origin is excised. If FDS is intact it should not be sacrificed. If it is found to be tight when inserting the graft or rod then one tail only of the FDS can be excised. If FDS has been divided as well it should be sharply transected 1–2 cm proximal to its insertion and removed, but before this is done a decision should be made as to whether the pulleys need to be reconstructed and if so whether the FDS is suitable for use for this purpose (see below).

At this point a decision will also need to be made as to whether a one-stage or two-stage approach is appropriate. If the pulleys are intact and open such that a graft will go through without the pulleys needing to be stretched or reconstructed first, then a one-stage approach is preferable. However, when there has been significant delay, or crushing element to the injury, or where infection has played a role in the rupture, this is unlikely to be the case and anything but the simplest stretching manoeuvre with the pulleys is likely to cause adhesion of any graft that is then passed through them at the same operation. Therefore in these cases a rod should be inserted plus or minus the reconstruction of any deficient pulleys as the first of two stages.

Insertion of the rod

All pulley remnants should be preserved and if necessary stretched with a haemostat. The silicone rod is passed from distal to proximal using a tendon retriever. The distal end is sutured to the FDP stump and the proximal end is left free in the palm, or in cases where there is damage and scarring in the palm such that the proximal juncture will need to be in the forearm, it is fed down through the carpal tunnel. Having placed it like this

with the distal end secure, the proximal end is pulled to test for bowstringing. If the silicone rod shows evidence of bowstringing the essential pulleys should be reconstructed.

Pulley reconstruction

The purpose of this is to recreate the essential features of the pulley system in order to prevent bowstringing of the flexor tendons, particularly in the region of the body of the proximal phalanx and middle phalanx where the original A2 and A4 pulleys would have been.

A tail of FDS can be used in the A2–3 region by leaving it attached at its insertion and suturing the other end to periosteum or pulley remnant, or using it to encircle as with a free tendon. Otherwise, there is usually enough tendon remnant available from the excision of FDP to use as a free tendon graft, or graft can be harvested from any of the usual donors. However, bearing in mind the need for further graft material for the tendon itself, fascia lata can be used for the pulley reconstruction and has the added advantage of providing a wider, flatter construct. If there is sufficient pulley remnant available, the graft can be used sutured to this and does not need to encircle the phalanx.⁴⁴ If not, the graft is used to encircle the rod and the relevant phalanx, at least twice and preferably three times in order to reconstruct a long pulley of equivalent strength to the original.⁴⁵ A minimum of 6 cm of graft is needed to encircle twice. At the level of the proximal phalanx the graft is passed deep to the extensor tendon, but at the level of the middle phalanx it should be passed superficial to the extensor tendon, or kept to a minimum bulk with only two passes.

The skin is then closed and hand splinted in the position of safe immobilization for the immediate postoperative period only. Beyond this, physiotherapy is aimed at ensuring that the full passive range of motion is maintained.

Second stage

The exposure is carried out through the same incision as for the first stage.

Harvesting the tendon graft

Potential tendon donors to be considered include the following:

- *Palmaris longus*: This is easily harvested with no donor deficit and keeps all the surgery to the one limb. However it is only sufficient for one palm-to-finger tip graft.
- *Plantaris*: This will yield enough graft for up to three forearm-to-finger tip grafts.
- *Extensor indicis proprius and/or extensor digiti minimi*: Each of these will provide one palm-to-finger tip graft.
- *Long extensors of the second, third and fourth toes*: Each of these will provide one forearm-to-finger tip graft.
- *Fascia lata*: Strips of fascia lata 1–2 cm wide can be rolled to form tendon-like structures, and sufficient is available to harvest as many forearm-to-finger tip grafts as are needed.

Passing the tendon through the flexor sheath

The tendon graft is sutured to the proximal end of the silicone rod and pulled through the newly formed flexor sheath from proximal to distal, minimizing any trauma to the flexor sheath.

Distal and proximal juncture repair

The distal juncture repair will be sited at the FDP insertion. The proximal end should be either just distal to the lumbrical origin from FDP, or in the forearm. This will have been decided at the time of silicone rod insertion and is predicated by the extent of involvement of the palm in the original injury with subsequent scarring. Several different methods for each of these have been described, and a number of different combinations can be chosen. However, it must be remembered in choosing a combination that it needs to be possible to test or adjust the tension prior to carrying out the repair of the second end so that the tension can be finely tuned at this point. This is judged by setting it so that the finger is just a fraction more flexed than it would normally sit in the normal resting cascade. This is normally done with a fixed-length technique distally, adjusting the tension proximally. Fixed-length distal juncture repair methods are the same as the repair methods used in zone 1 tendon repairs.

The commonest proximal repair in which the length can be adjusted is the Pulvertaft weave. Much interest has been shown in discussing the strength related to the number of weaves and the minimum number of weaves needed to provide sufficient strength is understood to be 3.⁴⁶ This is, however, a little misleading as the strength of the repair can only come from the sutures used between the two tendon ends as it does not matter how many weaves you provide, the tendons will come apart with minimal tension applied if there are no sutures used. The requirement for a minimum number of weaves is more likely to be related to the minimum length of overlap needed to fit in a sufficient number of sutures perpendicular to the line of force. The benefit of the Pulvertaft weave is the ease of setting the correct tension. This is done by pulling the two tendon ends to an estimated correct position, placing a single suture, then releasing the tendons to test for the resting cascade. If the tension is incorrect, the suture can be removed and the tension adjusted. Once the correct tension has been achieved the rest of the sutures are placed.

Another approach is to use a fixed-length technique proximally and adjust the tension at the finger tip. This is done by taking the graft right out through the end of the finger, laid along the full length of the distal phalanx. A clip is applied to the graft at the end of the finger and tension testing carried out, moving the clip as necessary to ensure the correct tension. Once it has been correctly tuned the two halves of the FDP stump are sutured to the graft and the end of the graft is cut flush with the skin at the finger tip.

Postoperatively a CAM regime is used similarly to that after a primary repair.

Complications following tendon graft

Possible specific complications include:

- **Rupture:** This requires exploration and re-repair if diagnosed quickly enough.
- **Adhesions:** These are managed in the same way as adhesions after primary repair.
- **Lumbrical plus finger:** If the graft was set too long the origin of the lumbrical is pulled too far proximal by the force applied through FDP. This leads to too much power being transferred through the lumbrical such that the finger becomes more resistant to flexion when the patient attempts active flexion despite being able to demonstrate a full range of flexion passively.
- **Quadriga:** This is the opposite problem and occurs when the graft is set too tight. In this case the affected digit lies too flexed in the resting cascade and flexes ahead of the other digits in attempted fist formation. When grasping an object the affected finger makes contact with the object ahead of the other digits and arrests any further pull on the FDP in this finger. Since FDP is a mass action muscle with all four tendons fixed in length in relation to one another this means that no additional power can be exerted through the other digits and grip strength is therefore weaker.⁴⁷

Summary

Tendon injuries are common. Outcomes are best when they are correctly diagnosed and treated at an early stage. Good surgical technique is essential in order to avoid rupture or adhesions, but of equal importance is the preparation of the patient so that they are expecting a long and complex rehabilitation programme. Patient compliance must be facilitated with a well-run, supervised programme that is delivered with clear instructions and goals.

Secondary reconstruction is complex and rarely results in the same level of function as a successful primary repair.

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CHAPTER 52

Extensor tendon injuries

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Introduction

Repair of extensor tendon injuries remains a common starting point in the careers of many aspiring hand surgeons. In contrast to their flexor tendon equivalents, extensor tendon repairs often appear relatively straightforward. The surgeon is free from the constraints imposed by the fibro-osseous canals during flexor tendon repair. Furthermore, the ease of surgical exposure, likely absence of proximal stump retraction, simpler suture placement configurations and straightforward postoperative mobilization regimes frequently results in the delegation of these injuries to junior surgical trainees.

However, although many extensor tendon repairs are relatively undemanding, these injuries can be more complex than anticipated. Lacerations over the dorsum of the wrist can produce significant proximal stump retraction, particularly of extensor pollicis longus (EPL), which can be notoriously difficult to find. Delayed presentations or injuries involving actual tendon loss produce deficits in the tendon which preclude direct repair without producing unacceptable tension at the repair site and tendon shortening. Therefore, the surgeon will need to be cognizant of the surgical principles and techniques involved in tendon grafts and tendon transfers.

The cross-sectional morphology of extensor tendons varies significantly according to the site of the injury. Thin flat tendons encountered over the dorsum of the fingers require a different suture technique in comparison to the more rounded thicker counterparts encountered over the dorsal wrist joint. These injuries often have concomitant bone and joint injuries, requiring accurate reduction and internal fixation, joint irrigation and capsular repair. Associated nerve injuries may be present, usually the sensory branches of the radial nerve and occasionally the posterior interosseous nerve, requiring microsurgical repair. Appropriate peri- and postoperative splinting, mobilization and hand therapy are essential to avoid early tendon ruptures and joint contractures and to minimize adhesions to overlying skin.

Consequently, although straightforward injuries will be ideal for the surgical trainee, complex extensor tendon injuries present the hand surgeon with a challenging test of their surgical capabilities.

Anatomy

Forearm

The lateral epicondyle is the origin of the wrist extensors extensor carpi radialis longus and brevis (ECRL and ECRB), extensor carpi ulnaris (ECU), extensor digitorum communis (EDC) and extensor digiti minimi (EDM). Distally, the proximal radius, ulna and interosseous membrane give origin to extensor indicis (EI), abductor pollicis longus (APL), extensor pollicis longus (EPL) and extensor pollicis brevis (EPB).

The radial nerve provides motor branches at or above the lateral epicondyle to brachialis, brachioradialis (BR) and ECRL. The nerve then divides into a superficial sensory branch and its motor branch, the posterior interosseous nerve (PIN). The sensory branch continues distally applied to the deep surface of BR into the wrist and hand. The PIN supplies ECRB and penetrates supinator, supplying it and the remaining extensors, and continues distally to the wrist closely applied to the posterior interosseous membrane.

Wrist

The musculotendinous junction is located approximately 4–5 cm proximal to the wrist joint, whereas at the level of the wrist, a condensation of the fascia over the extensor muscles produces the extensor retinaculum, which prevents bowstringing (Figure 52.1). The retinaculum allows passage of the extrinsic extensor tendons through six separate compartments, each a discreet synovial lined fibro-osseous tunnel:

- Compartment 1 – abductor pollicis longus (APL) and extensor pollicis brevis (EPB)
- Compartment 2 – extensor carpi radialis brevis (ECRB) and extensor carpi radialis longus (ECRL)
- Compartment 3 – extensor pollicis longus (EPL)

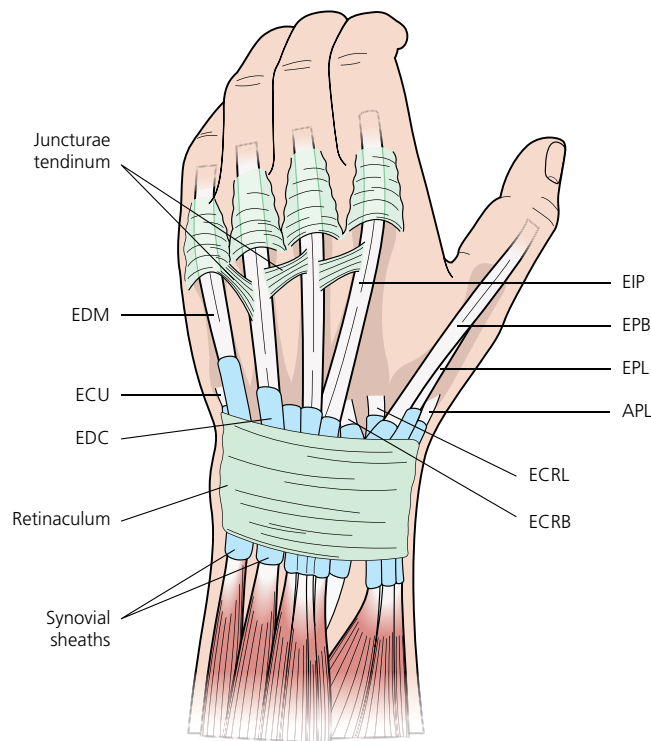


Figure 52.1 Dorsal hand and wrist extensor tendon structures. EIP, extensor indicis proprius; EPB, extensor pollicis brevis; EPL, extensor pollicis longus; APL, abductor pollicis longus; ECRL, extensor carpi radialis longus; ECRB, extensor carpi radialis brevis; EDM, extensor digiti minimi; ECU, extensor carpi ulnaris; EDC, extensor digitorum communis.

- Compartment 4 – extensor digitorum communis (EDC) and extensor indicis proprius (EIP)
- Compartment 5 – extensor digiti minimi (EDM)
- Compartment 6 – extensor carpi ulnaris (ECU).

After emerging from the extensor retinaculum, the tendons of EDC are joined by intertendinous connections, junturae tendinum. Although their presence is reliable, the level at which they occur, proximal to the metacarpophalangeal (MCP) joint, is not. They are invariably present between the EDC tendons of middle, ring and small fingers. Less reliable, however, is a juncturum between the communis tendons of middle and index fingers. Junturae do not attach to EIP, an aid in tendon identification.¹ The architecture of these structures has clinical significance when applied to radial sagittal hood (RSH) repair, discussed later.

Anatomical variations

Variations in both the configuration and number of extensor tendons are extremely common and are well described elsewhere.² In general, the tendons of EDC to the index and middle fingers are singular, whereas the ring and small finger tendons are occasionally multiple. EIP lies ulnar to the index finger communis tendon and is smaller in diameter. Occasionally, it may be duplicate or located radially.³ Both EDM and APL have one, two or four component tendons.

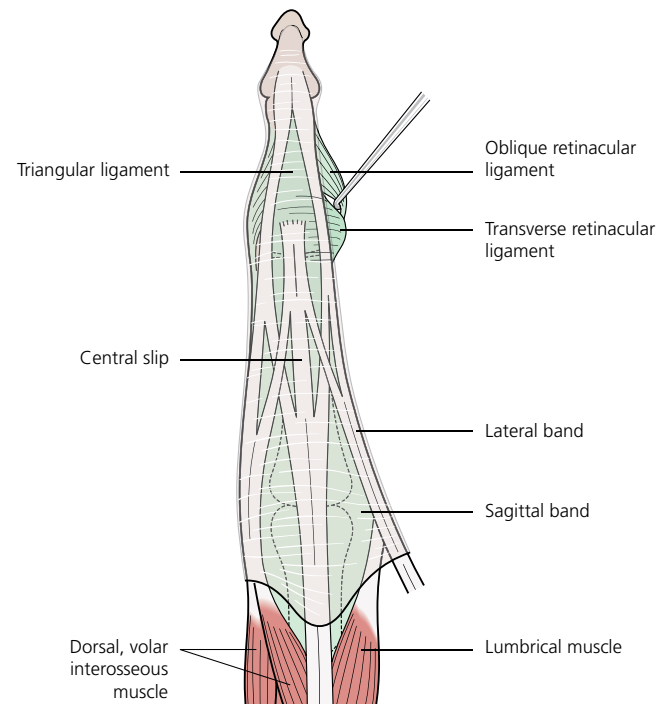


Figure 52.2 Intrinsic musculature.

Finger extension

Finger extension is produced through a combination of two distinct motor units that have separate neural innervation systems. The radial nerve innervates the extrinsic extensors, producing extension of the MCP joints. The intrinsic system, composed of dorsal and palmar interossei and lumbrical muscles, is innervated by both the median and ulna nerves.⁴

Metacarpophalangeal joint

Extension at the MCP joint is produced solely by the extrinsic extensors. Central alignment of the extensor tendon as it traverses over the MCP joint is critical and is maintained by sagittal bands, both radial and ulna, which attach to the volar plate and intermetacarpal ligament.⁵

Although division of the ulna sagittal band produces no clinical sequelae, a laceration or rupture of the radial sagittal band results in extensor tendon subluxation or dislocation into the adjacent ulna gutter.⁶ Consequently, the extensor tendon loses its moment arm across the MCP joint, resulting in incomplete extension and ulna deviation.

Fingers

Beyond the MCP joint, the extensor tendon trifurcates into a central slip and two lateral slips. The central slip continues distally in the midline to insert on the dorsal base of the middle phalanx, producing proximal interphalangeal (PIP) joint extension (Figure 52.2). It remains centralized by the transverse retinacular ligament. The lateral slips course distally around

both sides of the PIP joint, where they are joined by the lateral bands of the intrinsic muscles. The conjoined lateral bands meet, combine and attach to the base of the distal phalanx, producing distal interphalangeal (DIP) joint extension.

Intrinsic muscles

The interossei and lumbricals comprise the intrinsic musculature. The interossei course dorsal to the deep transverse metacarpal ligament and join the lateral slips around the mid point of the proximal phalanx. The lumbricals arise from the flexor digitorum profundus (FDP) tendon, pass volar to the deep transverse metacarpal ligament, and attach to the radial lateral slip of each finger.

At MCP joint level, the intrinsic muscles and tendons are both volar to the axis of rotation. However, when the muscles and tendons reach PIP joint level, both become dorsal to that axis. The lumbricals act as the primary intrinsic extension mechanism.⁷ In contrast, the interossei contribute to IP joint extension only when the MCP joints are simultaneously flexed.⁸ The extensor mechanism is anchored in position across the PIP joint by the transverse retinacular ligament. Synchronous PIP and DIP joint extension is achieved by a mechanism in which the central slip extends the PIP joint and the lateral bands, circumventing the PIP joint, extend the DIP joint.

The critical element in this synchronous movement is the balance between the three components. Most importantly, the lengths of the central slip and the two lateral bands must be such that synchronous extension is produced. Thus, as the middle phalanx reaches alignment with the proximal phalanx, so too is there concomitant alignment of the distal phalanx.⁹

The transverse retinacular ligament extends from the conjoined lateral bands to the flexor sheath at PIP joint level, and acts to prevent dorsal dislocation of the lateral bands.¹⁰ The oblique retinacular ligament (ORL) connects the conjoined tendons from the dorsum of the distal phalanx to the volar checkrein ligaments at the PIP joint. The ORL thus lies volar to the PIP joint and sits dorsal to the axis of the DIP joint, allowing coordination of flexion and extension within those joints.¹¹ The triangular ligament is a fascial band connecting the conjoined lateral bands and the terminal tendon. It prevents volar subluxation of the conjoined lateral bands during PIP joint flexion.¹²

Zones of injury

Fingers

The classification of Kleinert and Verdan featuring eight zones of extensor tendon injury has endured (Figure 52.3).¹³ A ninth zone, relating to purely muscular injury in the mid to

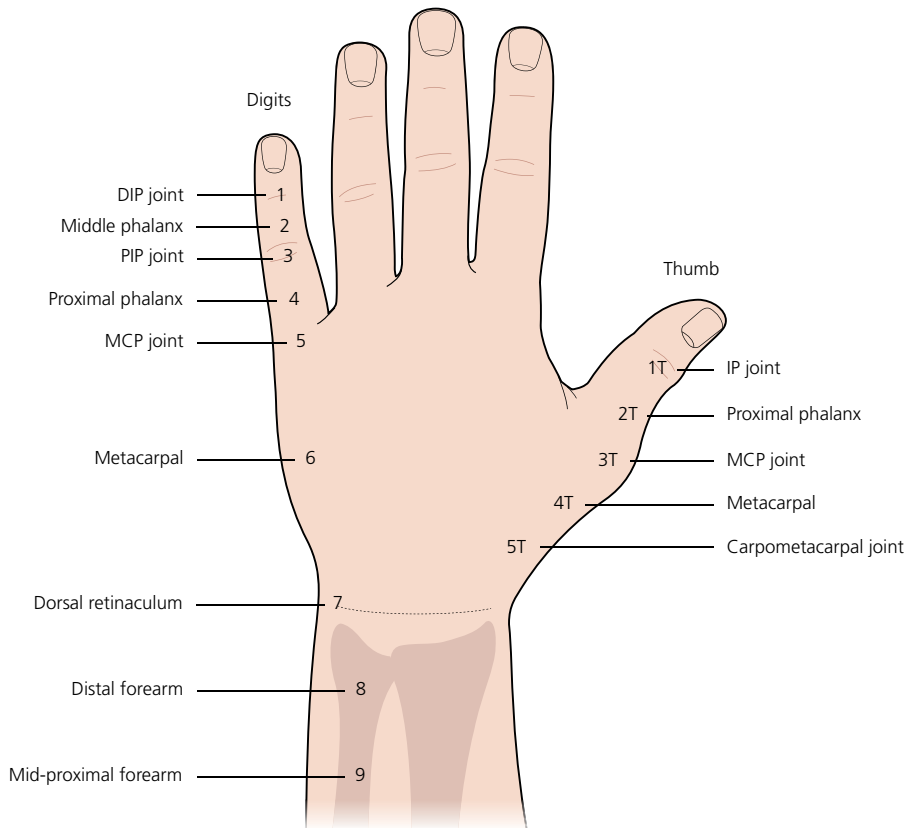


Figure 52.3 Extensor tendon zones.

proximal forearm, was described by Doyle.¹⁴ As an aide memoire, excepting zone 9, odd numbered zones are located over joints.

- Zone 1 – DIP joint
- Zone 2 – middle phalanx
- Zone 3 – PIP joint
- Zone 4 – proximal phalanx
- Zone 5 – MCP joint
- Zone 6 – dorsum of hand
- Zone 7 – extensor retinaculum
- Zone 8 – distal forearm
- Zone 9 – muscular area, middle and proximal forearm.

Thumb

APL, EPB and EPL all play separate roles in thumb extension. APL inserts into the base of the first metacarpal, EPB attaches to the base of the proximal phalanx while EPL maintains a broad area of contact with the base of the distal phalanx.

As it traverses the dorsum of the MCP joint, EPL is anchored on the ulnar side by the aponeurosis of adductor pollicis, whereas radially, abductor pollicis brevis (APB) performs a similar role. Consequently, EPL is held securely in a centralized position as it courses over the metacarpal head. Consequently, proximal stump retraction is found only when EPL division occurs proximal to the MCP joint.

- Zone 1 T – IP joint
- Zone 2 T – proximal phalanx
- Zone 3 T – MCP joint
- Zone 4 T – metacarpal
- Zone 5 T – carpometacarpal joint.

Suture techniques

The optimal suture material will maximize the strength of repair, prevent rupture, reduce skin adhesions and minimize tendon shortening. Additionally, suture techniques are predicated on extensor tendon thickness, shown by Doyle to vary from 0.55 mm in zone 2 to 1.7 mm in zone 6.¹⁴ I prefer to use a 4.0 non-absorbable core suture, and a 5.0 monofilament nylon suture for a cross-stitch or epitendinous suture.

The suture configurations are zone dependent. In general, I prefer:

- Zone 1: DIP joint, running suture incorporating skin and tendon
- Zone 2: distal finger – simple running suture oversewn with Silfverskiöld cross-stitch
- Zone 3–5: modified Kessler in thickest portion of tendon, dorsal cross-stitch
- Zone 6–7: modified Kessler in thickest portion of tendon with circumferential epitendinous suture.

Postoperative regimes

Extensor tendon repairs in zones 1 and 2 are immobilized in full DIP joint extension for 6 weeks, employing a splint or Kirschner wire fixation. Patients with zone 3, 4 and 5 injuries are managed with splinting in 30–40° of wrist extension, 20° of MCP joint flexion and full extension across the PIP joints for between 4 and 6 weeks. Zone 6, 7 and 8 injuries are splinted in wrist extension and mild MCP joint flexion for 4 weeks. EPL repairs are splinted in 40° of wrist extension, 10° of MCP joint flexion and IP joint extension for 4–6 weeks, with the thumb held in a retroposed position to minimize tension across the site of repair.

Static splinting in extension for 3–6 weeks followed by gradual mobilization has been the traditional postoperative management of these injuries. More recently, controlled active mobilization (CAM) protocols and dynamic splinting have been employed. CAM involves active joint extension while limiting joint flexion through a palmar splint. In comparison, dynamic splinting involves passive joint extension with an outrigger, again with a palmar splint blocking flexion.

Newport published a retrospective analysis of static splinting, concluding that loss of flexion was the most significant complication while the average degree of loss of flexion was greater than the extension lag. Furthermore, they found that distal zone injuries^{1–4} produced significantly worse results than the more proximal zones, and unsurprisingly, complex injuries were associated with poorer outcomes.¹⁵

Khandwala's study in 2000 showed no difference in outcomes when comparing CAM to dynamic splinting.¹⁶ For suitably motivated and supervised patients with injuries in zones 4–7, these methods are beneficial and have a very low rupture rate. In the very young, and patients suspected of being poorly motivated and/or non-compliant, static mobilization is appropriate.

Complications

Adhesion formation at the site of repair, to overlying skin and underlying bone, is the most common complication of extensor tendon repair, producing extensor lag and loss of flexion. An extensor tenolysis should not be considered until gains have plateaued from therapy, and usually no sooner than 4–6 months after the initial repair. Ideally, a good range of passive motion should be present prior to proceeding with tenolysis. Capsulotomy, collateral ligament release and flexor tenolysis may need to be considered, either at the time of tenolysis, or following a poor outcome from tenolysis.

Zone 1: Distal interphalangeal joint level

Acute injuries

Loss of active DIP joint extension resulting from extensor tendon disruption in zone 1 produces a mallet finger deformity. It results from forces producing sudden flexion across the DIP

joint, commonly a direct blow to the fingertip. The injury is usually closed and the tendon disruption may be accompanied with a fracture of the dorsal lip of the distal phalanx, with joint subluxation in larger fractures. Crush injuries, saw and knife wounds are among the more common mechanisms resulting in open injuries to the extensor apparatus and, on occasion, the underlying DIP joint. Radiological assessment is essential in management of these injuries. The dominant hand is most frequently injured, with the small ring and middle fingers accounting for 90% of all such injuries.¹⁷

In the absence of subluxation or a fracture involving less than a third of the articular surface, continuous DIP joint immobilization in full extension for at least 6–8 weeks is appropriate. A further 2 weeks of splinting at night and during strenuous exercise is commonly recommended.² Results of this non-operative treatment regime have reliably shown 80% of patients demonstrate a persistent residual extension lag of 10°, usually not a functional concern.¹⁸

Operative treatment is indicated in open injuries, patients unable to work with a splint, and those with a large dorsal bony fragment with palmar subluxation of the distal phalanx.¹⁹ Where possible, direct repair of the extensor tendon should be performed. If divided at its insertion, many methods, including overlying skin suture with splinting, pull-out wires, and Kirschner wire fixation of the DIP joint have all been advocated.²⁰ Recently, bone anchors have become a popular method of reattaching EDC to its bony insertion.

The most contentious area of treatment remains the management of the substantial fracture fragment. Again, many methods of reduction and fixation have been reported, including

Kirschner wires, tension band wiring, screws, hook plates and mini external fixation.¹⁹

An elegant method of closed reduction of mallet fractures is DIP joint extension block pinning. This technique involves flexing the DIP joint and passage of a Kirschner wire into the head of the middle phalanx (Figure 52.4). The DIP joint is then brought back into extension, or as near as the dorsal blocking wire will allow, and a longitudinal Kirschner wire is passed across the DIP joint.²¹ The wires can be removed after radiological confirmation of bony healing, usually at 4–6 weeks.

Chronic injuries

Prior treatment, passive DIP joint motion and attitude of the PIPJ are all vital considerations in the management of chronic mallet finger. An 8-week trial of splinting is often attempted as, although benefits may be minimal, there is no significant downside. Radiological evidence of DIP joint arthritis or chronic palmar subluxation both negate the efficacy of further conservative treatment.

Tenodesis involves elliptical wedge excision of several millimetres of skin and tendon, with the DIP joint wired in full extension for 6 weeks.

A central slip tenotomy can be employed for patients in the presence or absence of an established swan-neck deformity.²² Optimal results from this procedure are seen in patients with extensor lags up to 35°.

Thompson described the technique of spiral oblique retinacular ligament reconstruction (SORL). Although bone anchors have replaced the original pull-out suture described, the technique involves securing a tendon graft to the insertion of EDC. The graft is taken proximally between the volar plate and flexor

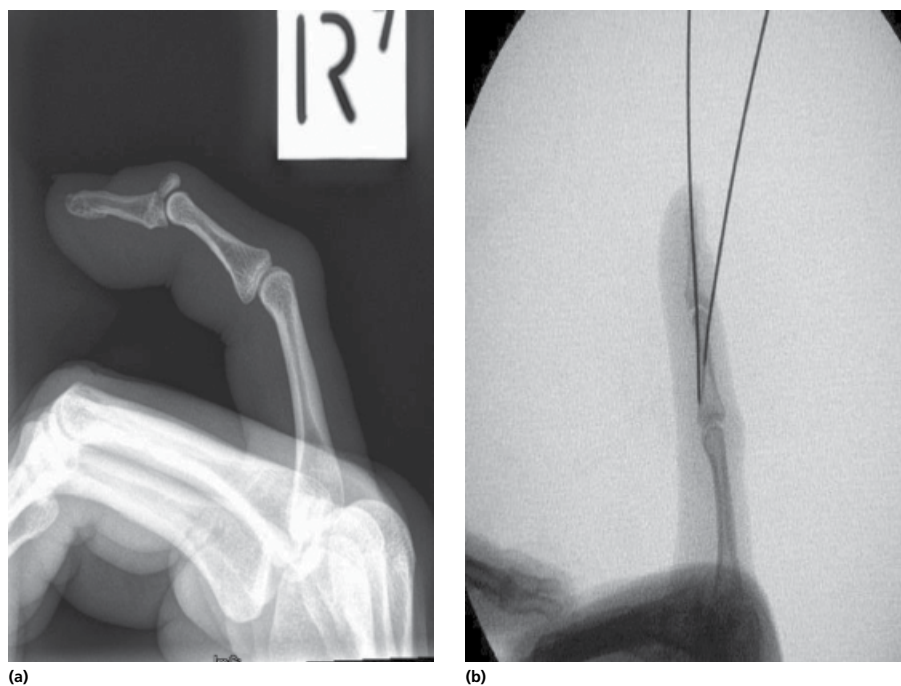


Figure 52.4 Dorsal blocking technique, DIP joint. (a) Zone 1. Preoperative. (b) Intraoperative.

tendons across the PIP joint and anchored into the proximal phalanx.²³ This procedure is appropriate for mallet finger deformities with extensor lags of 45° or greater. DIP joint fusion remains appropriate for arthritic and painful chronic mallet finger deformities and those with chronic palmar subluxation.

Zone 2: Middle phalanx level

Acute injuries

Knives, saw blades and crushing injuries are frequently responsible for extensor tendon disruption in this region. One or both of the lateral bands may be divided, as may the triangular ligament. Sharp lacerations are repaired with 5.0 nylon in a figure of eight manner or a running configuration. A cross-stitch as described by Silfverskiöld may also be added.¹⁴ Where a segmental defect precludes primary repair due to unacceptable tension, a lateral band turndown provides an elegant solution.¹⁴ Swan-neck deformities resulting from a chronic zone 2 injury are approached using the SORL reconstruction, described previously.

Zone 3: Proximal interphalangeal joint level

Acute injuries

Loss of PIP joint extension follows disruption of the extensor mechanism at, or just proximal to, the PIP joint. Lacerations and blunt trauma can produce injuries including any or all of

complete or partial divisions of the central slip and lateral bands, breaches of the joint capsule and fractures of the dorsal lip of the base of the middle phalanx. Palmar dislocation of the PIP joint results in central slip injuries ranging from attenuation to complete disruption.

Central slip injury results in the loss of PIP joint extension, allowing that joint to assume a flexed attitude. Should the injury result in volar migration of the lateral bands, the DIP joint subsequently goes into hyperextension, resulting in a boutonniere deformity, which may manifest within 10–14 days post injury.

Closed injuries

Treatment of an acute closed central slip rupture consists of splinting or Kirschner wire fixation of the PIP joint in full extension for 6 weeks, while encouraging active DIP joint flexion exercises to promote dorsal migration of the lateral bands.

Rarely, the central slip is avulsed with a fracture fragment from the dorsal lip of the middle phalanx. Small undisplaced fragments can be ignored, and managed by splinting or Kirschner wire fixation as described. The treatment of choice for larger displaced fragments is more controversial. Kirschner wire fixation and screw fixation have been advocated, as has excision of the fracture fragments and tendon repair to bone with pull-out wire or suture anchor.²⁰

Where possible, I prefer to reduce the fragment by placing a dorsal blocking technique as described previously (Figure 52.5). The wires are removed at 6 weeks.

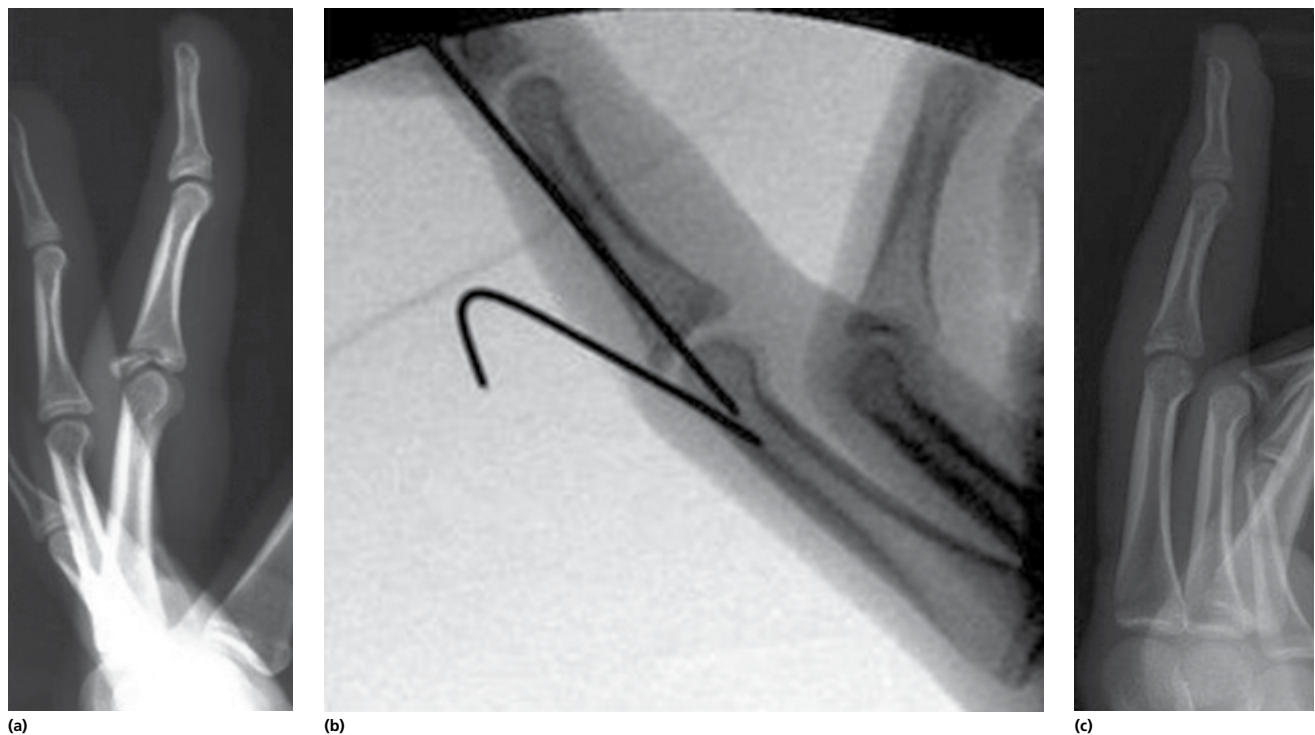


Figure 52.5 Dorsal blocking technique, PIP joint. (a) Preoperative. (b) Intraoperative. (c) Postoperative.

Open injuries

Wounds over the dorsum of the PIP joint raise two major questions. Has the central slip been breached, and if so, has the joint been entered? Transection of the central slip may be partial or complete, and may also involve one or both lateral bands (Figure 52.6). Primary repair with a core suture such as a modified Kessler with a braided synthetic suture on a taper needle, oversewn with a cross-stitch is used.

When the division is at, or near, the bony insertion of the central slip, reattachment to bone using a bone anchor provides a simple and secure option. Postoperative management requires splinting of the PIP joint in full extension for 5–6 weeks. Any concerns regarding the patient's postoperative compliance are best addressed by placing a Kirschner wire across the joint. Where a deficit exists in the central slip precluding primary repair, the distally based flap of extensor tendon as described by Snow is an elegant technique.²⁴

Aiche recommended central slip defects be repaired with midline advancement of both lateral bands, which are sutured together once dissected free of ORLs.²⁵ When the central slip is detached to a displaced intra-articular fracture of the dorsal lip of the middle phalanx, I employ a technique using dorsal blocking wires identical to that for treating a similar injury in a mallet finger.

Chronic injuries

Chronic boutonnière deformities result from traumatic central slip disruption or elongation secondary to inflammation. Volar migration of the lateral bands increases PIP joint flexion and DIP joint extension, eventually producing fixed contractures of both joints. Dynamic splinting and serial casting are indicated when passive DIP joint flexion is possible when the PIP joint is held in extension, and should also be considered preoperatively to optimize the passive range of motion within the joints.

In the absence of passive DIP joint flexion with the PIP joint held in extension, Fowler's central tenotomy corrects DIP joint hyperextension, and reduces PIP joint flexion. A mallet finger does not result, perhaps due to fibrosis and shortening of joint capsule and ligaments. Patients with fixed contractures require physiotherapy and splintage prior to any surgical procedure. Furthermore, the surgeon must be cognizant not to sacrifice preoperative PIP joint flexion for a postoperative stiff extended PIP joint.

If electing to proceed, various tendon relocation procedures have been described. Matev advocated division of the lateral bands, using one for central slip reconstruction, the other for DIP joint extension.²⁶ Littler and Eaton described division of both lateral bands distal to the PIP joint, conserving the lumbrical tendon for DIP joint extension, and translocating the lateral bands to the midline, fixing them to the base of the middle phalanx.²⁷ PIP joint fusion or arthroplasty are appropriate procedures for patients with arthritic PIP joints.

Zone 4: Proximal phalanx level

Acute injuries

When caused by sharp instruments, these extensor tendon injuries are usually straightforward. Regardless of whether the tendon wound is complete or incomplete, the anchoring roles of the sagittal bands prevent any proximal stump retraction. Repair is managed with a modified Kessler suture of non-absorbable (polyester) material and an epitendinous cross-stitch.

Major crush injuries, circular saws, chain saws and similar produce far more complex problems in this region. In these circumstances, defects of 10 mm or more may be produced not only in the extensor mechanism, but also in the dorsal cortex of the proximal phalanx as well as the overlying skin. Options for



(a)



(b)

Figure 52.6 Boutonnière deformity. (a) Zone 3. Boutonnière deformity 10 days post dorsal PIP joint laceration. (b) Zone 3. Central slip division with breach of dorsal capsule exposing PIP joint.

extensor tendon reconstruction include tendon grafting, tendon transfer or a reverse plasty of EDC as described by Foucher *et al.*²⁸

After initial wound debridement and underlying fracture stabilization, local flaps may be needed to provide stable skin cover. Since complex injuries in this region are frequently associated with adhesions, early mobilization is desirable.

Zone 5: Metacarpophalangeal joint level

Acute injuries

Septic arthritis

Open wounds located over the dorsum of the MCP joint are common and frequently result from a punch to a human mouth – the ‘fight bite’ injury. Apart from the contaminated skin wound, penetration of human teeth can cause partial or complete division of the extensor tendon and contaminate the underlying joint with oral flora. Due to the seemingly innocuous appearance of the initial wound, presentation is often delayed for several days, during which time contamination proceeds to florid infection.

Pain, swelling, erythema and discharge from the wound, usually the middle finger of the dominant hand, complete the common clinical picture. X-Rays can appear normal, display a foreign body (tooth), or show evidence of septic arthritis or osteomyelitis.²⁹

Cultures from affected joints commonly reveal infection with streptococcal and staphylococcal organisms.³⁰ Prompt wound exploration, preferably within 24 h, is required. The MCP joint must be entered, cultured and copiously irrigated. Depending on the operative findings, repeat irrigation may be required after a further 48 h. Repair of the tendon is best deferred until completely satisfied the underlying joint is free of infection. Since the tendon injury is frequently partial, early active mobilization can be commenced within one week after conversion to a clean wound.

Sagittal band injuries

Blunt MCP joint trauma, forced MCP joint flexion and, in a position of resisted extension, even seemingly insignificant injuries are all capable of sagittal band injury. The radial sagittal band, usually in the ring or middle finger, is the most commonly affected.⁵

Clinically, the patient may describe a snapping or clicking during MCP joint flexion, occasionally leading to confusion with a trigger finger. These patients demonstrate a subluxation of the extensor tendon on finger flexion (Figure 52.7).

More severely affected patients will display a complete dislocation of the affected extensor tendon into the ulna gutter. With the extensor tendon no longer centralized over the metacarpal head, the moment arm is lost, and MCP joint extension will produce both extensor lag and ulna deviation. Four weeks of buddy taping is appropriate for symptomatic patients, and those with subluxation, if commenced within 2–3 weeks of injury.



Figure 52.7 Subluxation of EDC middle and ring fingers.

Good results have been obtained from wearing an MCP flexion-blocking splint while allowing PIP joint flexion, for a period of 6–8 weeks.³¹

The most difficult group to treat are those who fail conservative management, and those presenting more than a month post injury. Kettelkamp *et al.* recommended direct repair of the RSH, aiming to restore centralization of the extensor tendon as it traverses the metacarpal head. When necessary, they advocated concomitant release of the ulna sagittal band.⁵

In long-standing cases, direct repair of the RSH may not be possible. A plethora of ingenious methods have been described to reconstruct the extensor hood.⁴ Personally, I find the most effective method to achieve centralization and prevent dislocation into the ulna gutter is Wheeldon's technique, involving use of an adjacent junctura tendinum between the middle and ring fingers (Figure 52.8).³²

At operation, a junctura tendinum connecting the affected digit and the adjacent ulna digit is identified. It is detached from the adjacent tendon, and draped over the affected extensor tendon, and imbricated into the palmar radial sagittal band, thus acting as a sling preventing ulna dislocation.

Although the anatomy of the distribution of the junctura tendinum is variable, it is rare not to find one available for this purpose. Should no tendinum be readily available, McCoy described a proximally based sling of the mid third of the extensor tendon looped around the lumbrical and sutured to itself, again acting as a sling to prevent ulna subluxation. The MCP joint is splinted in extension for 6 weeks, and PIP joint flexion is commenced after 2 weeks.³³

Open injuries

While lacerations to the sagittal bands are uncommon, they can produce loss of MCP joint extension as well as extensor tendon subluxation to the opposite side of the MCP joint.¹⁴ Most

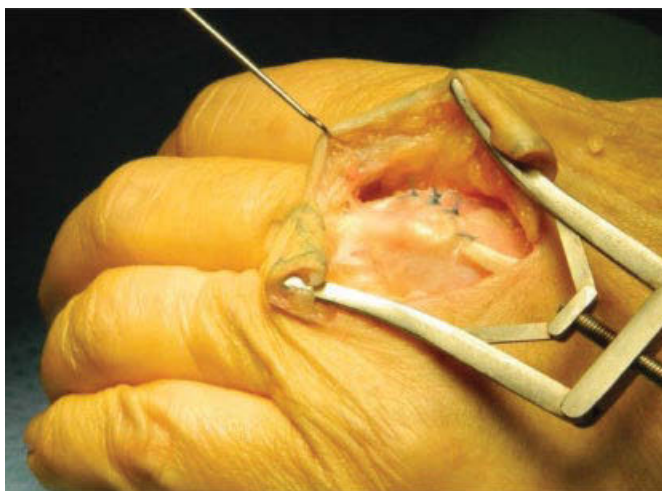


Figure 52.8 Wheeldon's technique for correction of extensor tendon subluxation in zone 5.

commonly affected are the radial sagittal bands to either the ring or middle fingers, and repair can be performed using a simple suture of braided polyester.

Zone 6: Metacarpal level

Acute injuries

In comparison to more distal zones, zone 6 repairs achieve more favourable outcomes as they are not complicated by underlying joint injury or damage to intricate flexor and extensor tendon systems.

Additionally, extensor tendons at this level have a smaller surface area, and are generally covered by substantially thicker subcutaneous tissue. All these factors combine to reduce the potential for adhesion formation after tendon repair. A braided polyester core suture in a modified Kessler or modified Bunnell configuration is appropriate, whereas use of a cross-stitch is at the surgeon's discretion. In a static splint the wrist is placed in 30° of extension, with the MCP joints in neutral. Dynamic mobilization is appropriate in compliant patients.

In late presentations, direct repair may not be possible, in which case end to end or end to side transfer to an adjacent tendon is appropriate. In more severe defects, tendon transfers may be required, with EIP providing an excellent transfer. Where the overlying soft tissue is satisfactory, a two-stage tendon graft utilizing silicone rods is another reconstructive option.

Zone 7: Dorsal wrist level

Acute injuries

Penetrating trauma at this level usually injures both the extensor retinaculum and the underlying extensor tendons. When the cross-sectional diameter of the tendon permits, a core suture repair using a 4.0 non-absorbable suture is performed. Frequently, the proximal tendon ends are markedly retracted, requiring

extensive exposure. Severe wounds, such as those from a chain saw, cause problems not only with tendon identification, but also with ragged tendon ends and significant segmental loss of tendon. Tendon grafting or tendon transfer may be necessary under these circumstances. Where possible, I prefer to repair at least some part of the extensor retinaculum to prevent subluxation or bowstringing of the tendons.

The surgeon should be aware of anatomical variations within APL, frequently in one, two or four slips, creating the potential for confusion when attempting to repair proximal and distal stumps. Repairs of APL and EPB are commonly associated with repair of sensory branches of the radial nerve.

At their osseous insertions, the tendons ECRL and ECRB are relatively flat and broad. The insertion of, now commonly available, anchoring devices has proved to be a more effective, simple and elegant technique to reattach these tendons than the pull-out wire techniques used previously.

Chronic injuries

Septic arthritis of the wrist, Colles fractures and aggressive bouts of dorsal wrist synovitis all predispose EDC and EPL to attrition ruptures. EPL rupture is best managed with an EIP tendon transfer whereas ruptures of EDC are treated with suture of the distal stump to adjacent extensor tendons. Alternatively, FCU can be used as a new motor.

Zone 8: Proximal and distal forearm

Open distal forearm injuries produce disruption at the musculotendinous junction. Achieving adequate strength of repair is difficult due to the diaphanous nature of the overlying fascia. Occasionally, a side-to-side tendon transfer may be required.

Penetrating injuries to the proximal half of the forearm, usually the result of glass, chain saws or knives, result in partial or total muscle belly divisions. Intramuscular fibrous septa may

help with orientation of muscle bellies, and also provide the most robust structures for suture placement. Concomitant nerve injury to both the superficial sensory branch and the PIN at this level are frequent and can be challenging to identify.

Injuries to extensor tendons of the thumb

Zone 1

Open injuries

Direct repair of tendon ends is usually possible as proximal retraction is minimal. Of greater concern is damage to the underlying capsule with exposure of the DIP joint. Vigorous lavage of the joint is required prior to capsular and tendon repair, and a single K wire is used to hold the joint in extension. Bone-anchoring devices are appropriate for reattaching EPL when division has occurred at its insertion.

Closed injuries

In the absence of a significant fracture or subluxation, acute closed injuries resulting in EPL disruption, 'mallet thumb', are treated with 8 weeks of splintage.³⁴ Indications for operative intervention extend to those cases with a large bone fragment or volar subluxation. These cases are managed using the dorsal blocking wire technique.

Zones 2 and 3

MP joint lacerations in the dorsum of the thumb can result in division in any or all of EPL, EPB and the joint capsule. Choice of suture, suture configuration and postoperative splinting are as previously described for zone 2 in the finger.

Zones 4 and 5

Injuries sustained over the first metacarpal may incur injury to EPL, APL and EPB. The cross-sectional morphology of APL and EPL allows for a core suture in a modified Kessler configuration. EPB is repaired by simple suture. Sensory branches of the radial nerve are numerous and require preservation or repair as appropriate.

Open injuries

Retraction of the proximal stump of EPL in these zones is likely. If found to be excessively damaged or delayed presentation does not allow it be pulled out to length, an extensor indicis (EIP) transfer is indicated. I prefer to use a Pulvertaft weave using a braided non-absorbable suture.

Closed injuries

Attrition ruptures of EPL are well described in patients with rheumatoid arthritis or previous plating of lower radial fractures. Inability to retropose the thumb while held in abduction is the diagnostic sign of EPL rupture. Minimal DIP joint extension may still be present, despite EPL rupture. Although a palmaris longus tendon graft can be used, an EIP tendon transfer remains my preferred solution.

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CHAPTER 53

Tendon transfers

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Introduction

Tendon transfer is the surgical transplantation of a normal muscle–tendon unit (MTU) into a new location to restore a non-functioning MTU. In 1770 a French surgeon named Missa repaired a middle finger extensor tendon injury by grafting the index and ring finger extensors, thus performing the first recorded tendon transfer.¹ However, the modern conception of tendon transfer as expressed above was formulated by Carl Nicoladoni in 1881,^{2,3} and further developed by Tomasz Drobnik with particular reference to radial nerve palsy in 1894.^{4,5} There was subsequently a dramatic expansion in the number and use of tendon transfer techniques by surgeons faced with the polio epidemics and war injuries of the early twentieth century. In this chapter we present the senior author's (SF) approach to tendon transfers for peripheral nerve injuries and tetraplegia (Figure 53.1). Some definitions are given in Box 53.1.

Indications

The essential indication for tendon transfer is the functional impairment of an MTU, either through interruption of its nerve supply, direct injury to or abnormal development of muscle. Indications for tendon transfer include:

- irreversible peripheral nerve injury,
- failed nerve transfer,
- adjunct to nerve transfer,
- traumatic loss of muscle or tendon,
- brachial plexus injury,
- spinal cord injury,
- central neurological injury including stroke, and
- congenital or developmental disorders.

In general, tendon transfers are only considered where such impairment is irreparable, as in nerve root avulsions, nerve injuries not amenable to repair, failed nerve transfers, and late

presentation of nerve injury with motor endplate fibrosis.⁶ Burkhalter has also advocated the use of early tendon transfer as an adjunct to nerve repair to retain function and reduce development of contractures during nerve recovery.^{7,8} Such a transfer serves to improve function during nerve regeneration, eliminating the need for external splinting, supplements the power of the muscles being reinnervated, and may serve as a definitive procedure where nerve regeneration is limited. However, such transfers should not be themselves associated with significant loss of function, or result in deformity following recovery of function in the affected nerve. For the majority of other cases, the senior author prefers to see neurological function plateau prior to considering tendon transfers. Based on the seminal works of Sunderland and Seddon, nerve regeneration progresses at approximately 1 mm a day.^{9,10} Depending on the location of injury, this may mean delaying surgery for up to 12–18 months.

Biomechanics

The majority of standard tendon transfer techniques are based on sound biomechanical principles. An understanding of basic biomechanics is helpful for the surgeon, particularly when faced with a difficult case where the standard approach may not be feasible. The ultimate aim of a tendon transfer is the creation of a new MTU capable of generating torque sufficient to move a target joint through a useful range of motion.¹¹ Torque is determined by the power (force-generating ability) of the muscle, and its leverage (mechanical advantage).¹² Other relevant biomechanical characteristics are the degree of tendon excursion, and the tensioning of the muscle during transfer.

Power and leverage

The power of a muscle is proportional to its physiologic cross-sectional area (PCSA).¹³ Leverage is determined by the moment arm, which is the perpendicular distance from the



Figure 53.1 Flexor digitorum superficialis (Royle, Thompson, Bunnell) opponensplasty. Source: Reproduced with permission of Donald Sammut.

Box 53.1 Definitions

- Tendon transfer: surgical transplantation of normal muscle–tendon unit (MTU) into a new location to restore function of a non-functioning MTU
- Arthrodesis: surgical immobilization of a joint by fusion of adjacent bones
- Tenodesis: surgical anchoring of a tendon to bone
- Tenodesis grip: passive grip due to finger flexor shortening with wrist extension

target joints axis of rotation to the line of pull of the muscle (Figure 53.2). It should be noted that the moment arm may vary depending on joint position, and any measurements should be taken with the joint in a useful position. Muscles whose line of pull pass further from the axis of a joint have relatively more leverage, and hence exert greater torque.¹⁴ Thus, for an MTU to be useful for a transfer, it must have sufficient power and leverage to move the target joint.

Tendon excursion

An additional consideration is the excursion of the MTU being considered for transfer. This is the distance a tendon can move from when it is relaxed to its fully contracted position. Tendon excursion and contraction velocity are proportional to muscle fibre length.¹³ In order to be a candidate for a transfer, an MTU must have sufficient excursion to move the target joint through a useful range of motion. The moment arm is

again relevant here (see above). As the moment arm increases, the amount of joint movement produced by a given amount of tendon excursion decreases. Hence, adjusting the moment arm of a transferred MTU is a tradeoff between range of movement and leverage.

Tensioning

Normal contraction of an MTU requires overlap of actin and myosin filaments in the sarcomeres of the muscle fibres. An excessively tight or loose muscle will not contract effectively, as is expressed by the Blix curve.¹⁵ Hence, it is critical for the surgeon to put the transferred MTU under appropriate tension. Previously it has been suggested that it may be preferable to place the transferred MTU under excess tension, relying on postoperative relaxation and lengthening to correct any errors.⁶ However, studies have demonstrated that this may overestimate the degree to which the muscle may remodel to accommodate its new length and result in a significant loss of muscle power.¹⁶ Ultimately, the surgeon should aim to neither over- nor under-tension the transferred MTU by setting it as close to its optimal length as possible. However, this is not easy to establish intraoperatively, as the muscle's slack length once it has been released from its insertion is not reliably related to its optimal length.¹¹ Nevertheless, an on-table estimate of the tension on a transferred tendon remains the usual method of determining the amount of tension to put on the transferred MTU. The senior author uses a comparison of distance from MTU origin to previous and new insertion as a guide.

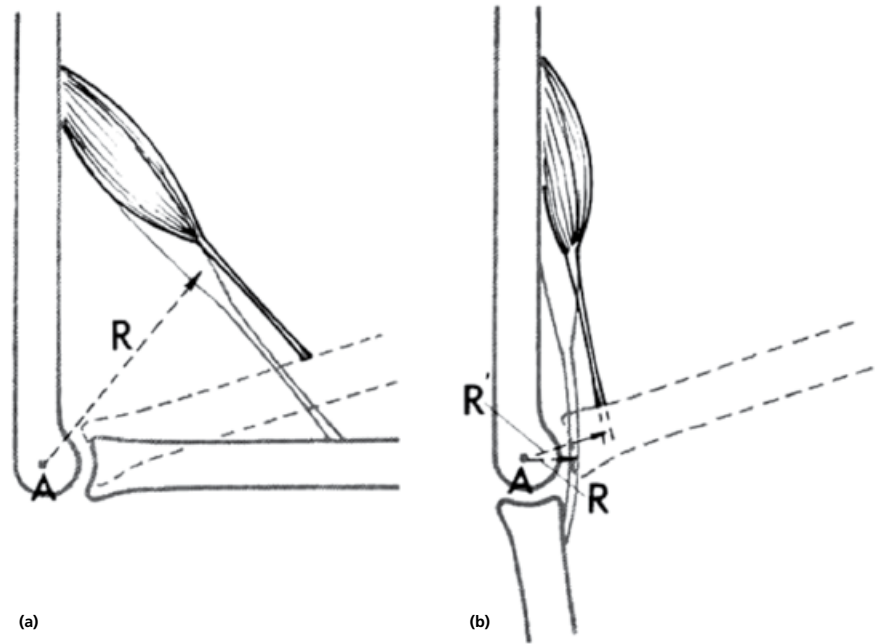


Figure 53.2 Moment arm. (a) The moment arm of a muscle whose line of pull passes distant from the axis of the joint has great mechanical advantage but acts through a relatively small range of motion. (b) By comparison, a muscle with a line of pull that passes close to the axis of the joint acts through a large range of motion but has little leverage. Source: Brand, 1974.¹⁴ Reproduced with permission of Elsevier.

Principles

General principles guiding tendon transfer have been developed and refined since the early twentieth century. They include the following:^{6,17–23}

- **Appropriate strength:** The donor MTU should be of comparable strength to the recipient, in order to provide useful movement at the target joint.
- **Adequate power:** The donor MTU should be capable of contraction against resistance, which corresponds to a grading of 4 or better on the Medical Research Council (MRC) scale.
- **Amplitude of excursion:** To be useful for tendon transfer, an MTU must have sufficient amplitude of excursion to be effective at moving the target joint.
- **Anatomic location:** The donor MTU should contract in parallel with the recipient, and be located such that its transfer results in a functionally useful line of pull. This may be optimized during the procedure by redirecting the tendon using pulleys.
- **Control:** The donor MTU must be effectively under voluntary control. Transferring a spastic muscle will result in a negative outcome.
- **Supple joints:** Preoperatively, maximum range of motion must be achieved for the joints to be moved by the transferred MTU. If possible, any existing soft tissue contractures should be dealt with during the procedure.
- **Expendability:** The benefit gained from the tendon transfer must be greater than the loss of the donor MTU in its original location.
- **Single function per transfer:** Each tendon transfer should be used to restore a single function to avoid compromising on strength and movement.

- **Suitable soft tissue bed:** The transferred MTU should not pass through scarred, inflamed or oedematous tissues that may inhibit its free movement by development of adhesions.
- **Synergism:** The patient will find it easier to learn to use an MTU whose original function is synergistic with the function it is replacing. If this is not practical, muscles that are usually activated together are also suitable for transfer, for example wrist flexors and finger extensors.

It should be borne in mind that these are not hard and fast rules that need to be followed in all cases. For example, Steindler proposed that the 'gliding apparatus' of the tendon should be preserved or reconstructed as much as possible, to minimize the risk of adhesions developing.¹⁷ Obviously, this is not possible for every patient, though if its course is through scarred, inflamed, or oedema tissues, then it may be desirable to revise the soft tissue bed using flaps or grafts. Nevertheless, these principles are useful guidelines for both assessing a patient's suitability for tendon transfer and also in selecting the best procedure.

Approach

Patients presenting with peripheral nerve palsies should have a comprehensive history taken, recording the symptoms, aetiology, time course and any investigations or procedures performed. A complete neurological examination should document the motor and sensory deficit and the limb should be further assessed for soft tissue injuries, especially for any areas of scarring, oedema or inflammation along the path of any tendon transfers. Measurements of the passive and active ranges of motion of all affected joints should be taken. Further imaging

and investigation by means of nerve conduction studies and electromyography may be performed where the diagnosis is not clear. Once the problem has been clearly defined, the reconstruction may be planned.

Surgical technique

A number of different techniques have been developed for the repair and transfer of tendons. An interlacing suture, described by Guy Pulvertaft in 1956 and henceforth known eponymously as the Pulvertaft weave,²⁴ has been proven to be superior to other techniques used historically, including the Kessler grasping suture and side-to-side repairs described by Mason and Nicoladoni.²⁵ Recently, alternatives to the Pulvertaft weave have been developed, such as the lasso²⁶ and spiral linking²⁷ repairs, which demonstrate comparable strength and may be easier and quicker to perform. The senior author's preference is to use the Pulvertaft weave (Figure 53.3).

Radial nerve palsy

Anatomy^{6,8,28-30}

The posterior cord of the brachial plexus continues as the radial nerve after having given off its terminal branches in the axilla. It comprises motor and sensory fibres from the ventral rami of C5–T1. While in the axilla it gives off its first two branches to triceps, to the long and medial heads. It crosses the lower border of teres major to enter the anterior compartment of the arm and immediately dives into the posterior compartment via the triangular interval. It then runs in the spiral groove (aka radial groove) of the humerus. While in the posterior compartment it gives its last two branches to triceps, one to the lateral head and a second to the medial head, as well as two sensory branches: the lower lateral and posterior cutaneous nerves of arm. Approximately halfway down the arm the radial nerve pierces the lateral intermuscular septum to re-enter the anterior compartment. Here it gives off a medial branch to brachialis and lateral branches to brachioradialis and extensor carpi radialis longus (ECRL). It then enters the cubital fossa under cover of brachioradialis, before dividing into deep and superficial branches. The deep branch

passes between the two heads of supinator to become the posterior interosseous nerve, while the superficial branch continues down the anterior compartment of the forearm under cover of brachioradialis. The posterior interosseous nerve immediately divides into branches to the extensor musculature of the forearm. The superficial branch of the radial nerve emerges on the radial side of brachioradialis at the wrist to cross the anatomical snuffbox and provide the sensory supply to the dorsum of the hand and the radial three and a half digits.

Clinical findings^{6,8,23}

The characteristic sign of radial nerve palsy is wrist drop. Due to the loss of the extensor musculature of the forearm, the patient is unable to extend the wrist or digits. In addition, grip strength is lost due to the weakness of finger flexion with the wrist in a flexed position. Low radial nerve lesions are defined as those below the elbow, thus sparing supply to brachioradialis and ECRL. In these cases, wrist extension is relatively preserved, though with radial deviation. Due to the proximal origin of the majority of the branches of the radial nerve, triceps function is only affected in very high lesions. The sensory deficit of radial nerve injury is less significant, only affecting the less sensitive dorsum of the hand and digits.

Approach^{6,8,23,31}

As discussed above (see Indications), some authors advocate using an early tendon transfer procedure to preserve function and prevent contractures in cases of radial nerve injury. Where this is not done, the use of a dynamic outrigger splint (Figure 53.4) results in greatly improved grip strength by fixing the wrist in extension.³² The key reconstructive aims are: wrist extension, metacarpophalangeal (MCP) joint extension, and thumb extension and abduction. These are generally performed in set combinations for ease of access and operative technique, with the main difference being the choice of MTU to be used for restoration of MCP joint extension.⁸

Wrist extension

For reconstruction of wrist extension, the pronator teres to extensor carpi radialis brevis (ECRB) transfer is ideal (Figure 53.5), and other options are rarely considered. If radial

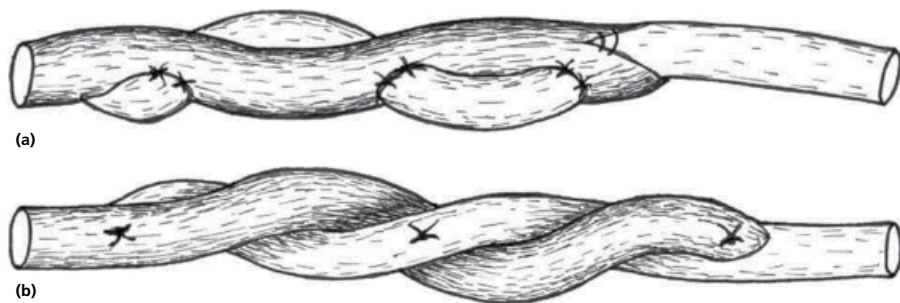


Figure 53.3 Tenorrhaphy technique. (a) Pulvertaft weave. The donor tendon is interlaced with the recipient tendon, and sutured in place. Usually three or four weaves are performed. (b) Spiral linking. The donor tendon is wound around the recipient tendon, with mattress sutures in each spiral. Again, a variable number of spirals may be performed. Source: Kulikov *et al.*, 2007.²⁷ Reproduced with permission of Sage.

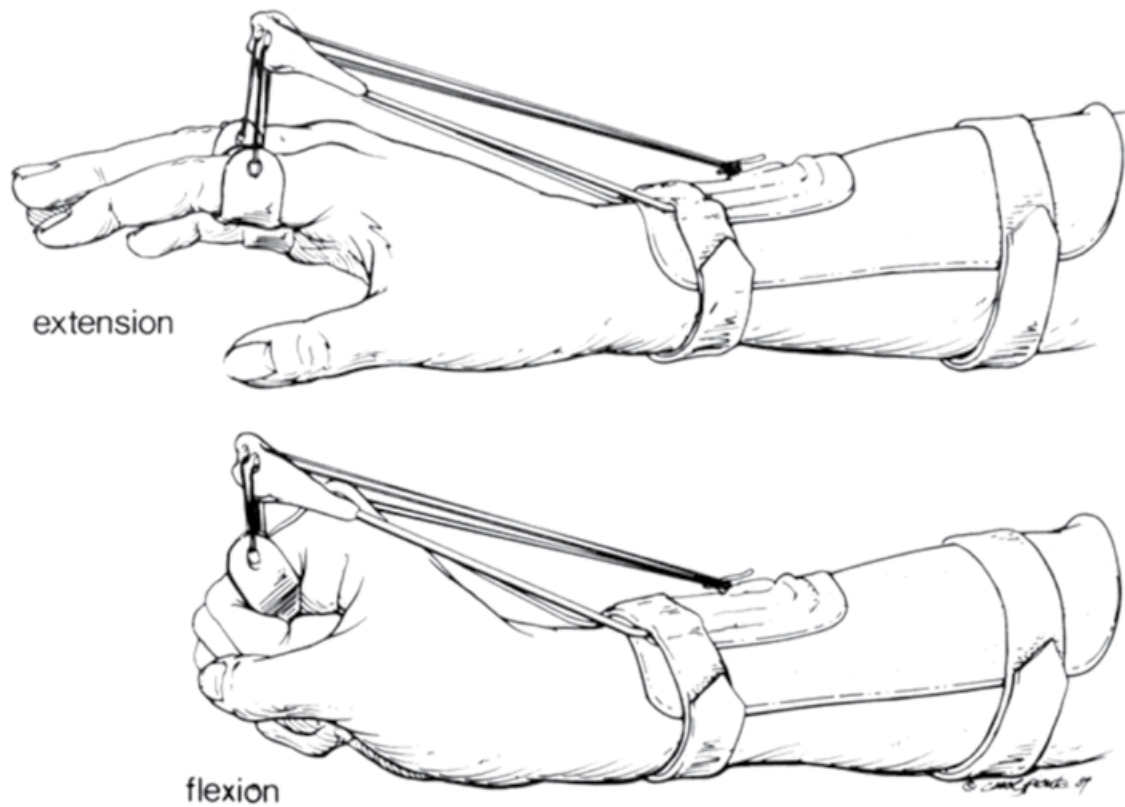


Figure 53.4 Splinting for radial nerve palsy. Use of a dynamic outrigger splint to enable finger grip by fixing the wrist in extension. This is an application of the tenodesis effect: the synergistic action of wrist extension with finger flexion. Source: Colditz, 1987.³² Reproduced with permission of Elsevier.

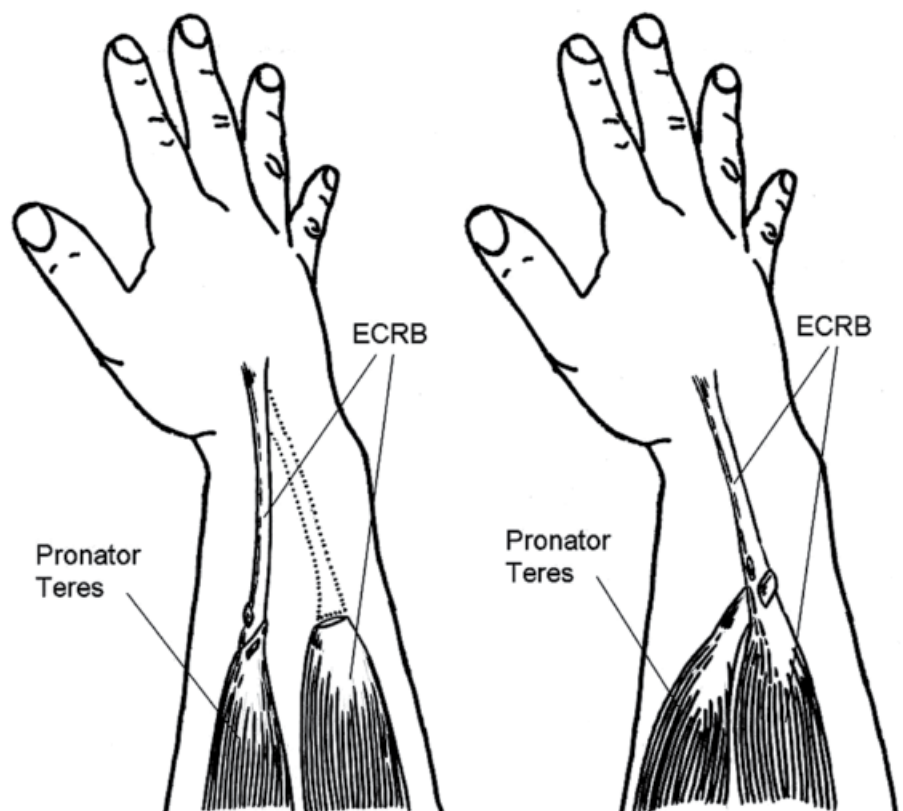


Figure 53.5 Pronator teres to extensor carpi radialis brevis transfer. Left: end-to-end. Right: end-to-side. Source: Sammer & Chung, 2009.⁶ Reproduced with permission of Lippincott Williams & Wilkins.

nerve recovery is not anticipated, this should be performed end to end to achieve a more direct line of pull. Otherwise, the transfer should be performed by an end-to-side technique, to allow ECRB to resume wrist extension once it regains function.⁶ Pronator teres should be released from its insertion with a strip of periosteum to provide a robust attachment.³³ It is tunnelled subcutaneously superficial to brachioradialis and ECRL, and inserted into the tendon of ECRB.⁸ If desired, ECRL may be synchronized to ECRB, with pronator teres redirected to the new extensor complex.

Metacarpophalangeal joint extension

To restore MCP joint extension, a tendon transfer to extensor digitorum communis (EDC) is required. The surgeon is presented with a number of alternatives for this purpose (Table 53.1). Of these, the flexor carpi radialis (FCR) transfer^{34–36} is generally preferred in low radial nerve palsy, as its loss is balanced on the radial side of the wrist by the preserved function of ECRL.⁶ By comparison, use of the flexor carpi ulnaris (FCU)²¹ transfer sacrifices the only remaining muscle acting on the ulnar side of the wrist, resulting in radial deviation. However, both of these transfers suffer from the major drawback of having relatively low excursion for their intended function. For this reason, the flexor digitorum superficialis (FDS) transfer^{37,38} is sometimes preferred, as it has significantly greater excursion, allowing full extension of the digits without requiring wrist flexion. Whichever donor is used, they may be attached to EDC either by leaving it in continuity, or by dividing the tendons of EDC to allow end-to-end attachment of the transferred tendon (Figure 53.6). If the tendons are divided, they may be passed superficial the extensor retinaculum to optimize the line of pull.

Thumb extension

Extensor pollicis longus (EPL) is the common target for tendon transfers to restore thumb extension. It is divided at its musculotendinous junction and transposed to the radial aspect of Lister's tubercle. Palmaris longus is divided at the wrist and

freed up proximally, passed over the radial side of the wrist, then inserted into the distal stump of EPL. The line of pull of the transferred tendon should cross the anatomical snuffbox, resulting in combined extension and abduction with muscle contraction (Figure 53.7). An alternative is to use the ring finger FDS tendon to power palmaris longus. This is usually done during a Boyes transfer, when part of FDS is also being used for MCP joint extension.

Median nerve palsy

Anatomy^{30,39–42}

The median nerve is formed by a root from each of the medial and lateral cords of the brachial plexus. The larger lateral root carries the contribution from C5, C6 and C7, and the smaller medial root carries the contribution from the C8 and T1. It thus receives motor and sensory fibres from the ventral rami of all the segments contributing to the brachial plexus. It initially lies anterolateral to the third part of the axillary artery, but after crossing the lower border of teres major to enter the anterior compartment of the arm it crosses in front of the artery to lie on its medial side. The median nerve lies medially in the cubital fossa, entering the forearm by passing between the two heads of pronator teres. The nerve then runs under the fibrous arch of the origin of FDS, continuing down the forearm bound to the deep surface of FDS by areolar tissue. Within the forearm it gives off the anterior interosseous nerve, which supplies the deep layer of forearm flexor musculature. Injuries above where the anterior interosseous nerve is given off are usually defined as high median nerve lesions. Proximal to the wrist the median nerve gives off its palmar cutaneous branch, and then enters the carpal tunnel on the radial side of the tendons of FDS. The recurrent motor branch supplies the thenar muscles, and the remainder of the nerve provides the palmar digital nerves for the radial three and a half digits, and also supplies the radial two lumbricals. Note that there is some variation in the supply of the thenar muscles (see below, under Ulnar nerve palsy). There is also frequently variation in the pattern of innervation due to median to ulnar connections in the forearm, such as the Martin–Gruber anastomosis.⁴³ A median to ulnar connection, or Marinacci anastomosis,⁴⁴ is less common. It is important for the surgeon to recognize that the presence of these connections may result in unusual presentations of peripheral nerve injury.⁴⁵

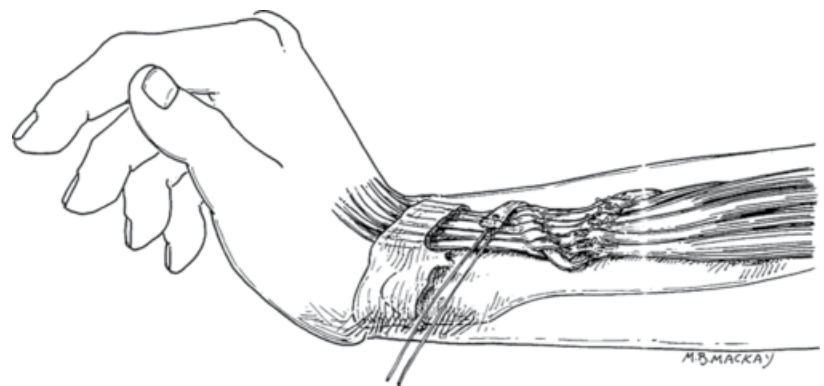
Clinical findings^{23,41,42}

Injury to the median nerve below where the anterior interosseous nerve is given off results in loss of thumb flexion and opposition, with preserved forearm flexor function. In high median nerve palsy this is also lost, with the exception of some wrist and finger flexion provided by FCU and the bellies of flexor digitorum profundus (FDP) supplied by the ulnar nerve. Sensation is lost over the palmar surface of

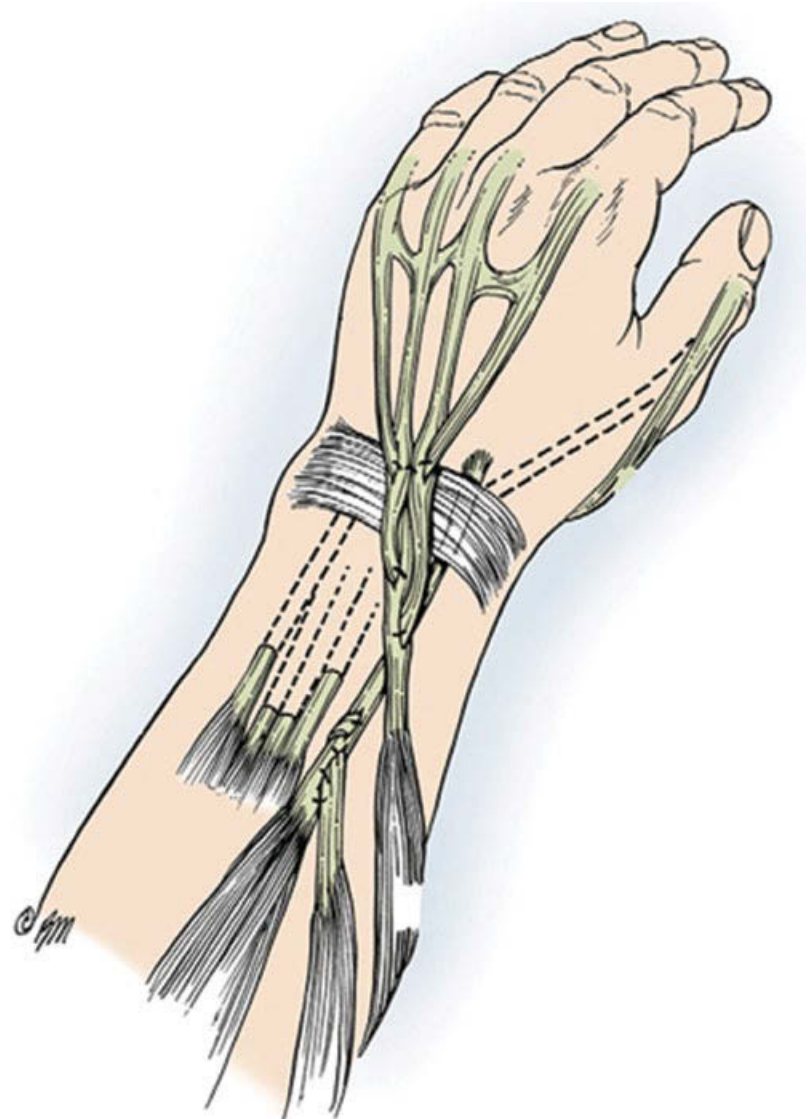
Table 53.1 Approach to radial nerve palsy

Reconstructive aim	Preferred options	Alternatives
Wrist extension	Pronator teres to ECRB	Rarely consider brachioradialis (if function preserved) or FCR
Metacarpophalangeal joint extension	FCR to EDC ^{34–36}	FDS to EDC ^{37,38} FCU to EDC (Jones ²¹)
Thumb extension and abduction	Palmaris longus to EPL	Ring finger FDS to EPL

ECRB, extensor carpi radialis brevis; FCR, flexor carpi radialis; EDC, extensor digitorum communis; FCU, flexor carpi ulnaris; EPL, extensor pollicis longus.



(a)



(b)

Figure 53.6 Flexor carpi radialis to extensor digitorum communis (EDC) transfer. (a) EDC left in continuity. Source: Richards, 2003.³¹ Reproduced with permission of Elsevier. (b) EDC divided. Source: Ingari & Green, 2010.⁸ Reproduced with permission of Elsevier.

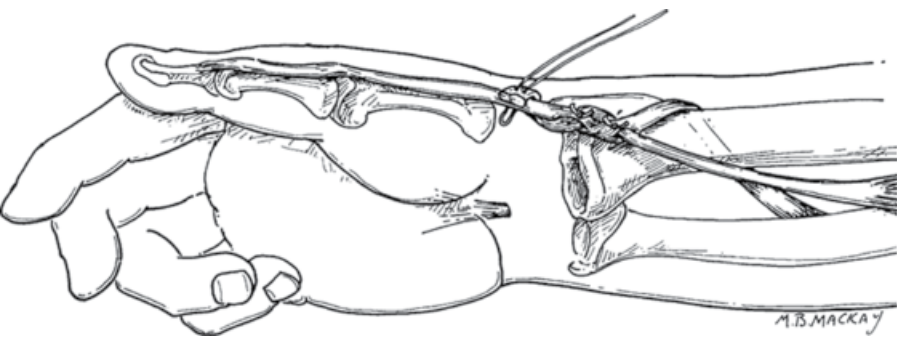


Figure 53.7 Palmaris longus to extensor pollicis longus transfer. Source: Richards, 2003.³¹ Reproduced with permission of Elsevier.

the hand and radial three and a half digits. This is has great functional significance, as sensation in this distribution is essential for precision movement. Palmar sensation is preserved in compression or injury of the median nerve in the carpal tunnel.

Approach^{23,31,41,42}

The key reconstructive aims are: thumb opposition, thumb interphalangeal (IP) joint flexion, and index distal interphalangeal (DIP) joint flexion (Table 53.2). However, it should be noted that even with restoration of thumb opposition, many patients’ hand function remains poor due to the debilitating sensory deficit.⁴² For this reason, opponensplasty may not be appropriate for every patient.

Thumb opposition (opponensplasty)

The usual target of opponensplasty procedures is the tendinous insertion of abductor pollicis brevis (APB) at the proximal phalanx of the thumb. Bunnell stated that any tendon transfer intended to restore thumb opposition should be passed ‘through a tendon pulley constructed at the pisiform bone or by passing it around the tendon of the flexor carpi ulnaris’, and be ‘inserted into the ‘dorso-ulnar aspect of the base of the proximal phalanx of the thumb to give pronation.’⁴⁶ Any donor MTU that satisfies these requirements may be used for opponensplasty. The extensor indicis proprius (EIP)⁴⁷ opponensplasty (Figure 53.8) provides effective thumb opposition with the minimal functional sacrifice. It is also effective even in high median nerve lesions where other candidates for transfer are paralysed. The EIP tendon is identified on the ulnar aspect of the index finger EDC tendon and is divided immediately proximal to the extensor expansion. Alternatively, if further length is desired it may be harvested with a length of extensor expansion (see Figure 53.12), though this is usually unnecessary for opponensplasty. The tendon should be freed from its attachments in the distal forearm and passed around the ulnar aspect of the forearm to the tendinous insertion of APB at the proximal phalanx of the thumb.

Alternatives to the EIP opponensplasty include the palmaris longus,⁴⁸ superficialis^{46,49,50} and abductor digiti minimi

Table 53.2 Approach to median nerve palsy

Reconstructive aim	Preferred options	Alternatives
Thumb opposition	EIP opponensplasty ⁴⁷ ADM opponensplasty ⁵¹	FDS opponensplasty ⁴⁶ Palmaris longus opponensplasty ⁴⁸
Thumb IP joint flexion	Brachioradialis to FPL	ECRL to FPL ECU to FPL
Index DIP joint flexion	FDP side to side (lose independent flexion of fingers)	ECRL to FDP

EIP, extensor indicis proprius; ADM, abductor digiti minimi; FDS, flexor digitorum superficialis; IP, interphalangeal; FPL, flexor pollicis longus; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; DIP, distal interphalangeal; FDP, flexor digitorum profundus.

(ADM)⁵¹ opponensplasties. The palmaris longus opponensplasty may incorporate palmar fascia in continuity with the tendon or the contralateral palmaris longus as a free tendon graft in order to gain sufficient length to reach the APB insertion. It is tunnelled subcutaneously from distal forearm to the thumb. This method is best for a low median nerve lesion with an intact palmaris longus. One drawback is its line of pull, which results in less effective opposition than that offered by other options.⁴² This may be partially overcome by passing the tendon via a pulley created at the pisiform as suggested by Bunnell.⁵²

The FDS opponensplasty utilizes the tendon to the ring finger (Figure 53.9). The line of pull may be optimized by bringing the tendon through a window in the flexor retinaculum as described by Snow.⁵³ This is also more secure than simply diverting the tendon at the distal end of the carpal tunnel. The tendon is divided via a distal incision in the finger or palm, and transferred across the palm to the tendinous insertion of APB. One shortcoming of this method is the significant rate of complications including deformity and extensor lag of the donor digit.⁴² Harvesting the FDS via a palmar rather than digital incision may reduce this risk.⁵⁴

The ADM opponensplasty (Figure 53.10) offers the further benefit of restoring the appearance of bulk to the thenar

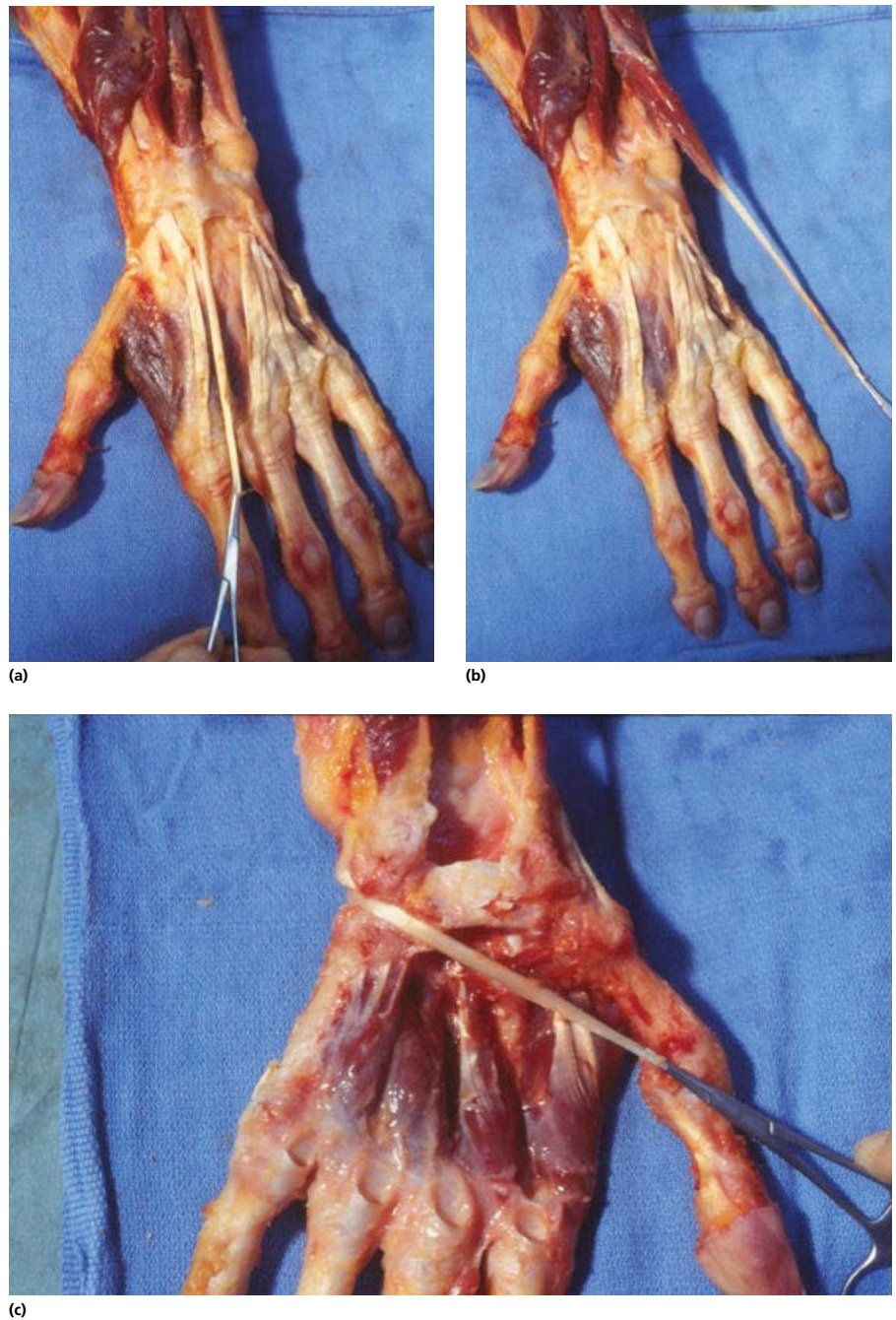


Figure 53.8 Extensor indicis proprius (EIP) (Burkhalter) opponensplasty. (a) EIP identified on the ulnar aspect of the index finger extensor digitorum communis (EDC) tendon. (b) EIP divided immediately proximal to the extensor expansion and freed from attachments in distal forearm. (c) EIP passed around ulnar aspect of wrist to the tendinous insertion of abductor pollicis brevis at the proximal phalanx of the thumb. Source: Reproduced with permission of Donald Sammut.

eminence. It is released from its insertion and transferred across the palm to APB. This may be complicated by compression of the ulnar nerve by oedema or scar where it is crossed by the transferred tendon. To overcome this, Littler and Cooley,⁵⁵ Ogino *et al.*⁵⁶ and more recently Cawrse and Sammut⁵⁷ have suggested modifications that may reduce this risk, either by partial or complete detachment of ADM from its proximal origin.

Thumb IP and index DIP joint flexion

In high median nerve injury, thumb IP and index DIP joint flexion may require restoration. For thumb IP joint flexion, ECRL or extensor carpi ulnaris (ECU) may be transferred to (flexor pollicis longus (FPL). For index finger DIP joint flexion, ECRL may be transferred to FDP, or a side-to-side synchronization of FDP may be performed, relying on the ulnar-innervated component of the muscle.



(a)



(b)



(c)

Figure 53.9 Flexor digitorum superficialis (FDS) (Royle, Thompson, Bunnell) opponensplasty. (a) The FDS tendon to the ring finger being brought out through a window in the flexor retinaculum. (b) The tendon is divided through a distal incision in either the finger or palm. (c) The tendon is tunneled subcutaneously and attached to the tendinous insertion of APB via an incision on the radial aspect of the thumb. Source: Reproduced with permission of Donald Sammut.

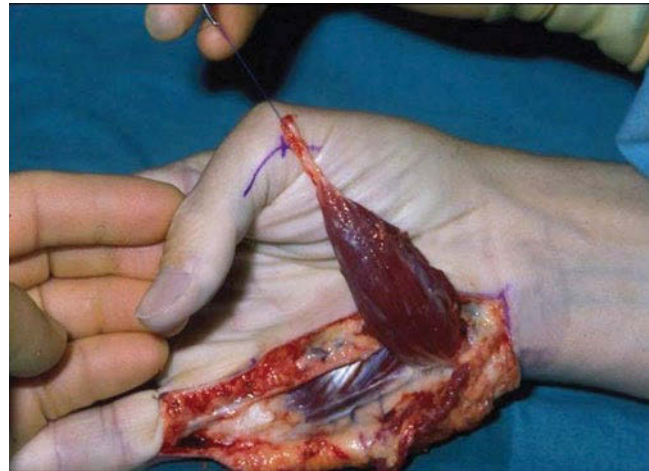


Figure 53.10 Abductor digiti minimi (ADM) (Huber) opponensplasty. If desired, flexor digiti minimi (FDM) may be transferred together with ADM for greater strength, as shown. Source: Reproduced with permission of Donald Sammut.

Ulnar nerve palsy

Anatomy^{30,41,42,58,59}

The ulnar nerve commences as the continuation of the medial cord of the brachial plexus in the axilla, comprising fibres from the ventral rami of spinal segments C8 and T1. It also usually receives a contribution composed of C7 fibres from either the lateral cord of the brachial plexus or the median nerve in the axilla; these are destined for FCU in the forearm. The nerve lies on the medial side of the axillary artery, and enters the posterior compartment of the arm by piercing the medial intermuscular septum. It then passes between the medial epicondyle and the olecranon, through the cubital tunnel, and enters the anterior compartment of the forearm by passing between the two heads of FCU. It passes down the forearm beneath FCU and superficial to FDP, giving branches to those muscles, and accompanied by the ulnar artery. High ulnar nerve injuries are those that occur above where these branches originate. Just proximal to the wrist, the nerve emerges and gives off volar and dorsal sensory branches. At the wrist, they pass through the ulnar (Guyon's) canal, superficial to the flexor retinaculum, to enter the hand. It then divides into superficial and deep branches. The superficial branch provides the palmar digital nerves for the little finger and the ulnar aspect of the ring finger, and supplies the palmaris brevis muscle when present. The deep branch innervates the the intrinsic muscles of the hand with the exception of the thenar muscles and two of the lumbricals. This pattern is variable, however, with the supply to the lumbricals shared unevenly between median and ulnar nerves in some cases. Frequently opponens pollicis and FPB may also receive a branch, receiving either a sole supply from the ulnar or dual innervation from both ulnar and median nerves.

Clinical findings^{23,41,42}

The claw or intrinsic minus hand is virtually pathognomonic of ulnar nerve injury. The loss of the intrinsic muscles of the hand

and unopposed pull of the long extrinsic tendons result in hyperextension of the MCP joints and flexion at PIP and DIP joints. Specific testing may elicit Froment's sign: flexion of the thumb IP joint when holding a piece of paper between thumb and index finger, indicating weakness of adductor pollicis and the first dorsal interosseous. High ulnar nerve lesions are above the origin of the branches to FCU and FDP, resulting in reduced grip strength and radial deviation of the hand. An ulnar nerve palsy also results in loss of sensation over the ulnar one and a half digits.

Approach^{23,31,41,42}

The key reconstructive aims are: restoration of key grip by thumb adduction and correction of clawing. It is usually not necessary to reconstruct index finger abduction to achieve a useful key grip, as the patient is able to stabilize the index finger against adjacent digits (Table 53.3).

Thumb adduction (adductorplasty)

The common target for restoration of thumb adduction is, unsurprisingly, adductor pollicis. Again, a variety of donor MTUs may be used. Common choices include the ring or middle finger FDS,⁶⁰ the ECRB,^{61,62} or EIP.⁶³ In the case of ECRB and EIP, additional length may be required in order to reach adductor pollicis. For EIP this may be achieved by harvesting a length of the central tendon of the extensor expansion in continuity with the tendon (Figure 53.11), but ECRB requires a free tendon graft. Whichever donor is used, in order to recreate the line of pull of adductor pollicis, a pulley is required. This may most easily be achieved by passing the tendon around the second or third metacarpal (Figure 53.12).

Correction of clawing

The essential problem in the intrinsic minus digit is the hyperextension instability of the MCP joint, with flexion of the PIP and DIP joints (Figure 53.13). Procedures for correction of clawing thus focus on correction of this deformity.

Stiles and Forrester-Brown⁶⁴ first described transferring a single slip of each FDS tendon to EDC to correct clawing. Their technique was modified by Bunnell,⁶⁵ who transferred each slip onto the lateral bands of the extensor expansion.

However, this frequently resulted in complications due to adhesion formation and overcorrection.⁴² Bunnell's procedure was further modified by Littler,⁶⁶ whose technique is now widely known as the modified Stiles–Bunnell procedure. The FDS tendon of the middle or ring finger is divided over the PIP joint and split into four slips. Each slip is then passed through the lumbrical canals and inserted into the radial lateral band only. There have subsequently been many further modifications to the technique, though the essential elements – the division of a single FDS tendon into four and the attachment of the resulting slips into the individual digits – remain the same. The senior author's preferred method is that described by Riordan⁶⁷ and Zancolli,⁶⁸ in which the A1 pulley is used as the insertion of the slips of FDS (Figure 53.14). Alternatives to FDS include wrist flexors and extensors as described by Brand,⁶⁹ though these require free tendon grafts of sufficient length to reach their targets.

Combined palsy

Patients presenting with combined peripheral nerve palsies should have their individual problems and severity carefully characterized. This requires a detailed examination and further investigation with electromyography and nerve conduction studies with an aim to establishing both remaining function as well as deficiencies. Once this is done, a tailored approach to the individual patient may be planned. Innervated free muscle transfer may be considered for difficult situations where there are no suitable donor tendons, or where a standard transfer would result in unacceptable loss of function.

Tetraplegia

Restoration of upper limb function is a priority for tetraplegic patients, comparable to or of higher significance than bladder, bowel, sexual and lower extremity function.^{70–72} It allows the tetraplegic patient some autonomy, permitting completion of some personal activities of daily living (ADLs) independently or with the aid of appliances. Surgical restoration of upper limb function by means of tendon transfers originated with the work of Bunnell in the mid-twentieth century.⁷³ Significant advances in the techniques used were made by Moberg, who abandoned the prior focus on providing the patient with a tip-to-tip tripod pinch grip in favour of a lateral pinch or key grip between the thumb and index finger.⁷⁴ This gave the patient a functional grip without the stiff hand resulting from the multiple tenodeses required to achieve a thumb fixed in opposition.⁷⁵ Even more recently, single-stage reconstructions aimed at reducing hospital stay and improving patient outcomes have been described. The approach to the surgical reconstruction of upper limb function in tetraplegia used at a major spinal unit in Australia is described in this section (Box 53.2).

Table 53.3 Approach to ulnar nerve palsy

Reconstructive aim	Preferred options	Alternatives
Thumb adduction	EIP adductorplasty ⁶³ FDS adductorplasty ⁶⁰	ECRB adductorplasty ^{61,62}
Correction of clawing	FDS of middle or ring finger to A1 pulley of affected digit (modified Stiles–Bunnell)	FCR or ECRB to radial lateral bands of ring and little fingers ⁶⁹

EIP, extensor indicis proprius; FDS, flexor digitorum superficialis; ECRB, extensor carpi radialis brevis; FCR, flexor carpi radialis.

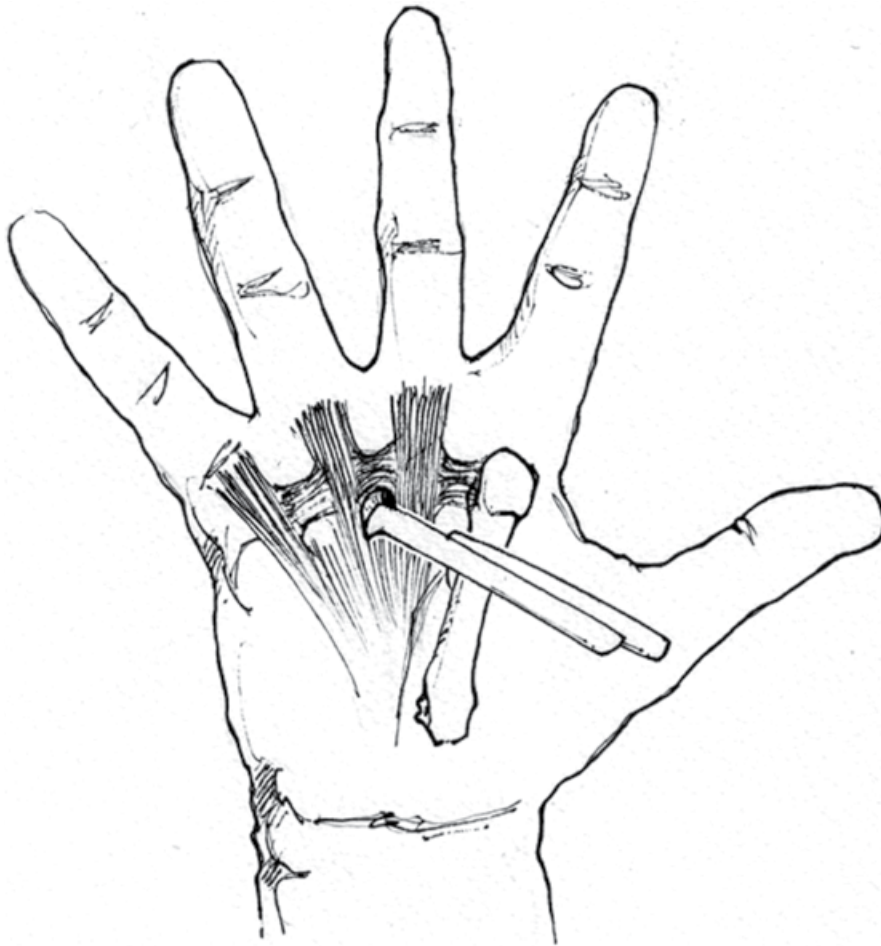


Figure 53.11 Adductorplasty. Both flexor digitorum superficialis (FDS) and extensor indicis proprius (EIP) tendons are shown. It should be noted that when using EIP it is desirable to hook the tendon around the second metacarpal, as shown. Source: Reproduced with permission of Donald Sammut.

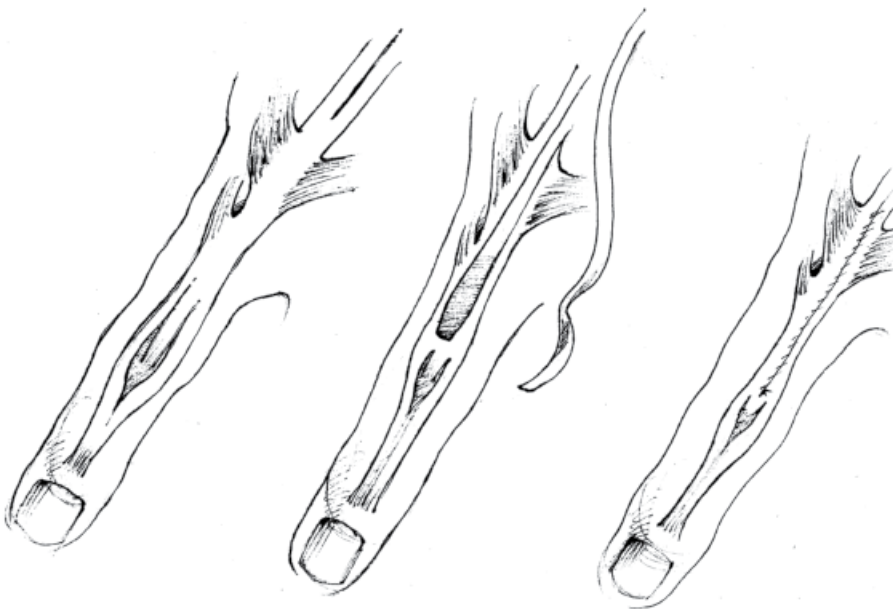


Figure 53.12 Extension of extensor indicis proprius (EIP) length for adductorplasty. Left: Appearance of extensor tendon insertions into the dorsum of the left index finger, note the EIP tendon on the ulnar aspect of the extensor digitorum communis (EDC) tendon to the index finger. Middle: EIP tendon released from insertion, including part of the central tendon of the extensor expansion, redirected to target adductor pollicis. Right: Lateral slips sutured to central tendon of extensor expansion. Source: Reproduced with permission of Donald Sammut.

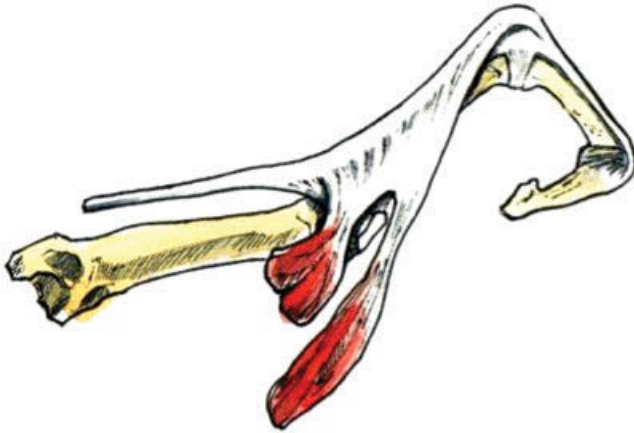


Figure 53.13 The intrinsic minus digit. Unopposed action of the long extrinsic tendons and weakness of the intrinsic muscles results in instability and hyperextension of the metacarpophalangeal joint, with flexion of PIP and DIP joints. Source: Reproduced with permission of Donald Sammut.

Assessment

Careful assessment is of key importance. Neurological stability should be ascertained, and the functional level clearly established. Current functional capabilities should also be reviewed, including bed mobility and transfers, and use of occupational aids or appliances including wheelchairs. Any psychological issues should be addressed, and the patient's motivation and expectations assessed. Patient must be highly motivated and involved in their treatment and postoperative rehabilitation to allow recruitment of transferred muscles and any previous issues with compliance should be examined in detail. Unrealistically high expectations regarding the level of function attainable post procedure should be gently corrected. A general medical history should be taken, as significant ongoing medical problems are a relative contraindication.

Contraindications to surgical reconstruction of upper limb function in tetraplegia include:

- unavailability of suitable muscles for transfer;
- severe pressure sores/generally unwell;
- severe muscle spasticity or joint contractures; and
- inadequate patient preparation or motivation.

Examination

Careful examination and recording of upper limb function are important in the preoperative period, both for patient selection purposes and to compare pre- and postoperative function. Residual muscle function is assessed and the patient given a categorization for each upper limb according to the international classification (see below). Potential donor muscle groups should be assessed for spasticity and range of motion of elbow, wrist, and hand joints measured.⁷⁶ Standard preoperative examination should be undertaken to screen for any medical issues which may be problematic in the perioperative period.

Classification

Moberg recognized that using a patient's spinal level of injury may not accurately reflect the degree of motor impairment and residual function. He devised a classification system for use in planning surgical restoration of upper limb function based on which muscles remained with strength of grade 4 or greater according to the familiar Medical Research Council scale, that is a muscle of either full strength or whose strength is reduced but may still move the joint against resistance.⁷⁴ This classification system was adopted and improved at the first International Conference on Tetraplegia in Edinburgh in 1978, the result of which is International Classification for Surgery of the Hand in Tetraplegia (Table 53.4), the Giens revision of which is still in widespread use. The patient receives a separate classification for each arm, and sensory function is recorded together with the sensitivity group as either O (ocular input only), or Cu (intact tactile function, defined as 10 mm or better two-point discrimination at the thumb). This system is useful to provide a quick summary of a patient's residual function in order to help plan surgery. In general, relatively high lesions that result in absent triceps and intact biceps and brachioradialis with one or both of ECRL and ECRB fit into groups 1 to 3. These patients will require reconstruction of elbow extension in addition to forearm reconstruction. Mid-level lesions with intact triceps function with or without pronator teres and FCR occupy groups 3–5, and these groups generally have a good outcome from tendon transfers. Elbow extension is preserved, and the surgeon may proceed directly to forearm reconstruction. Pronator teres function also allows supination/pronation, which is otherwise difficult to restore. The remaining groups (6–9) are only lacking function in the intrinsic muscles of the hand, with or without the long extensors of the fingers and thumb. There are many surgical options for these patients, who may even be treated as for an ulnar nerve palsy.

Preoperative

Preparing the tetraplegic patient for tendon transfers to restore upper limb function should begin as early as possible. Splinting of joints in a useful position to prevent problematic contractures should commence at an early stage. The patient and members of their social support network, including spouse, family and carers, should be educated regarding the operation and rehabilitation. Allied health reviews regarding these topics should be performed, and any appliances required for the postoperative period ordered, including electric wheelchairs. If there are any outstanding medical issues these should be addressed and optimized in the preoperative period. The procedures for a given patient should be individualized according to their residual function and goals. Use of a 'one size fits all' approach to tendon transfers in tetraplegics is a recipe for disaster. As a general rule the less functional upper limb is operated on first.

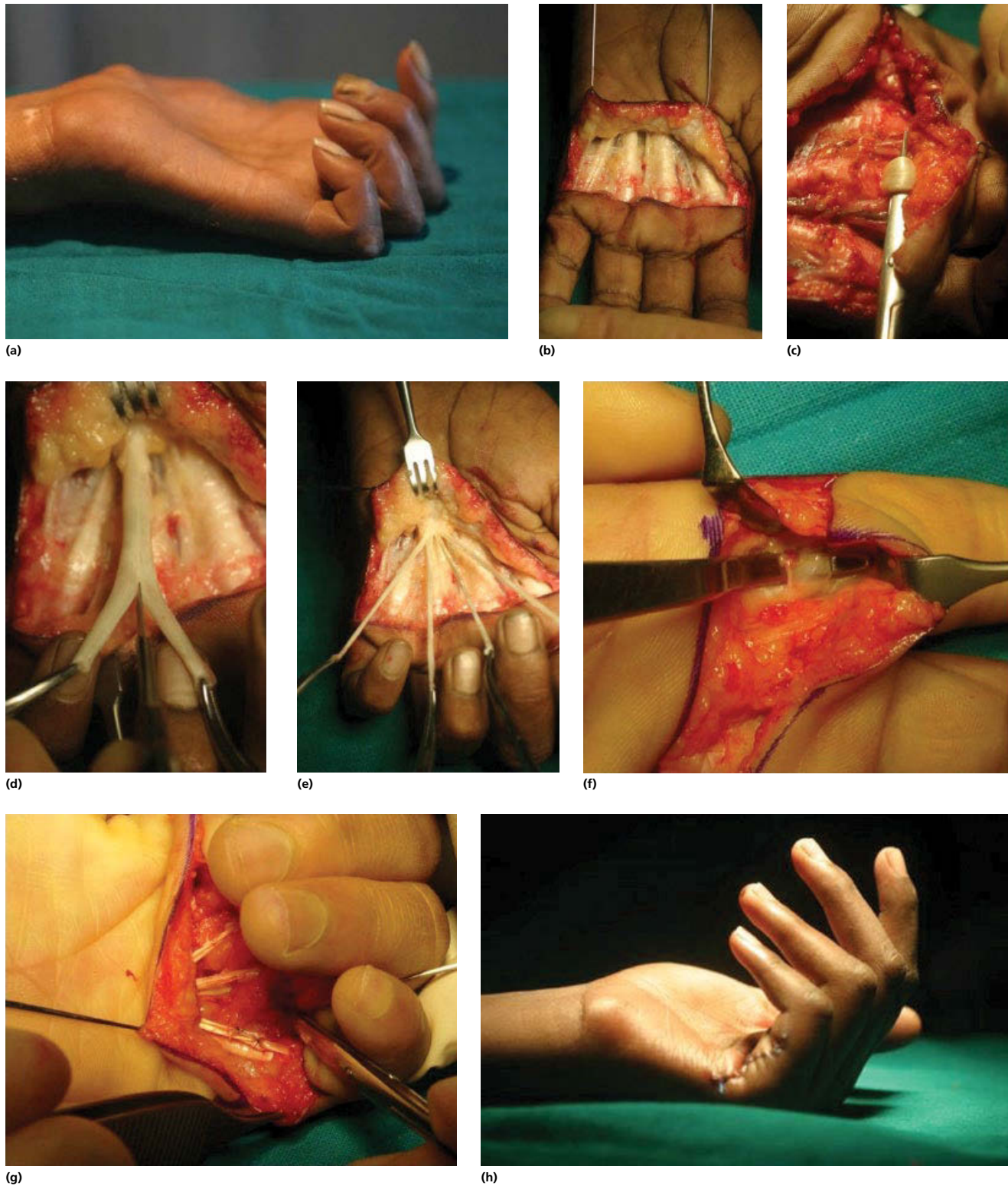


Figure 53.14 Correction of clawing using four-tail division of flexor digitorum superficialis (FDS) and weave into A1 pulley. (a) Preoperative appearance. (b) Skin incision at the distal palmar crease. (c) Exposure of FDS tendon to ring finger. (d) The tendon is divided over the PIP joint, and incised longitudinally. (e) Division of the tendon into four slips, one for each finger. (f) The A1 pulley is identified over the flexor aspect of the PIP joint. (g) The individual slips are inserted into the A1 pulley. (h) Postoperative appearance. Source: Reproduced with permission of Donald Sammut.

Box 53.2 Further principles of tendon transfer in tetraplegic patients**Level of injury**

The commonest levels of cervical spinal cord injury are between the C4 and C7 vertebral levels.⁷⁵ In general, injuries above the fifth cervical cord segment are not amenable to surgical restoration of upper limb function by means of tendon transfers due to lack of transferable muscles with useful function. Assessment of residual neurological function forms a crucial part of the workup and is considered in greater detail in *Assessment*, below.

Timing of surgery

Patients must be neurologically stable in order to determine their level of impairment. Furthermore, the postinjury period is fraught for patients managing difficult psychological and social changes, and significant motivation required to achieve a good outcome from tendon transfers. However, the appropriate amount of time to wait post injury before embarking on surgical restoration of upper limb function will differ considerably between patients. As such, firm cut-offs are misleading, and may under- or overestimate the amount of time required for an individual. In general, however, a minimum of 6–12 months is required for the patient to reach neurological stability following injury as prior to this significant gains may still be made with rehabilitation alone.

Multidisciplinary team

High-quality nursing care and input from allied health staff in preparation for surgical reconstruction of upper limb function and during the postoperative phase are crucial for a good outcome. This helps prevent postoperative complications and streamlines rehabilitation and retraining of muscle groups in their new function. As such, the ideal environment for patients undergoing this complex procedure is within an established programme with a multidisciplinary team, comprising reconstructive surgeons experienced in tendon transfers, rehabilitation medicine physicians, hand therapists, physiotherapists and specially trained nurses.

Goals

The goal of surgical restoration of upper limb function in tetraplegia is the improvement in quality of life and enabling independent completion of ADLs. The means by which this is achieved is by restoration of elbow and wrist extension by means of tendon transfers and augmentation of the passive tenodesis effect to enable hand grip by means of tenodeses and arthrodeses.^{74,79}

Elbow extension

Restoration of triceps function allows improvements in bed mobility, transfers and stability, wheelchair skills, operating reach, ability to push off bedclothes, social contact, and enables further tendon transfers for reconstruction of forearm function. There are two established options for donor muscles: posterior deltoid and biceps.

The posterior deltoid to triceps transfer for restoration of elbow extension in the tetraplegic patient is a procedure pioneered by Moberg in the 1970s.⁷⁴ This takes advantage of the mechanically advantageous position of the deltoid muscle and its separate innervation by the axillary nerve (Figure 53.15). Initially tendons harvested from the toe extensors were used to extend the donor muscle to the triceps insertion, but more recently the Y-shaped Dacron graft designed by Jacques Tessier has proven an effective

Table 53.4 International Classification for Surgery of the Hand in Tetraplegia (Giens classification 1984, adapted from Edinburgh classification 1978)

Sensitivity group	Motor characteristics	Description of function
0	No muscle below elbow suitable for transfer	Flexion and supination of elbow
1	Brachioradialis	
2	Extensor carpi radialis longus	Extension of wrist (weak or strong)
3	Extensor carpi radialis brevis	Extension of wrist
4	Pronator teres	Extension of wrist and pronation of forearm
5	Flexor carpi radialis	Flexion of wrist
6	Finger extensors	Extrinsic extension of fingers (partial or complete)
7	Thumb extensors	Extrinsic extension of thumb
8	Partial digital flexors	Extrinsic flexion of fingers (weak)
9	Lacks only intrinsics	Extrinsic flexion of fingers
X	Exceptions	

Source: Zancolli *et al.*, 2002.⁷³ Reproduced with permission of Elsevier.

alternative.⁷⁷ The skin incision is made over the midaxial line of the arm, with care taken not to injure the upper lateral cutaneous nerve of arm. In a plane superficial to the deep fascia over deltoid, the muscle is dissected out and the posterior third mobilized. The Tessier graft is secured to posterior deltoid proximally and to the tritendinous triceps insertion distally.

Biceps brachii to triceps transfer has been used to restore elbow extension in tetraplegia since the 1950s.⁷⁸ It is generally less preferred than posterior deltoid transfer due to the greater loss of elbow flexion power, increased difficulty in rehabilitation and retraining, and the risk of radial nerve injury during biceps transfer.^{77,79} However, some research has suggested that biceps transfer allows stronger elbow extension, resulting in antigravity movement in a greater proportion than in posterior deltoid transfers.⁸⁰ It should be noted that an intact and functional brachialis is required for a biceps transfer to be considered as an option. The approach is made through a transverse incision in the cubital fossa. The biceps tendon is released from its insertion into the radial tuberosity, with care taken not to injure the lateral or medial cutaneous nerves of forearm. A second incision is made on the medial side of the arm and the medial intermuscular septum divided to allow the biceps tendon to be passed posteriorly. A further incision is made over the olecranon, through which the biceps tendon is fixed to the tendinous insertion of triceps and the olecranon using sutures with or without a tenodesis screw.

Forearm reconstruction

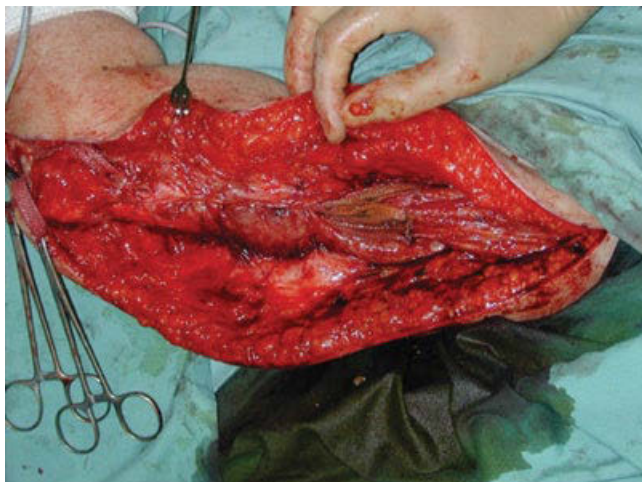
With intact elbow extension, forearm reconstruction may be attempted. Through a combination of tendon transfers and tenodeses it is possible to achieve wrist extension, finger grasp



(a)



(b)



(c)

Figure 53.15 Posterior deltoid to triceps transfer. (a) Preoperative markings. (b) Tessier Dacron graft secured to posterior deltoid proximally. (c) Graft secured to triceps insertion distally.

and lateral key pinch between thumb and index finger. Finger grasp is achieved by augmenting the tenodesis grip associated with wrist extension. The Alphabet procedure was devised by Fridén *et al.* as a combination of procedures consisting of seven individual operations done as a single-stage reconstruction for hand function tetraplegics. It has been shown to be effective in providing hand function in patients with C6 tetraplegia.⁸¹ It comprises a number of procedures which may be performed together or separately (Box 53.3). The split FPL to EPL tenodesis (also known as New Zealand tenodesis⁸²) is designed to stabilize the IP joint of the thumb. Free grafting of the ring finger FDS tendon to the extensor hood by looping it under the EDC tendons results in the fingers assuming an intrinsic plus position. Thumb CMC joint arthrodesis fixes the thumb in a position useful for a lateral key grip. One alternative used by the authors is that of a silicone interpositional spacer (Swanson implant) in the first webspace between the first and second metacarpals (Figure 53.16). Brachioradialis to FPL and ECRL to FDP tendon transfers are to restore thumb and finger flexion

Box 53.3 Alphabet procedure (Advanced Balanced Combined Digital Extensor Flexor Grip reconstruction)

- 1 Split FPL to EPL tenodesis
 - 2 Free tendon graft (FDS 4) to extensor hood digits 2–3 and 4–5
 - 3 Thumb CMC joint arthrodesis^a
 - 4 BR to FPL tendon transfer
 - 5 ECRL to FDP tendon transfer
 - 6 EPL to dorsal forearm fascia tenodesis
 - 7 ECU to ulnar head tenodesis
- From Fridén *et al.*, 2011.⁸¹

^aAlternatively, a synthetic spacer in the first webspace may be used (see Figure).

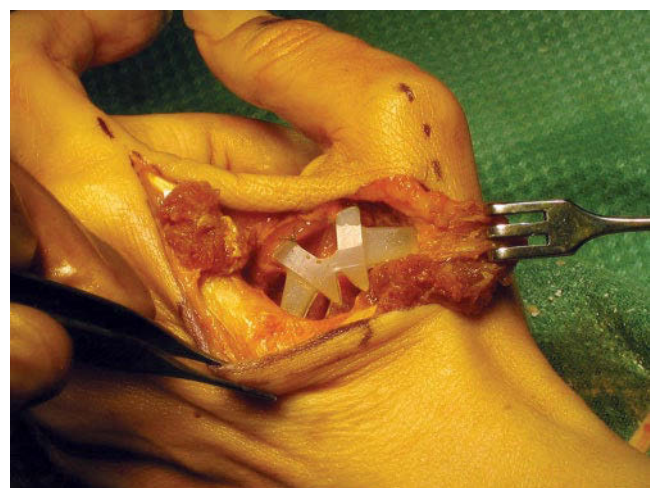


Figure 53.16 Swanson spacer interposed between first and second metacarpals.

respectively. EPL is rerouted to the dorsal forearm fascia to passively extend the thumb during wrist flexion, and finally ECU is shortened and attached to the ulnar head to stabilize the wrist.

Postoperative care

Patients who have undergone tendon transfer procedures, particularly those who are tetraplegic or immobilized, require high-quality postoperative care to prevent complications, and allow commencement of rehabilitation and retraining. In the absence of significant contraindications it is important for all these patients to be on prophylactic anticoagulation due to the high risk of venous thromboembolism in the postoperative period. Traditional teaching dictates that a 3–4 week period of static immobilization must follow any tendon transfer procedure, to allow tendon healing and reduce risk of early rupture.^{21,41,79} However, in recognition that early active mobilization has become widespread practice following tendon repair procedures,^{83–86} there is mounting evidence that such rehabilitation protocols may improve outcomes following tendon transfer procedures as well,^{87,88} an approach particularly advocated by Brandsma.⁸⁹ Such protocols may reduce oedema and development of adhesions, and allow earlier commencement of rehabilitation and retraining.⁸¹

According to a systematic review performed by Sultana *et al.*, benefits of such protocols include reduced cost of rehabilitation, improved function in the short term, similar long-term outcomes, and no increase in risk of complications such as tendon pull-out.⁸⁸ It is important to emphasize that careful patient selection is essential when deciding on a rehabilitation programme. For success with early active mobilization patients must be particularly motivated, compliant and able to follow complex instructions from hand therapists. In both early or delayed mobilization protocols, mobilization should be introduced in a gradual fashion by hand therapists experienced in managing patients following tendon transfer or repair procedures, followed by progressive strengthening exercises and finally, full activity.^{41,90} Devices such as electrical stimulators and biofeedback may be used for muscle reeducation, though studies of their effectiveness have been disappointing.^{91,92}

The senior author's preference is to reserve early active mobilization for selected patients. Otherwise, the protocol requires immobilization for two weeks followed by passive range of motion exercises, and then a gradual return to normal activity after 6–8 weeks. Following restoration of elbow extension with either deltoid to triceps or biceps to triceps transfer it is our routine to immobilize the elbow in a Bedsloe caliper splint for 12 weeks. Initially the caliper should be set to allow a range of motion of 0 to 15°, to be gradually increased each fortnight if the previous increase is tolerated.

Conclusion

Tendon transfers have been used since the late nineteenth century to restore function for patients with irreversible neurological or musculoskeletal injury. Recently, with the development of microsurgical techniques and improvement in our understanding of neuroanatomy, nerve repair and transfer and innervated free muscle transfer have become the usual treatments of choice for many of these problems. However, there remains a role for tendon transfer, either for early function during nerve recovery, or for those whose pathology are not amenable to nerve repair or transfer. This chapter should equip the surgeon with the essential knowledge of tendon transfer: the principles, approach and techniques required to perform these procedures.

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CHAPTER 54

Amputations, replantation and thumb reconstruction

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Complete and partial digit loss in the hand has serious sequelae: physically, psychologically and financially. Surgical intervention is not always indicated. However, when used appropriately, replantation and reconstruction can make a real difference to long-term function. The key is matching the reconstruction to the patient and choosing the right time.

Although surgical and rehabilitation techniques have evolved, there are still aspects of repair and reconstruction that we need to improve. For example, we still have limited outcomes in sensory recovery after nerve repair. In complex digit reconstruction and replantation we use autologous donor sites, leaving defects and scars.

Occupational injury accounts for a significant number of amputations, including agricultural and industrial accidents.¹⁻³ These are complex crush and avulsion injuries, and digit salvage and achieving functional outcomes can be challenging, even to the most skilled and experienced hand surgeon.⁴

Aetiologies

Digit loss can result from either systemic or local factors. Systemic illness such as diabetes, vasculitis and arterial insufficiency all present extremely challenging clinical scenarios. These cases should be managed in a multidisciplinary setting and systemic therapies such as prostacyclin, steroids and immunosuppressants will play a significant role in achieving successful outcomes.

In the treatment of malignant disease of the soft tissue or bone wide local excision or amputation may be the only option for local tumour control. This may need to be followed by adjuvant chemotherapy or radiotherapy and again multidisciplinary management is obligatory. Planning and timing of reconstruction is critical. It can be useful to confirm clear tumour margins before embarking on complex reconstruction.

The commonest cause of digit loss at any level is trauma. Avulsion, crush and thermal injuries are the more difficult to treat, largely as the zone and extent of injury are often underestimated at the time of presentation.

Clinical history and assessment

The potential options for replantation or reconstruction following amputation are based on assessment of the patient and the amputated part.

The patient's age, occupation and pastimes along with their previous medical history will aid the decision-making process. It is important to consider the psychological implications of injury and reconstruction. The patient's future plans and how they perceive themselves and the injury will all affect the outcome.

In assessing the amputated part again there are three elements to consider: the condition of the part, the functional importance of the part and the ischaemic time if the injury is proximal.

Management principles and amputation options

Non-operative management

Success in elective amputation or terminalization of an amputated digit is reliant on achieving a pain-free, padded, sensate, mobile finger tip. If the residual finger end is neither padded nor sensate then it is unlikely that it will be involved in useful function. In cases of ischaemic loss in diabetics this can be a real challenge as the remaining soft tissues are poorly vascularized, often insensate and the risk of infection is higher. Here it is best to ensure all necrotic tissue, including bone, is excised and to leave the finger end to close by secondary intention. This will take considerable time but long term the results are good.

Distal finger tip injuries in healthy patients, where the distal phalanx remains covered with soft tissue, can be managed non-operatively. This results in a sensate tip with reasonable padding and there is no other donor site. Treatment in this fashion can take several weeks and the patient needs to be informed so they can adjust their lifestyle accordingly.

Operative principles

In distal finger tip injuries where the injury, the patient or the surgical expertise preclude replantation then homodigital, cross fingers flaps and terminalization of the finger tip are all viable options.

It is important to remember that terminalization will almost always result in further shortening of the skeleton to achieve soft tissue cover. The digital nerves should always be isolated and trimmed so that they are lying in a well-padded proximal location and are not likely to cause painful neuromata over time. In distal injuries the residual nail bed should be excised to avoid a hook nail deformity. The flexor and extensor insertions should be preserved where possible.

In more proximal loss at the level of either the proximal or distal interphalangeal joints, the condyles should be shaved to improve the appearance, the tendons on the extensor and flexor surface should be allowed to retract so as to avoid a quadregi effect and sufficient volar skin is left to cover the finger end.

Ray amputation is often reserved for the index finger and second metacarpal ray. It is rarely performed as a primary amputation unless for soft tissue tumour excision. As it is often performed to improve cosmesis following proximal digit loss then a volar approach is superior to avoid a visible scar. It also provides better visualization of neurovascular structures. In the central rays transposition of the adjacent ray to close gapping between fingers and avoid grip incontinence is a reasonable option. Balancing the loss of grip strength and the improvement in cosmetic appearance are the key factors in deciding whether to transpose an adjacent ray. If grip strength is important then transposition should be avoided.^{5,6} Careful dissection of digital neurovascular structures is important to avoid hypersensitivity and neuroma formation in the palm of the hand.

Replantation

Indications

The traditional indications for digit replantation include multiple digits, thumb, single digit loss distal to the flexor digitorum superficialis (FDS) insertion and all amputations in children. However, with the advent of improved microsurgical techniques and better outcomes of survival and function, other indications that previously we would have deemed unlikely to yield long-term success are now only relative contraindications.^{7,8}

We know now that digital replantation is possible up to 24h after the injury,⁹ in children under 1 year¹⁰ and after other organ transplantation.¹¹ We can also store amputated digits temporarily elsewhere on the body (contralateral forearm, groin, axilla) while the zone of more proximal damage is reconstructed and made suitable for replantation.^{12–14}

It is also possible to get good functional recovery from isolated index finger amputations proximal to the FDS insertion. Buntic *et al.* have demonstrated total active motion of 170° in zone 1 injuries and 133° of total active motion in zone 2 injuries.¹⁵

However, it is also worth remembering that a poorly sensate, stiff index finger will be bypassed for functional purposes.

Assessment and preoperative preparations

The initial clinical assessment of parts and patient should include analysis of the type of injury – crush, avulsion, thermal injury, guillotine and compression. In transportation of parts they should be kept moist and cool. It is imperative that the amputated part is not frozen or directly in contact with water or ice. Preoperative photographs and X-rays are always worthwhile to document the soft tissue and bony injury level.

Operative principles

Preparation of patient and part can occur simultaneously. The time spent discussing operative planning with the anaesthetic and theatre teams is invaluable. Communication about types of fixation, timing, operative sequencing is essential for ensuring optimal surgical time management. This is very important, in particular where ischaemic time is already extensive and critical, such as macro limb or multiple digit replantation.

A hyperdynamic circulation with peripheral vasodilatation is desirable during the operative procedure. This can be combined with close monitoring of core and peripheral temperatures and aiming for a difference of less than 1° while ensuring the patient is warm and comfortable. Local anaesthetic regional blockade will also help with ensuring a pain-free setting for the patient both during and after surgery and can be substituted for general anaesthesia in situations where the patient is not suitable.¹⁶

During the induction of anaesthesia the amputated parts can be assessed, prepared and marked. In general the injury will dictate the access incisions but where there is choice Brunner incisions yield good access on volar surfaces and are amenable to cover vessels and nerves after revascularization and the ensuing swelling.⁸ It is worth marking vessels and nerves; even though they may appear obvious now, they may not be later once osteosynthesis is completed, and it is frustrating and time wasting to relocate structures a second time. It can be difficult to identify veins dorsally but either they can be marked at the time of initial assessment, if easily seen, or you can wait until the arterial repair is flowing and see where they are located then. The former is easier for repair as the tourniquet is still inflated and visibility is far superior.

Operative sequencing is important but we think the most important part is that the operator is at ease with the routine chosen, whether it is the work from volar to dorsal or in more traditional method of achieving bone fixation first followed by tendon repair. It can be advantageous to repair the flexor tendons first prior to bony fixation as this gives very clear visibility of the flexors for repair and aids in stabilizing the parts so that bony fixation is easier too, but clearly if the option of a volar fixation plate is used then this is not compatible.

In replantation of digits, including the thumb, achieving osteosynthesis and repairing tendons prior to microvascular or nerve repairs is important as otherwise there is a risk

of disruption of your microscopic-assisted repairs and/or problems with vessel length.

In choosing the method of fixation, the level of the injury, bone comminution, distance from joint surfaces and soft tissue access will dictate the possibilities.⁸ Internal fixation can be a very successful option as you already have good visualization of proximal and distal components and so greater anatomical precision is possible. Also early postoperative mobilization and rehabilitation will be possible with greater potential for long-term functional success.

Skeletal shortening is beneficial, in particular if there is comminution of parts. It aids in achieving direct repair of neurovascular and tendinous structures. It will also aid the soft tissue cover, which often is not fully considered until the end of the case and closure over vessel or nerves can be problematic. Bone shortening will save time and improve long-term functional outcomes, in particular for macro or multiple replantations where nerve repairs are mixed and more distant from motor and sensory end organs.

It is easier and quicker to achieve plate fixation/retrograde Kirschner wiring to the mobile amputated part first once the level of bone synthesis is decided. A plate can be placed dorsal or volar depending on the level of injury and the bone cortices available.

Standard tendon repair techniques using epitendinous and multistrand core sutures are well documented and these should be employed to achieve the optimal tendon repair.^{17–21}

Correct evaluation of level and extent of arterial vessel injury in the hand and amputated part is crucial. In avulsion injuries the level of intimal damage exceeds far beyond the level of tendon and bone injury. In order for the revascularization to work successfully, inflow perfusion pressure is very critical. It is worth assessing and ensuring good inflow before proceeding to vessel repair. It is easier to decide to vein graft at this early stage than have a tight repair or no inflow and have to repeat and revise anastomoses later. If there is any doubt or concern about the inflow it is always worth checking with the anaesthetist that the patient is haemodynamically stable, warm and well perfused, so that you can then focus on more specific local factors to ensure adequate inflow.

Sensory outcomes are critical in ensuring long-term functional use so nerve coaptation should be performed with good visualization of nerve ends and ensuring proximal and distal nerve ends are of good quality.²² This can be achieved with ease after arterial anastomoses and while the tourniquet is still inflated.

The alignment for venous repair is dictated by the bone fixation and if anatomical alignment has been achieved then co-located proximal and distal vein ends will be visible.⁸ Venous drainage can be achieved through direct repair, vein grafting or vein transfer from an adjacent digit.^{23,24} The latter is a very worthwhile option, excluding the need for vein grafting and ensuring the recipient vein is out of the zone of injury as it is from an adjacent unaffected digit.

Having achieved success in the osteosyntheses, tendon repairs and revascularization it can be distressing to find that there is inadequate skin cover at the end of the procedure, so time spent planning skin incisions and flaps early on is invaluable. Skin closure should be loose and sutures infrequent to allow for tissue swelling and any blood that will seep from wound edges in the early postoperative time.

Ring avulsions

Ring avulsions have been classified by Urbaniak and subsequently Kay and Adani. These classification systems are based on the severity of vascular, bone and joint injury, correlating these with the predicted functional outcome.^{23,25–28} In the Kay classification system, which is probably the most widely used today, type I describes an injury where the digit vascularity is adequate with or without a bony injury. In type II and III injuries the digit vascularity is compromised and in the latter there is also a bony injury. The presence of a bony injury and vascular compromise, either arterial or venous, predicts a more sinister outcome for the digit, with a higher risk of early or late failure. The poorest prognosis is for those classified as type III injuries where a bony injury is combined with both arterial and venous insufficiency.²⁵ Most recently, Chung and Sears concluded that outcomes from ring avulsion of fingers (78%) was comparable to that for replants in general (80%). This was a systemic review article and they have recommended replanting complete finger avulsions where the proximal interphalangeal joint and the FDS tendon are preserved.²⁹

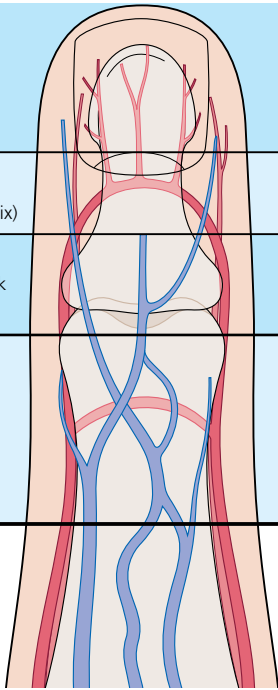
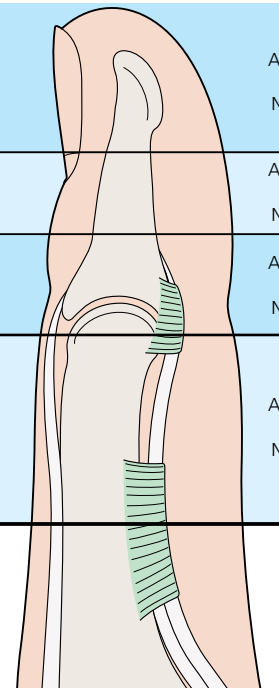
Distal digital replantation

Distal digital replantation has regained popularity over the last two decades. In the early era of microsurgery, distal replantation was deemed to be more technically demanding. This, combined with doubts about functional outcomes and increased costs, led to a drift away from very distal replants. However, there are several published series of outcomes from distal replantation showing good functional outcomes and a high success rate in specialized centres.^{30–32}

Finger tip injuries and amputation levels have been classified by Allen, Foucher and Tamai.^{7,33,34} In distal replantation, Sebastian and Chung have proposed a new system based on the difficulty of the microvascular and neural repairs. We have found this paper to be both practical and very useful in balancing and explaining the potential for success/failure in the various amputation groups³⁰ (Figure 54.1).

Success rates for replantation of distal finger tips rely on the experience and microsurgical expertise of the operator. Although the vessel diameters of both the distal arteries, and in particular distal veins, are very small it is still possible to achieve and maintain patency of vessel anastomoses.

Assessment of the amputated part is essential to decide if there are suitable patent vessels with the potential for revascularization. It is the paucity and size of available veins for drainage that is the limiting factor for successful outcome and survival.^{32,35}

Zone 1 Amputations (distal to FDS insertion)	Zone 1 Distal amputations (finger tip amputations) (distal to FDP insertion, at the root of nail bed)	Zone 1A (distal to lunula through sterile matrix)			Artery: Very difficult Vein: ≈Impossible Nerve: ≈Impossible
		Zone 1B (between lunula and root of nail bed, through germinal matrix)			Artery: Difficult Vein: Very difficult Nerve: Very difficult
		Zone 1C (between FDP insertion and neck of middle phalanx, periarticular)			Artery: Easy Vein: Difficult Nerve: Easy
	Zone 1 Proximal amputations (between FDP and FDS insertions)	Zone 1D (between neck of middle phalanx and FDS insertion)			Artery: Easy Vein: Easy Nerve: Easy

The fingertip is defined as the portion of the finger distal to the flexor and extensor tendon insertions

Thumb amputations are classified using the same descriptors. Zone 1 thumb amputation refers to amputations distal to the mid-proximal phalanx of the thumb (distal to first web)

Figure 54.1 Sebastin and Chung classification system for distal digital amputations, outlining the difficulties in microsurgical vessel and nerve repairs at each level. The finger tip is defined as the portion of the finger distal to the flexor and extensor tendon insertions. Thumb amputations are classified using the same descriptors. Zone 1 thumb amputation refers to amputations distal to mid-proximal phalanx of the thumb (distal to first web). FDP, flexor digitorum profundus; FDS, flexor digitorum superficialis. Source: Sebastin & Chung, 2011.³⁰ Reproduced with permission of Lippincott Williams & Wilkins.

The operative method is similar to more proximal replantation procedures. It is important to tag the vessels, and nerves if available, to aid ease and speed for future repair. The distal phalanx can be secured with a small Kirschner wire where appropriate. It is worthwhile repairing the nail bed early in zone 1 and 2 amputations as this aids in stabilizing the finger tip while performing the vascular anastomoses. The location of nerves and vessels varies slightly, depending on the amputation level (Figure 54.1). In zone 1 and 2 injuries it is not always possible to locate a dorsal or even a volar vein in the amputated part until after the artery is flowing. Even if veins are visible, the anastomoses are challenging due to vessel size and visibility. However, success rates are higher when venous drainage has been achieved.³⁰ Alternative method for temporary venous drainage, such as external bleeding or venocutaneous fistulas, can be used successfully but these need to be explained to the patient along with the risk of transfusion.³⁶ Clearly there is a definite risk–benefit ratio that needs to be discussed and agreed with the patient in relation to achieving successful finger tip replantation versus terminalization of the affected finger^{30,32} (Figure 54.2).

The benefits of distal replantation include preservation of finger length, better range of motion of the distal interphalangeal joint and less risk of finger tip hypersensitivity. These have to be offset against a longer hospital stay and operative time with ensuing greater costs. There are cultural and social factors

which influence the expectations and aspirations of this group of patients which are clear in the published literature. The majority of distal finger replantations are undertaken in Asia.^{30,37}

As the incidences of more proximal and multiple digit amputation are declining secondary to health and safety education and awareness it may be these cases of very distal replantation will need to be performed in specialized microsurgery centres.³¹

Postoperative care

After surgery, a splint is applied. In the early postoperative time this does not need to be rigid but needs to protect the replanted digit(s) from injury and also to promote a comfortable position for the patient and limb. It is important to ensure that any dressing is not constrictive or tight circumferentially and that the dressing used will not dry out into tight constricting bands over time. Patients should be cared for in warmed environment with expert nurses. Maintaining a good perfusion pressure, along with adequate analgesia, is critical to outcome success as with all microvascular procedures.

Regular monitoring of any revascularized parts is essential but it is also important to try to maintain a stress-free environment, especially in children where the risk of vasospasm is higher. Thus haemodynamic monitoring should be minimal in children unless there is a clinical reason to justify provoking a stressed or agitated young child.

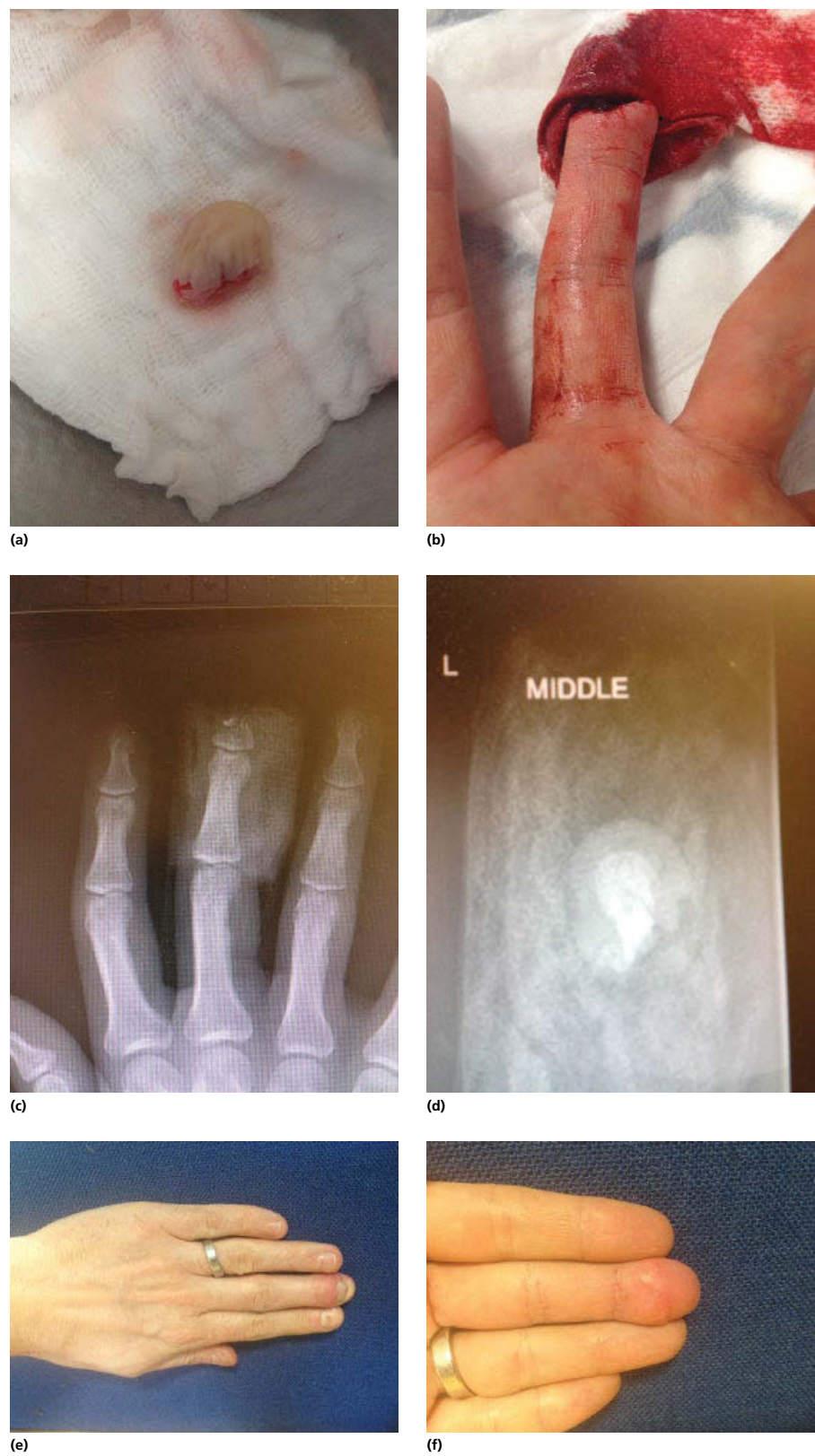


Figure 54.2 (a) Amputated distal tip of middle finger. (b) Proximal finger with amputated tip. (c) Finger X-ray demonstrating amputation level. (d) X-Ray of amputate. (e, f) Replanted finger at two weeks.

If there is any suggestion of arterial or venous compromise then immediate re-exploration should be performed. It is better to release sutures, and check vascular status early if there is doubt, as the longer the interval between low/no flow in the vessels and re-exploration the less likely that salvage will be possible.

Complications

The complications of digit replantation can be classified in a timeline of occurrence.

Failure to achieve or maintain flow is particularly disheartening, but can usually be avoided by careful patient and part selection. This is usually due to a combination of factors, including the type of injury, for example a crush injury that has not been clarified or appreciated in the preoperative assessment. There can be errors in transportation of the amputated part, which on occasion is left in direct contact with ice and is frozen, causing cellular damage that is irreversible.

If there is failure of the arterial flow then it is always worthwhile returning to Virchow's triad and assessing the blood flow, vessel wall and the circulating blood.

The blood flow can be compromised locally from tight dressings or skin flaps that have become restrictive following local swelling, or it may be compromised systemically if there is a poor circulating volume or the perfusion pressure is low.

There may be residual vessel damage from the injury which necessitates a vein graft for reconstruction of the vessel wall. There may be procoagulant factors in the circulating blood related to smoking, inflammation or systemic disease. Each should be addressed in a systematic and timely fashion.

Compromise of venous drainage can present in two ways: early, with excessive bleeding or later, with blue discoloration and swelling of the digit. Assessment is similar to that for arterial occlusion and on occasion simply releasing dressing and sutures will allow return of flow if the constriction is realized in a timely fashion. Late venous compromise is often related to infection and the chance of regaining flow is low. It is important to pick up problems with venous outflow early, as once it is prolonged the arterial flow will cease as well.

Infection is really only a problem if it interferes with vessel flow and usually presents as this, but other soft tissue or bony infection does not present that often.⁸

Cold intolerance can cause significant disability postoperatively, but is related to the injury process rather than replantation.³⁸

Complications of bony union and position can ultimately cause concern long term, in particular when combined with stiffness or tendon adhesions. So it is always worth taking the time to achieve good bone apposition and correct alignment at the time of replantation rather than have to address problems at a later stage.

Outcomes

In children, survival following replantation is lower than in adults. This is usually attributed to the mechanism of injury in children being more likely to be a crush/avulsion injury.^{8,39} Saies

et al. demonstrated an overall survival for digital replantation in children of 63%. The salvage rate was 72% when there was laceration, versus 53% when there was crush or avulsion injury.⁴⁰

In adults, the outcomes in patients with diabetes and smokers are statistically worse.⁴¹ Rates of survival of replanted finger in adults range from 60% to 94%,^{42,43} again depending on systemic factors and the type of amputation mechanism.

The recovery of useful digit (54%) and thumb (61%) sensation was analysed by Glickman and MacKinnon in 1990. The mechanism of injury, age of the patient, cold intolerance, digital blood flow and sensory re-education all influenced the recovery level.²²

The rate of secondary surgery is high, with published rates of up to 93% in proximal replants.⁴⁴ Tenolysis, skin grafting, bone grafting, joint fusion and osteotomy are all common in injuries proximal to the FDS insertion and in more severe degloving injuries.^{42,45}

Thumb

Anatomy and biomechanics of movement

The thumb has two phalanges and one short metacarpal articulating with the trapezium. It is this latter articulation that makes the thumb unique and sets it apart functionally from the other digits of the hand. The carpometacarpal joint is both concave and convex on each articular surface, a saddle joint. On an anteroposterior X-ray view the trapezium is concave and the metacarpal base is convex but in the lateral view the opposite is true. It is this unique geometry of the basal joint that allows multiplanar motion, and in particular opposition.⁴⁶

There are four extrinsic tendons to the thumb – one flexor, two extensors and one abductor – and four intrinsic muscles – abductor, adductor, flexor and opponens pollicis. Each functional movement of the thumb is achieved through a complex sequence of contracting and relaxing of these musculotendinous units. It is this fine balance of flexibility and stability which makes our thumb useful.

The sensitivity of the thumb tip enables fine touch and precision pressure grips. The radial and median nerve supply the thumb on the volar and dorsal surfaces with two central volar digital nerves and multiple small dorsal nerves supplying the thumb and webspace.

The thumb is supplied by an arcade of arteries on the flexor and extensor surfaces.^{47–49} The princeps pollicis is often the named dominant blood supply and supplies the radial and ulnar digital vessel in 73% of cases.⁴⁸ Ames *et al.* demonstrated a superficial and deep vessel to the first webspace in 54% of 40 hands dissected.⁴⁷ Parks *et al.* demonstrated a single end artery to the thumb in 75% of 50 cadavers they studied, correlating with the risk of ischaemia from injection, thrombosis and injury.⁴⁹

There are both dorsal and volar venous networks of the thumb. The dorsal system commences with a venous arcade at

the level of the nail bed. The venous diameter gradually increases where the veins converge and travel proximally to drain into the metacarpal arcade and eventually the cephalic vein.⁵⁰

Thumb reconstruction

Thumb replantation and reconstruction has evolved over the last 30 years and we are now more demanding of the outcomes of reconstruction functionally and cosmetically. This is largely related to improved microsurgical techniques and facilities and more refined rehabilitation programmes. Through the new access to internet and social media, patients are also more aware of outcomes and we are better at sharing ideas and competing for the best outcomes.

However, there are limits to our success, including incomplete nerve regeneration and recovery, a donor site defect due to use of autologous reconstruction and limited resources for the psychological aspects of loss and reconstruction.

The frequency and method of thumb amputation has also changed over time, with health and safety at work and changes in population education and awareness.

Clinical history and assessment

The aetiology of thumb loss and information about associated medical conditions are important in deciding the reconstruction options available for each case. The type of injury (i.e. avulsion, guillotine, crush) defines the level of bone and soft tissue injury, including the possibility of extension of the vascular injury, in particular in cases of avulsion.

The survival of thumb replantations are poorer in smokers and this should be considered when discussing outcomes preoperatively.^{51,52}

During examination, an assessment of remaining soft tissue cover, defects in sensation and associated joint stiffness or injury should be considered.

Management principles and options

Acute management of thumb injury and loss is similar to that for other digits. As the functional implications of thumb loss are so great, preservation of length, mobility and sensibility is critical.

Methods for thumb reconstruction have been classified into three groups – distal third, middle third and proximal third. An extensive description of this classification system and proposed reconstructive methods was published by Muzaffer *et al.* in 2005.⁵³

Loss through the distal third of the thumb

Injuries that are distal to the interphalangeal joint of the thumb are classified as distal third injuries. Although there may be some loss to the thumb length with these injuries, the effect on functional use is not significant.⁵⁴ However, ensuring adequate soft tissue cover and sensation is critical to outcome. As with any pulp injury, if the area is small and suitable for non-operative treatment then this is optimal. Sensation is then preserved, there is no donor site and in general mobility is also maintained.

Operative options

In defects where there is exposed distal phalanx or there is significant pulp loss, then adjacent flaps can be considered. These include homodigital flaps, such as Moberg's homodigital advancement flap, which enables closure of volar soft tissue defects. This is a bipedicle volar flap incorporating the digital nerves and arteries with overlying skin. In cases where this is causing significant flexion at the interphalangeal joint then a proximal releasing incision on to the base of the thumb will give better advancement. It is important, as with all islanded volar flaps, not to dissect the arterial pedicle too extensively to avoid damage to the arteries or accompanying draining venae comitantes.

Adjacent digits can also be used for soft tissue cover. In order to avoid the possibility of a webspace contracture, cross-finger flaps from the index finger are generally avoided. Foucher designed an islanded flap based on the first dorsal metacarpal artery. This is a reliable flap with a skin paddle over the extensor surface of the proximal phalanx of the index finger. The pedicle is located over the first dorsal interosseous and it can be raised as a sensate flap.

As with all islanded digital flaps, the pedicle must not be dissected extensively as this will disrupt the venous drainage. The arc of transposition is at the base of the webspace and the flap can be tunnelled subcutaneously to reach the thumb tip. This is an excellent local flap for covering the tip of the thumb, preserving length and providing adequate soft tissue cover where patient or surrounding s preclude any more advanced techniques.

We have used this for a polytrauma case where the patient had an extensive list of other significant injuries, including an intracranial bleed, spinal and pelvic fractures and the thumb tip arrived to the hospital frozen. It is worth noting that in cases such as this, visual dissection of the pedicle is not possible as there is extensive subcutaneous haematoma and swelling. However, it allows immediate and efficient closure of the thumb without shortening. The full-thickness graft to the dorsum of the index finger heals remarkably well. This leaves all options for further reconstruction available for the future once the patient has recovered mentally and physically (Figure 54.3).

There are other less commonly used flaps, such as islanded pulp flap from the ulnar border of the middle finger or free toe pulp from the great toe, both of which are reliable and are worth considering where local options are limited.

Loss through the middle and proximal thirds of the thumb – toe transfers

The vascularized transfer of a toe for thumb reconstruction is a well-established technique. In 1966 Buncke described his experience with hallux to hand transplant in a Rhesus monkey.⁵⁵ Subsequently, Cobbett went on to report a case of transfer of the great toe to replace an amputated thumb.⁵⁶ The suitability of either a big toe, a modification of this transfer, or a second toe for reconstruction is dependent on the operating surgeon, the



Figure 54.3 (a) Multiple trauma case with thumb amputation (see text for details). (b) Postoperative X-ray demonstrating preservation of bony length. (c) Four weeks postoperative Foucher flap to preserve length and provide soft tissue cover.

injury and the patient. There are pros and cons to each in relation to the appearance of the reconstruction and the donor site.

The principles of toe transfer harvest are similar to those for other microvascular flap harvest. A knowledge of the arterial and venous anatomy of the foot, in particular of the first web is critical.^{57,58} The timing of reconstruction is usually delayed in order to ensure psychological and physical preparation, but immediate reconstruction has been reported without increased morbidity.^{59,60} The advantages of immediate reconstruction are apparent in that digit reconstruction is immediate, including the nerve regeneration and mobility. However, as these are highly specialized transfers, the chance for revision and reconstruction if there is microvascular failure is slim. In cases where there are other associated injuries, immediate reconstruction is precluded.⁶¹

In planning a thumb reconstruction using a toe transfer there are several elements to consider. These include the level of bony injury, the quality of the remaining bone and joints, the motor units remaining for flexion and extension and the potential for regaining useful sensation.

It is not advisable to transfer the metatarsophalangeal joint of the great toe as this is important both functionally and aesthetically for the foot. The metatarsophalangeal joint of the second toe rests in extension and the arc of motion of this joint lies in extension rather than flexion. Thus where there is very proximal thumb loss, pollicization of the index finger should be considered even if it too is injured.⁶²⁻⁶⁴

As with all free tissue transfers there are global factors that are key. These include ensuring microsurgical skill of the operating team and an anaesthetist familiar with the principles of microvascular transfers. The general operative principles are similar to those outlined for replantation but the advantage in late reconstruction is that the whole team and patient have more time to prepare. The key elements acutely relate to maintaining flow and avoiding vasoconstriction once the toe has been transplanted. Long-term functional outcomes will reflect tendon and nerve recovery and use.

The procedure can be performed by one or two teams depending on what is the tradition in your unit. We have traditionally had a single team with a lead surgeon and the benefits of this are that the knowledge of vessels and soft tissue dissection does not need to be transferred. We have found this to be an efficient and safe method for microvascular transfers and the operative times are comparable to a two-team approach.⁶⁵

In preparing the hand, the level of future osteosynthesis is central and all other soft tissue structures can be measured from this site. Adequate exposure of the digital nerves, blood vessels and tendons for repair is essential. It is worth remembering that sufficient access and visibility is needed for repair of longitudinal structures, and time spent dissecting and preparing at this initial phase is always worthwhile. Once the toe has been transferred, the ischaemic clock is ticking and the transfer itself can obscure the proposed site of repair of tendons and nerves in particular.

The choice of exposure for tendons and nerves will depend on the previous injury and it is unusual to have to excise any skin, unless it is scarred and restrictive. The choice of vessels for inflow and outflow will vary, but where possible remaining out of the zone of injury is optimal as here you avoid scars and vessel dissection is quicker and easier. In thumb reconstruction there are several options but the radial artery and cephalic vein at the anatomical snuffbox are almost always available, and the vessel size is ideal for revascularization of a toe transfer.

There are several excellent articles on toe-to-hand transfers which detail techniques for flap dissection.⁶⁵⁻⁷¹ In harvesting a second toe it is usual to harvest an extension of skin overlying the metatarsophalangeal joint onto the dorsum of the foot and leave the plantar surface free from scar. There are various methods of flap dissection but probably the most common nowadays is early retrograde dissection of the vascular pedicle.^{65,71}

This means identifying the dorsal veins first and then proceeding to the first webspace and identifying a suitable arterial system linked with the tibial vessel of the second toe. In about 80% of cases a reliable dorsal system is present and the artery can be dissected proximally to join the dorsalis pedis with relative ease after dividing the intermetacarpal ligament.^{57,58} In the other 20% of cases the plantar system is dominant and retrograde dissection of the arterial pedicle is more time consuming as the vessel courses beneath the interosseous muscle to the great toe, with various muscular branches that need to be ligated or coagulated.

Once the arterial and venous pedicles have been identified and dissected then the extensor tendons, dorsal nerves, plantar digital nerves and flexor tendons can be dissected methodically. Finally, the soft tissues are freed from the metatarsal along the level of the metatarsophalangeal joint from the fibular side first, while visualizing the flap pedicle so as to avoid damage to important structures.

At this stage the tourniquet can be deflated and the flap pedicle can be checked. On occasion it may take a few minutes for the toe to regain the pink blush of well-vascularized tissue and it is worth covering it with a warm damp swab and taking a break rather than increasing the risk of further vasospasm by interfering with the pedicle.

Prior to dividing the vascular pedicle it is worth rechecking the measurements of the longitudinal structures from the point of bony fixation.

After removing the toe from the foot, attention is then focused on preparation of the toe for bony fixation and tendon repair. The most common sites for bony fixation are at the base of the proximal or middle phalanges of the second toe or, in a great toe, transfer through the proximal phalanx or at the base of the distal phalanx. This is because the metatarsophalangeal joint of the great toe is functional and every effort should be made to preserve it where possible. In cases of second toe transfer it is actually better aesthetically to remove the whole ray and leave the patient with a linear scar between the great and third toes.

The type of fixation used can vary, depending on the access, level of bone visible and the technical skill and preference of the operator. In adults, where possible internal plate fixation is preferable and assists in postoperative early mobilization. However this is not always an option and simple fixation with longitudinal Kirschner wires is adequate. The wire or plate can be fixed to the transfer first after the cartilage has been removed from the joint surface. It is important to remember not to damage the growth plate in children.

In acute reconstruction after tumour resection the flexor pollicis longus tendon can be repaired distally and this is easily achieved before the bone is fixed as visibility of the tendon ends is excellent.

Attention can then be turned to the extensor tendon and flexor tendon if not already repaired. The dominant extensor tendon for the distal joint of the second toe in particular can vary, as this can be assessed easily prior to tendon repair. Finally, all microvascular structures are repaired while the tourniquet is inflated. It is worth repairing the nerves at this stage as visibility and access are optimal. In long-term outcome, recovery of sensation is critical to enable functional use (Figure 54.4).

Once all structures have been repaired, the tourniquet can be deflated, the vascular clamps are removed and vessel flow can be assessed. A topical vasodilator is often applied across the anastomotic site at this stage; the hand is covered with a warm damp swab and left to perfuse.

The foot closure after second toe harvest requires removal of the metatarsal and repair of the intermetatarsal ligament, followed by direct skin closure. This yields an acceptable donor site.

Closure of the foot after harvest of the great toe can be more problematic, as local flaps and skin grafts are required to maintain length. The donor site is cosmetically poor. Hence the use of modified great toe transfers, which yield much more acceptable donor sites.



Figure 54.4 Free toe transfer for thumb reconstruction in a child after traumatic loss.

Modifications of great toe transfer

The free neurotized wraparound flap from the big toe was described by Morrison *et al.* in 1980.⁷² This incorporates part of the distal phalanx of the great toe with the nail bed, neurovascular pedicle and soft tissue cover, so that the donor site of the great toe is much improved and preserved. The thumb is reconstructed either by this alone or with an interim iliac crest bone graft to increase the length of the construct. The transferred toe is narrower and more 'thumb like' than the traditional great toe transfer, but still has the aesthetic and functional elements to provide a valuable and useful thumb reconstruction. Further modifications of this method using pre-operative distraction lengthening, extended length and combined with a second toe proximal interphalangeal joint have all been described.^{73–75}

Distraction lengthening

Continual distraction lengthening of the thumb was first described by Matev in 1973.^{76,77} This is a useful technique for lengthening the thumb metacarpal and preserves the inherent sensation of the thumb soft tissue envelope. This is a very reliable technique and the bony defect can be left to ossify spontaneously or it can be bone grafted to achieve bony union and stability.^{78,79} The length of bone distracted can be up to 3 cm while still maintaining adequate soft tissue cover and sensation.⁷⁹ This technique can also be used in conjunction with vascular toe transfers to improve the donor defect of the foot.⁷⁵

However it is worth remembering that the distracted thumb is stiff and this, combined with often inadequate soft tissue cover, means that the reconstruction may not be as useful as expected. However, distraction definitely has a role in thumb and digit reconstruction and is a valuable skill for the reconstructive surgeon. A simple linear distractor is adequate for thumb lengthening as rotation is not usually a problem.

Pollicization

In proximal loss of the thumb, apart from microvascular toe transfers the only other current option for reconstruction is pollicization of another digit on the hand. In 1984 Michon *et al.* compared the functional and cosmetic results for reconstruction of the traumatically lost thumb using toe transfers and pollicization of the index finger. They concluded that toe transfers offer better overall functional outcomes where available, but in isolated proximal thumb loss pollicization can yield very functional and cosmetically acceptable results.⁶³

Pollicization of the index finger was first described by Gosset in 1949.⁸⁰ Since then, modification of surgical technique, indications and outcomes have all been published extensively.^{53,80–84} In proximal thumb loss, pollicization offers the ability to recreate a sensate stable thumb of adequate length to oppose to the other remaining digits of the hand. The index finger is most commonly transposed and injury to this digit does not preclude transposition. Injured index fingers can be transferred in isolation or in combination with a great toe wraparound flap.^{64,85}

The principles for recreating a functional thumb are similar for traumatic and congenital absence but in traumatic loss there is more flexibility in recreating intrinsic and extrinsic motion. The proposed digit for pollicization should offer the potential for recreating a thumb of adequate length, alignment, stability, sensibility and mobility that would render it functional and cosmetically acceptable.^{80,84,86–90}

This procedure involves careful planning of skin flaps to recreate a webspace and provide cover for the new thumb. The dorsal veins and nerves are dissected and preserved where possible to ensure adequate venous drainage and preservation of sensation. The extensors to the digit are then identified and can be used for recreation of extension and abduction of the thumb where appropriate. The distal extensor expansion can be divided longitudinally to recreate distal attachments for the intrinsic thumb abductor and adductor, where preserved, as well as a central element to recreate long extensor.

In order for the digit to move untethered to the position of the thumb, the vascular arterial pedicle must be dissected sufficiently proximally and the radial digital artery of the middle finger will need to be divided from the common artery. In the trauma setting, adequate arterial supply to this adjacent digit should be confirmed before dividing its radial digital artery. The digital nerves must be dissected to allow adequate transposition and movement and this requires the intraneural dissection of the common digital nerve between the index and middle rays.

The pulley system of the proximal portion of the flexor tendons is released and the intrinsics are dissected from the metacarpal to enable shortening and adjustment of the bone.

The second metacarpal must be shortened, rotated and transposed to yield, where possible, a thumb of matching length to that on the contralateral hand. Once the digit is free then it can be repositioned in the place of the absent thumb, followed by tension-free realignment of the longitudinal structures. In traumatic reconstruction the length of the second metacarpal resection, the adjustment of the tendon alignment, the reconstruction of the short muscles of the thumb and the positioning of the skin flaps are dependent on the level and extent of bone and soft tissue loss.

Webspace

The first webspace between the thumb and index finger allows the thumb a unique range of functional rotational motion. The skin envelope is soft, thin and pliable to allow for a wide range of motion between the thumb palm and digits, including adduction, abduction and opposition. This specialized area is often involved either primarily or secondarily in injuries to the thumb and first ray.

A contracture of this webspace will involve not only the skin but also the fascia and muscles it clothes. It is important in releasing the webspace that all tight and restricting structures are freed, sparing the neurovascular bundles and motor branches from median and ulnar nerves. This usually requires releasing the palmar and dorsal fascial layers overlying and

encasing the adductor pollicis and the first dorsal interosseous. The origin of the transverse component of the adductor from the third metacarpal may need to be divided. The ensuing soft tissue defect will almost always be greater than anticipated.⁹¹

The reconstruction options vary depending on the degree of contracture, residual soft tissues and available local and remote donors. It is usual to have to use at least local flaps for web expansion, including single or multiple Z-plasties and transposition flaps from nearby digits. However, in choosing any of these local options it is important to remember that they are probably also within the zone of injury, and preoperative markings and flap options should incorporate previous scars.⁹²

In injuries where the webspace has been obliterated by tissue loss or ischaemia, the reconstructive options should include pedicled or free tissue transfers from distant sites. There are several routine flaps available for transfer available, including groin, lateral arm, posterior interosseus, first webspace of the foot, or really any area where expendable pliable thin soft tissue is available⁹³ (Figure 54.5). We routinely use free groin flaps for web reconstruction where the defect requires imported soft tissue. The donor site is hidden and the flap size can vary considerably, with the advantage of being able to thin the flap bulk at the time of transfer^{94,95} (Figure 54.6).

Postoperative care

The immediate postoperative care requires attention to the hand and patient. The thumb should be protected by either a bulky dressing or a splint. It is the application of the dressing which is most crucial. The dressing should be non-constricting at the time of application and also have the potential to remain so after any postoperative swelling or bleeding. In the free tissue transfers the patient should be kept warm and comfortable with a well-filled circulating blood volume to promote distal perfusion. These microvascular transfers should be monitored by experienced nursing staff and any concern about flap vascularity should be escalated. This allows for early exploration and flap salvage.⁹⁶

Rehabilitation and outcomes

Rehabilitation and hand therapy are the cornerstones for good outcome, as with all hand surgery. In adults hand therapy can commence early once the viability of the flap or transfer is confirmed and the operating surgeon is comfortable.

Good functional outcomes after thumb reconstruction are recorded in the literature.⁹⁷ Parvizi *et al.* have shown the functional benefit of thumb reconstruction. They have concluded that occupation and the patient's relative focus on appearance must be considered when evaluating the best options for reconstruction and the overall benefit to each individual.⁹⁸ Functional evaluation of thumb replantation or reconstruction with a great toe transfer are comparable, with Buncke *et al.* actually demonstrating better inter phalangeal joint motion in great toe transfers than in amputated thumbs that had been crushed or avulsed.⁹⁹



Figure 54.5 (a) Burn contracture of the first web space, thumb and index finger. (b, c, d) Lateral arm free flap to reconstruct the web space, release the thumb and index fingers.

Metacarpal hands

Mutilating hand injuries are rare but devastating. They are often associated with multidigit loss and destruction of the sensory tactile glabrous skin of the digits and palm. Michon first ascribed the term *metacarpal hand* to these injuries in 1974. He divided it into two broad groups:

- the complete loss of all digits,
- 'thumb without fingers'.

The outcome then, as now, is dependent on the sensory capacity of the remaining or reconstructed hand, as without reliable sensation it is unlikely that the hand will be used functionally or reliably.¹⁰⁰

Subsequent classifications by Wei *et al.* and Kotkansalo *et al.* have focused on more specific classification in relation to amputation level and reconstructive options for thumb and fingers.^{101,102} In the Wei classification, type I describes those hands where all the fingers have been amputated at the level of the middle of the proximal phalanx or more proximally, while the thumb is uninjured or amputated distal to the interphalangeal joint. In type II injuries the thumb is also amputated proximal to the level of the interphalangeal joint.¹⁰¹ The other salient feature in these injuries and with reconstruction is the state of the skin or soft tissue covering the remaining hand and wrist as this will also play a critical role in the level of functional recovery and use of the hand.

Management principles and options

These cases should be treated, as with all major traumas, in a systematic fashion with primary exclusion or treatment of all life-threatening associated injuries. This is pertinent, in



Figure 54.6 Free groin flap for dorsum hand and web space reconstruction.

particular with blast injuries, where trunk and other limbs are likely to be involved as well.

Specific treatment of the hand requires attention to preservation of skeletal structures, including joints, and the preservation neurovascular structures where possible to allow future reconstruction. It is important not to shorten or remove specialized tissue to enable closure. It is far better to preserve all viable tissue and digit length and cover with pedicled or free groin flap or other fasciocutaneous flap where possible. Soft tissue cover should be expedited and, if possible, should be achieved immediately. In cases where there is significant contamination or doubt exists about the viability of parts then it is acceptable to delay cover. Ideally, however, soft tissue cover should be achieved within 48 h to prevent further tissue loss from desiccation. It is important when achieving cover at this early stage to use pliable and padded soft tissue flaps rather than skin grafts. The latter will contract over time and will limit movement and constrict any remaining webspaces, thus compromising residual function further.

Once soft tissue cover has been achieved, there is then time to focus on reconstructive options available for thumb and digit reconstruction. It is also important to consider the webspace and pliability of the skin of the hand to ensure that thumb and digit motion is enabled as much as possible.

The psychological effects of these severe hand injuries should also be managed in parallel. Several studies have indicated the high incidence of posttraumatic stress disorder and the link between physical and psychological outcomes in this patient

population. It is important to address these psychosocial elements early to promote psychological wellbeing and this will aid physical recovery.^{103–105}

In type I injuries (Table 54.1), where the thumb is adequate then one can focus on reconstruction of the webspace and digits. In cases where the skin is preserved then the focus can remain entirely on establishing a competent sensate prehensile hand. This can be achieved using toe transfers to the 2–5 metacarpal rays. The location of the transfers should depend on the flexibility of the thumb and the competency of the webspace. Where the thumb is intact but is stiff or the soft tissues are not pliable, then it is best to transfer onto the radial digits so as to promote useful grip.

Where the thumb and soft tissues are pliable and the basal joint is competent then transferring the toes to the more ulnar rays can achieve both narrow pulp-to-pulp grip, along with large object grasp. Finger reconstruction can be achieved with second toe transfers or *en bloc* transfers of second and third toes. The options for reconstruction will be limited by the donor site that is acceptable to the patient and surgeon.

In type II injuries (Table 54.2), the thumb is also compromised and here the challenge is producing a hand that yields the most functional grips using the repertoire of toe transfers and lengthening procedures available. Reconstruction of the thumb takes priority over the other digits, and trying to achieve symmetry of thumb appearance to the contralateral side, if present, combined with the need to reconstruct multiple digits means the possibility of using the great toe as donor

Table 54.1 Classification of metacarpal hand type I

Subtype	Level of finger amputation	Recommendations
IA	Distal to the MCP joint	1. Bilateral 2nd toes for amputations distal to the webspace 2. Combined 2nd and 3rd toes for amputation proximal to the webspace (transproximal phalanx transfer)
IB	Through the MCP joint with intact metacarpal articular surface	Combined 2nd and 3rd toes (composite joint transfer)
IC	Through the MCP joint with damaged metacarpal articular surface or transmetacarpal amputation	Combined 2nd and 3rd toes (transmetatarsal transfer)

Source: Wei *et al.*, 1997.⁷⁰ Reproduced with permission of Lippincott Williams & Wilkins.
MCP, metacarpophalangeal.

Table 54.2 Classification of metacarpal hand type II

Subtype	Level of thumb amputation	Recommendations
IIA	Distal to the metacarpal neck	Whole or trimmed great toe transfer (transproximal phalanx transfer)
IIB	Proximal to the metacarpal neck with adequate thenar muscle function	1. Preliminary distraction lengthening or interpositional bone graft followed by whole or trimmed great toe transfer, or 2. Transmetatarsal 2nd toe transfer
IIC	Any level with inadequate thenar musculature	1. Thumb reconstruction in a second stage following finger reconstruction, and 2. Tendon transfer to restore opposition
IID	Any level with damaged carpometacarpal joint	Same as in IIA and IIB, but aiming at reconstructing immobile thumb post

Source: Wei *et al.*, 1997.⁷⁰ Reproduced with permission of Lippincott Williams & Wilkins.



Figure 54.7 (a) Metacarpal hand following burn injury – preoperative. (b) Postoperative view showing hand span after a free groin flap to create the webspace, a free toe transfer for thumb reconstruction and skin graft to release the remaining digits.

for the thumb with use of the second and/or third toes for reconstruction of the other digits. Contralateral ring finger transfers offer the advantage of length and mobility over multidigit transfers.¹⁰⁶

If the soft tissues have also been compromised or lost then these will need to be reconstructed as a first stage (Figure 54.7). It is important at this stage to adequately release any contractures of the webspace and to resurface any areas of contracted skin where vascular or neural pedicles will cross. This can be challenging, in particular if there have been burns or avulsions. Although a small amount of skin and soft tissue can be harvested with the toe transfer it is really important that any skin closures at the time of digit transfer are not tight or under tension to prevent compression and pedicle compromise.

At the time of toe transfer, in particular, in these cases of severe hand injury attention paid to pedicle length and pathway is critical. It is always worth documenting by illustration or photos the location of vascular and neural pedicles so that at times of secondary surgery a clear picture of these vital structures is available. The site of osteosynthesis should be measured before harvesting the toe to ensure appropriate length of tendons, nerves and the vascular pedicle. It is important to remember that in these multidigit reconstructions the overall hand length is shortened and thus anticipating the site of the reconstructed thumb pulp and pulp of the reconstructed digits is worthwhile.

Postoperative care

A multidisciplinary programme of postoperative care should commence immediately. This includes physiotherapy, occupational therapy, psychology and nursing care. In the

immediate postoperative time it is important to ensure a system of monitoring transfers in a regular and robust fashion and we have routinely used clinical observation.⁹⁶

Further surgery to adjust tendons, scars, soft tissue bulk and revise osteosyntheses is common in this complex patient group.^{102,107}

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CHAPTER 55

Compartment syndrome in the extremities

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Introduction

Compartment syndrome in either upper or lower extremity is often underestimated and may have detrimental consequences for functioning of the involved extremity. Muscle groups in the extremities are separated by fascial sheaths to ensure that muscles move in the right vector and achieve maximal efficiency during contraction. Fascial sheaths also provide additional surface area for muscle origins because bone alone does not offer enough surface area. Fascial sheaths protect neurovascular bundles.

Groups of muscles in the arms and legs, together with blood vessels and nerves, are contained in compartments surrounded by non-elastic fascia and/or bone; therefore, whenever bleeding and/or oedema occur, increased pressure is inevitable. This increased pressure can harm the intracompartmental soft tissues, leading to detrimental consequences. Focusing on the different compartments in both the upper and lower extremities a distinct architecture can be recognized.

In this chapter we address the anatomy of both upper and lower extremities to illustrate the rationale for the occurrence of compartment syndrome, its consequences and management strategies.

Anatomy

Fascia

The thickness of the fascia is determined by the amount and direction of the applied force. Comparing the fascia of the lower leg (i.e. fascia cruris) to the fascia of the forearm (i.e. fascia anti-brachii), not only can a significant difference in thickness be noted, favouring the fascia cruris, but also a difference in the direction of the fascia fibres. The fascia cruris fibres run in a circular direction, whereas the fibres of the fascia anti-brachii do not have a uniform shape or direction. This difference can be explained by the constant contraction and relaxation of the calf muscles, which is of much higher amplitude than that of their forearm counterparts.

Upper extremity

Forearm

The forearm contains three compartments: dorsal, volar and mobile wad compartments. The *dorsal compartment* contains the abductor pollicis, extensor pollicis longus, extensor carpi ulnaris, extensor digiti minimi and extensor digiti communis muscles. Inferior to the muscles, the radius and ulna can be found, separated by the interosseous membrane. The *volar compartment* is separated into superficial and deep muscles. The deep muscles include the flexor pollicis longus, flexor digitorum profundus and pronator quadratus. The superficial muscles are the palmaris longus, pronator teres, flexor digitorum superficialis, flexor carpi ulnaris and radialis. The ulnar and radial bone and the interosseous membrane border the compartment dorsally. Volar borders are the subcutaneous antebrachial fascia. This fascia also separates the volar compartment from the *lateral compartment* (i.e. the *mobile wad*), which holds the brachioradialis, extensor carpi radialis brevis and longus muscles.

Hand

Looking at the different osteofascial compartments of the hand, 10 separate compartments can be distinguished: four dorsal interossei compartments, three palmar interossei compartments, the adductor pollicis compartment, and the thenar as well as hypothenar compartments.

Lower extremity

Lower leg

Anatomically, the lower leg can be divided in four compartments: the deep and superficial posterior compartments and the lateral and anterior compartments.

The fascia cruris, tibia, fibula and interosseous membrane surround the anterior compartment. The anterior tibial muscle, extensor hallucis longus and extensor digitorum longus are the muscles in this compartment. The anterior tibial artery and veins run along these muscles distally and the deep peroneal nerve innervates them.

The lateral compartment comprises the long peroneal and brevis muscles and the common peroneal nerve proximally. The crural fascia, intermuscular septum fascia and fibula compartmentalize these structures.

The superficial posterior compartment is separated from the deep compartment by the transverse intermuscular septum and is ensheathed by the fascia cruris. This compartment holds space for the plantar, soleus and gastrocnemius muscles.

The deep posterior compartment is encircled by the already mentioned transverse septum, the tibial and fibular bones and the interosseus membrane; it also contains the tibial nerve and posterior tibial and peroneal arteries and veins. Muscles that are located in this compartment are the posterior tibial, flexor hallucis longus and flexor digitorum longus muscles.

Foot

Three compartments cover the full length of the foot: medial, lateral and superficial compartments. In addition, five intrinsic compartments are described (i.e. four interossei compartments and an adductor compartment) and a calcaneal compartment.

The medial compartment holds the flexor hallucis brevis and the abductor hallucis muscle. The lateral compartment contains the abductor digiti quinti and the flexor digiti minimi muscles. In the superficial compartment the flexor digitorum brevis, the flexor digitorum longus tendons and the lumbrical muscles are located. Also, the medial plantar nerve is running through this compartment. The interossei and adductor compartments hold the interossei muscles and the adductor muscle, respectively. The calcaneal compartment not only comprises the quadratus plantae muscle, but also the posterior tibial, medial and lateral plantar nerves. All vessels of the foot run through this compartment: the posterior tibial, medial and lateral plantar arteries and veins.

Pathophysiology

Each compartment has an ideal pressure for optimal tissue perfusion, based on the balance between the hydrostatic and osmotic pressure. The intracapillary hydrostatic pressure normally varies between 20 and 30 mmHg and relates to the difference between venous and arterial pressures.¹ The osmotic pressure or interstitial fluid pressure ensures prevention of outflow and ranges between -5 and +5 mmHg.²

Disturbance of this pressure balance can occur as a result of an increase of intracompartmental pressure (ICP) or a decrease of compartmental volume caused by bleeding or swelling of the tissue or a reduction of external volume due to high-pressure compression or casting. It is generally accepted that an increase of the interstitial fluid pressure above the intraluminal pressure of the capillary will prohibit adequate perfusion of the intracompartmental structures.³

As soon as the perfusion is jeopardized, hypoxia will lead to oedema and the initiation of anaerobic metabolism. This

cascade leads to a further increase of ICP and thus an even greater decrease of capillary flow. Subsequently, only an intervention can halt this process. If intervention is not performed within 3 h after the onset of ischaemia, an impaired muscle response will occur. Six to eight hours after ischaemia the muscle and soft tissue will have suffered irreversible damage.⁴

Diagnosis

Subjective

Compartment syndrome is a clinical challenge where the clinical history foretells the physician the problem. Classically pain, pallor, pulselessness, paraesthesias and paralysis (also called 'the five P's') are described as the clinical symptoms related to compartment syndrome. Although pain and loss of sensibility are not universal findings,⁴ in our survey only one of our patients was asymptomatic (see below ischaemic contracture). In all other patients symptoms were present but unfortunately not adequately recognized or treated.

The review by Wall *et al.* confirmed 'the five P's', except their observation that pulses may be present. Moreover, the pain may be out of proportion to the injury and there is a palpable painful tenseness or swelling of the compartment.⁵ Also important is pain on passive stretch of wrist and fingers in the developing compartment syndrome of the upper extremity. Progressively, the increased pressure will lead to ischaemia of the nerves, resulting in late-onset clinical signs as sensory and motor deficits.

In the upper extremity a supracondylar fracture of the humerus is the most frequent cause for the development of an ischaemic contracture, followed by a fracture of the forearm. These fractures should be monitored carefully with regard to compartment syndrome, especially in children.⁶

It is important to note that compartment syndrome is often missed or discovered later because of modern early treatment with drugs to diminish pain. In most modern hospitals it is supposed to be a medical mistake if patients have pain. At our unit and in our hospital compartment syndromes have been missed for this reason.

Objective

Intracompartmental pressure

An objective measurement for the diagnosis of compartment syndrome is evaluation of ICP. Aside from the decision of evaluating pressure, one should perform a fasciotomy immediately if there is any doubt about compartment syndrome. In our medical practice, working in a trauma referral centre dealing with substantial injuries to the extremities, it is common practice to perform preventive open fasciotomies, especially if vascularization is compromised more proximally.

Many authors have described the assessment of pressure monitoring for the diagnosis of compartment syndrome. Some

of the techniques described are the side-portal needle, the slit catheter,⁷ the Whitesides' infusion technique⁸ and a regular 18-gauge needle.

All techniques are based on the same concept, which is in fact an intracompartmental placement of a specific measuring device (i.e. the subtle difference between the different techniques), a fluid column and a transducer for measurement of ICP. As such, either a continuous or a repeated ICP measurement can be established. Ideally, the same clinician monitors the ICP with an interval range ranging from 30 min to 12 times a day.⁶

A recent experimental study by Hammerberg *et al.* compared the reliability of different devices, both digital and analogue, using three different terminal devices.⁹ The devices investigated were the 18-gauge bevel-tipped needles, the side-ported bevel-tipped needles and the slit catheters. They concluded that the differences between these three terminal devices are small and unlikely to have an impact on clinical decision making for fasciotomy. However, when the electronic measurement approach is compared with the analogue Whitesides' technique, the electronic technique is favoured because it does not require prior preparation. Nevertheless, Hammerberg *et al.* recommended using the slit catheter for continuous monitoring of one compartment. In the event, multiple compartments are at risk or multiple measurements in the same compartment are required. The clinician should use either a standard 18-gauge or side-port bevel-tipped 18-gauge needle.⁹

Near-infrared spectroscopy

Near-infrared spectroscopy uses a differential light absorption to measure the level of haemoglobin, thus providing a measure of oxygenation. Recognizing decreased muscle oxygenation is correlated to the intracompartmental tissue perfusion gradient and thus could be helpful in the diagnosis of acute compartment syndrome.¹⁰ Furthermore, Reisman *et al.* showed an inverse correlation between tissue perfusion and ICP. They noticed an 8% difference, with the perfusion dropping below 10 mmHg.¹¹ As near-infrared spectroscopy can scan at different depths intracompartmentally, it seems to be useful for monitoring patients at risk of compartment syndrome.

Treatment

If a compartment syndrome is not adequately released, necrosis with all its consequences will be unavoidable. In the presence of the aforementioned symptoms our advice is to act with aggressive decompression of compartments without delay. Surgery is the only treatment that can prevent the disastrous consequences of compartment syndrome by restoring the microcirculation and preventing further damage to the muscles and nerves. Measures such as opening of the plaster cast and elevation of the extremity are not sufficient in these cases. Sheridan and Matsen found that the frequency and severity of complications of decompression were directly related to the promptness with

which it was performed.¹² In cases of a suspected vascular injury, decompression has to be combined with vascular exploration and restoration of blood flow. At our unit most decompressions are performed preventively. This is especially the case in crush injuries of the extremities, and particularly if vascularization of the involved extremity is at risk. We do not have a wait-and-see policy and thus do not use this objective evaluation on a regular basis. Table 55.1 provides an overview of fasciotomies carried out in our unit between 2002 and 2012 in order to prevent compartment syndrome or further damage due to ischaemia in cases of compartment syndrome. This table illustrates our policy not to wait and see.

Upper extremity

Forearm

When addressing compartment syndrome, the superficial and deep flexor compartments, the dorsal compartment and mobile wad in the forearm, the carpal tunnel and the intrinsic compartments should be released. The most affected muscles in compartment syndrome of the forearm are the deep muscles in the volar compartment. Different approaches have been reported to release the compartments. They all share, however, the same principle of placing a long longitudinal incision over the forearm. Ronel *et al.* investigated the best approach for releasing the compartments of the forearm.¹³

To open the volar compartment, a radial approach was described. An incision is made radial to the volar midline, starting at the proximal wrist crease and extended to the antecubital fossa. In this way the superficial fascia can be seen and opened to enter the deep muscles radial to the flexor carpi radialis.

Central and ulnar approaches were also investigated. For the central approach the volar incision is planned in the midline, the fascia is opened and, approaching either ulnar or radial to the palmaris longus, the deep muscles can be inspected. The ulnar approach requires an incision radial to the flexor carpi ulnaris. The dorsal compartment is opened through a midline incision in the dorsum of the arm. After opening the superficial fascia the compartment should be adequately decompressed.

Gelberman *et al.* described incisions that were prolonged over the carpal tunnel and introduced a curved incision for easy access to the muscles without harming the brachial artery or major nerves.¹⁴ The senior author uses a similar approach as described by Gelberman.

Hand and wrist

A well-executed study by DiFelice *et al.* illustrated the compartments of the hand and proposed a protocol for releasing them.¹⁵ Placing two incisions on the dorsum of the hand – one on the thenar region and one on the hypothenar region – allows access to all compartments. Another incision over the carpal tunnel allows release of the carpal tunnel as well as Guyon's canal.

Regarding the dorsal incisions, the incisions should be made over the second and fourth metacarpals. The incision over the

Table 55.1 Overview of fasciotomies in upper and lower extremities in the preventive (i.e. primary) trauma setting and with evident symptoms of compartment syndrome to prevent permanent damage due to ischaemia (secondary), Department of Plastic & Reconstructive Surgery and Hand Surgery, Erasmus MC, University Medical Center, October 2002–October 2012

Upper extremity (n = 231)							
Primary	n = 186	CTR	GR	IR	HHT	DF	VF
	Crush (76)	76	57	54	33	40	41
	Multitrauma (44)	41	24	26	17	16	17
	Deglovement injury (24)	21	10	13	8	6	5
	Glass/stab injury (31)	31	11	2	1	5	7
	Burn injury (6)	6	5	6	5	4	4
	Circular saw injury (5)	5	2	0	0	0	0
Secondary	n = 45						
	Extravasation (12)	12	9	6	5	8	10
	Postoperative bleeding (20)	18	9	6	1	6	7
	Infection (13)	11	6	6	6	6	6
Lower extremity (n = 48)							
Primary	n = 33	Anterior compartment	Superior posterior compartment	Deep posterior compartment	Lateral compartment	Foot	Upper leg
	Crush (15)	9	8	8	9	9	
	Deglovement injury (13)	5	5	5	5	8	
	Fracture (5)	3	3	3	3	3	1
Secondary	n = 15						
	Extravasation (4)	1	1	2	2	4	
	Postoperative bleeding (5)	1	1	1	1	3	1
	(Postoperative) swelling (5)	5	1	1	1	3	1
	Infection (1)	1	1	1	1	1	

CTR, carpal tunnel release; GR, Guyon release; IR, intrinsic release; HHT, hypo- and hyperthenar release; DF, dorsal forearm release; VF, ventral forearm release.

Data extracted from the surgery records from the Department of Plastic & Reconstructive Surgery and Hand Surgery of the Erasmus MC, University Medical Center, October 2002–October 2012.

second metacarpal creates access to the first dorsal interosseous muscle, the adductor pollicis, the second dorsal interosseous and the second palmar interosseous. In contrast to our experience, DiFelice advises release of the interossei from its origin (i.e. the connection to the second metacarpal bone) as well. The adductor pollicis fascia can be found radial and inferior to the first dorsal interosseous. The incision over the fourth metacarpal logically gives access to the fourth and third interosseous compartments using a similar approach to that described over the second metacarpal.

To release the hypothenar fascia a longitudinal incision should be placed just ulnar to the fifth finger. This will prevent harming the motor nerve bundles. The thenar incision should be parallel to the first metacarpal bone. Be careful not to lacerate the motor bundles innervating the thenar muscle.

To open the carpal tunnel, the well-accepted longitudinal incision is placed at the centre of the tip of the fourth metacarpal in full flexion, running distal towards the flexing point of the wrist. The flexor retinaculum is the roof of the carpal tunnel and the floor of Guyon's channel. After opening the skin, the retinaculum is followed ulnar to the point where the ulnar nerve is

visualized. Subsequently, the tendon sheath between the os pisiforme and os hamatum can be opened to release Guyon's channel. To release the carpal tunnel the flexor retinaculum can be opened.

The senior author opens the thenar and hypothenar sheaths via the carpal tunnel incision and not via separate incisions, therefore using only three incisions – two dorsal for the interosseous muscles and one volar in the hand – to adequately release the compartments.

Lower extremity

Lower leg

Exposure of all four compartments is required to prevent (further) necrosis in the acute compartment syndrome, including the tarsal tunnel and compartments in the foot.

In the literature, two techniques used to release the compartments in the crural area are frequently described.¹⁶ Either a single lateral incision can be made or a two-step anterolateral and posteromedial approach can be used. The first author favours the two-step approach and therefore we choose to discuss this technique in detail.

Two-step anterolateral and posteromedial approach

The first incision is anterolateral, which allows exposure of the anterior and lateral compartments. The incision should be placed over the anterior intermuscular septum. The fascia is then opened and, moving laterally, the lateral compartment can be decompressed. Be aware of the superficial peroneal nerve, which exits the lateral compartment 10 cm cephalad from the lateral malleolus. Decompressing the anterior compartment the fascia should be opened medial from the intermuscular septum. The anterior tibial and extensor hallucis longus muscles protect the deep peroneal nerve and peroneal artery. This paradigm, as described by Kashuk *et al.*, allows an easy and safe dissection.¹⁷

The posteromedial incision is placed medial from the tibia and over a length of 15 cm sufficient exposure is created for a safe decompression. Some promote extending the incision with a medial retromalleolar incision, which evidently will release the tarsal tunnel.¹⁸ After placement of the first incision, the fascia can be opened and anterior retraction will prevent laceration of the saphenous vein and both the saphenous nerve and vein. After the superficial compartment is decompressed, approaching the insertion of the soleus muscle can expose the deep compartment. Retraction of this muscle exposes the fascia of the long flexor digitorum, which can then be opened. It is important to identify the neurovascular bundle (i.e. posterior tibial nerve and artery), which is located between the soleus and anterior tibial muscle.

Recommendation

The posteromedial incision is designed to create a safer decompression of the deep and superficial posterior compartments. In our practice, the two-step incision technique is mostly used to prevent inadequate deep flexor compartment release and unnecessary damage, as this technique opens the compartments under direct vision. This is especially important in a training institution.

Foot

For the foot, various techniques have been described and a nice overview is given by Frink *et al.* in which the dorsal fasciotomy is promoted.^{16,19} This approach uses two incisions medial to the second metatarsal bone and lateral to the fourth metatarsal bone. Taking care of the superficial veins and nerves, the fascia can be located and should be opened longitudinally over each interosseous compartment. Subsequently, the medial compartment is decompressed with an incision parallel to the plantar surface, starting just caudal to the insertion of the Achilles tendon (i.e. tarsal tunnel). The incision exposes the medial and abductor compartment. Lifting the abductor compartment opens the calcaneal compartment. The superficial and lateral compartments can be opened, consequently, through the calcaneal compartment. The superficial compartment is found just lateral to the medial compartment and after opening the medial fascia longitudinally, the flexor digitorum brevis can be retracted inferior and the medial fascia of the lateral compartment exposed.

Our approach to foot compartmental release is to use two dorsal incisions for the dorsal compartments. The incision over the tarsal tunnel is further lengthened distally on the medial side parallel to the plantar fascia to open the abductor, calcaneal, superficial and lateral compartments of the foot.

Late sequelae

Volkmann's contracture of the upper extremity

The classic case of a patient with an ischaemic Volkmann's contracture is a child sustaining a supracondylar humeral fracture treated with a cast. The following days the child is in considerable pain and although the cast is changed the pain is not relieved. Subsequently the fingers start clawing gradually and the wrist is no longer able to extend. Passively the fingers can only be stretched when the wrist is flexed completely. Furthermore the sensibility of the hand is severely impaired.

In 1881, Richard von Volkmann described a condition of muscle ischaemia, necrosis and subsequent contracture of the forearm. This condition was the result of diminished arterial blood supply due to the application of tight, constricting bandages to the injured arm.²⁰ In 1908, J.J. Thomas collected 107 cases with ischaemic contracture from the literature. He reported that contractures were also possible in patients without treatment with splints or bandages; extrinsic pressure could therefore not be the only cause of Volkmann's contracture.²¹ The current understanding is that various injuries can cause swelling of the soft tissues in relatively tight osseofascial compartments.²² The deep flexor compartment of the forearm is the first to be damaged as it closest to the bone, with a tight surrounding fascia.²³ Also the flexor digitorum profundus is most distant from the arterial anastomotic pathways available to re-establish circulation. The centre of the muscle, providing tendons to the middle and ring fingers, is particularly vulnerable.²⁴

In more severe cases or when the trauma is more in the vicinity of other compartments, the infarction can also encompass the superficial flexor muscles, the extensor muscles and the intrinsic hand musculature. When ischaemia is prolonged, the muscles become necrotic and are eventually replaced by fibrous tissue and fat. In addition, the initial ischaemia can cause nerve damage, which becomes worse when the surrounding muscle becomes fibrotic.

If compartment syndrome is not treated adequately, Volkmann's ischaemic contracture will occur (Figure 55.1). As our understanding of the pathogenesis of compartment syndrome has improved over the years, more specific diagnostic and therapeutic measures have been introduced, reducing the incidence of Volkmann's ischaemic contracture.^{26–30}

Unfortunately ischaemic contracture still occurs as a result of an underestimation of the symptoms, with serious consequences for the function of the involved extremity. Not only is a fully developed contracture of the flexor muscles with impaired sensibility in the forearm extremely incapacitating, it can also cause



Figure 55.1 This 4-year-old girl sustained a supracondylar fracture (six months earlier) resulting in a severe Volkmann's ischaemic contracture with proximal interphalangeal contractures and loss of motion in fingers and wrist as well as sensibility loss in both ulnar and median nerve distribution. Source: Hovius & Ultee, 2000.²⁵ Reproduced with permission of Elsevier.

psychological trauma. In the United States, failure to diagnose an acute compartment syndrome is a well-known cause of litigation against the medical profession.³¹

An established contracture varies from a few affected deep flexor muscles with no nerve damage to a complete infarction of one or more muscle compartments with extensive nerve damage. Treatment should therefore be custom-made and directed to improve overall function. Although several methods of treatment have been reported, long-term results in different treatment regimens are rarely described.^{28,32–34}

In our study we analysed our cases (25 patients) and reported on the long-term results of the different treatment methods for Volkmann's contractures.³⁵ Patients filled in questionnaires concerning their disability, together with their satisfaction with the treatment. They were examined physically, including arm length, and tests were performed to measure

strength and sensibility. In addition, motor function was classified based on Buck Gramcko's classification. Most patients sustained supracondylar fractures and the mean age was 13 (range 2–60) years. Follow-up had a mean of 7 (range 3–29) years. Post-trauma severe pain was encountered in 88%, loss of sensation 80%, blue fingers 60% and skin necrosis in 28%. In the majority, sensibility, motion and strength improved in the first months. In five patients sensibility did not improve.

Treatment varied, ranging from conservative splinting through excision of fibrous tissue, neurolysis, tenolysis, proximal 'muscle slide', tendon lengthening, tendon transfers and nerve grafting to free vascularized, innervated musculocutaneous flaps.

The treatment of an established contracture depends strongly on the severity of the infarction and the affected muscle and nerve tissue. Decisions about when and how to operate are not easy and are often discussed in literature.

Based on our study, we operate on patients with poor or insufficient hand function, with severe muscle necrosis diagnosed by MRI and with neurological signs in an earlier stage to prevent further joint stiffness and nerve damage by fibrosis.^{23,33,35,36} Severe muscle necrosis is easily differentiated from viable muscle tissue at an early stage, and can subsequently be removed. In patients with a so-called 'prolonged pain syndrome' we strongly advise early neurolysis and excision of fibrotic tissue, as in our experience it will immediately resolve the pain.³⁵

In patients with good to reasonable hand function we suggest initial conservative treatment with hand therapy and splinting while waiting for spontaneous improvement of sensibility and motor function. If after three months hand function does not improve or diminishes, surgical intervention has to be considered.

In patients with insufficient hand function with infarction of a considerable part of the deep and superficial flexors and neurological signs, excision of fibrotic muscle tissue, neurolysis and tenolysis is advised.³⁵ Good results can be obtained if excision and neurolysis are combined with tendon transfer, specifically in the early cases. We do not recommend tendon lengthening only, due to the high recurrence rate of the contractures and possible loss of power grip strength.

In patients with poor hand function with extensive infarction diagnosed by MRI, we advise a free vascularized innervated muscle transfer. We prefer a two-stage procedure with early excision in the first stage followed by the flap and if necessary a nerve graft in the second stage a few months later. If in the first stage the nerve is in continuity without deformity, a neurolysis is performed. In our experience quite often a substantial recovery of sensory function will occur in these cases. If in the first stage the nerve is deformed, a nerve graft is planned in the second stage, combined with the free muscle transfer (Figure 55.2). The nerve is mostly deformed when a substantial number of the forearm flexor muscles are fibrotic. In the majority of our patients both hand mobility and sensory function improved

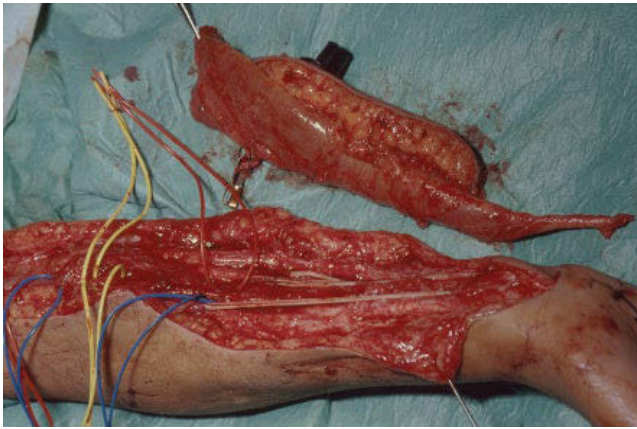


Figure 55.2 Second phase: median and ulnar nerve reconstruction with sural cable grafts and a free vascularized innervated gracilis flap for flexor function (same patient as Figure 55.1). In the first phase scar tissue excision, neurolysis and flexor tenotomy was performed. Source: Hovius & Ultee, 2000.²⁵ Reproduced with permission of Elsevier.

tremendously after free muscle transfer (Figure 55.3). Although the contractile force of the gracilis muscle will stay limited for grip strength, a considerable increase of power grip strength was noticed in our cases.

We have described the considerable difference in length of the forearm and hand on the affected side in relation to the healthy side in our patients who still had open growth plates.³⁵ Reasons for this length difference might be impaired oxygenation of growth plates and impaired muscle function and damaged nerve tissue. We therefore recommend informing children and/or parents or caregivers about the possibility of a length difference of the forearm developing when they have an ischaemic contracture of the forearm.

Volkmann's contracture of the lower leg

Zwipp discussed the late sequelae after compartment syndrome in the lower leg and foot.³⁷ The contractures can be subdivided into five groups. Group I consists of small toe contractures, including hammertoes (i.e. intrinsic muscle necrosis and contractures of the m. flexor brevis) and claw toes (ischaemic damage of the m. flexor brevis and flexor digitorum longus). Group II comprises the pathology of the hallux, which can be either isolated muscle necrosis of the flexor hallucis longus (resulting in a claw deformity during knee bending) or a contracture of the flexor hallucis longus. Group III is the combination deformity of groups I and II. Group IV is the pes equinus deformity based on the contractures of the superficial dorsal compartment (without nerve dysfunction) or based on dysfunction of the anterior compartment (either muscle necrosis or nerve paralysis). Group V is the pes equinovarus deformity, which is caused by the tibial, peroneal or combined ischaemic nerve injury.

There is little information about the surgical options in the literature. The group of Hansen proposed scarred muscle



Figure 55.3 Extension (a) and flexion (b) of fingers following free vascularized innervated flap 2 years postoperatively (same patient as Figure 55.1). Source: Hovius & Ultee, 2000.²⁵ Reproduced with permission of Elsevier.

excision (including the respective tendon),³⁸ but the recent work of Zwipp *et al.* promotes a percutaneous release of tendons by Z-plasties, which can adequately correct the contracted toes and/or foot. In the experience of the first author most cases have sustained an ischaemic event of the anterior compartment with or without the lateral compartment. The lack of extension of the forefoot and toes is corrected by tendon transfers if possible, such as the transfer of the viable posterior tibial tendon to create either extension of the forefoot or extension of the toes. Other examples we have encountered include contracture of the toes following extension at the ankle joint. Release of fibrosis and tendon transfers is mostly performed in these cases.

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CHAPTER 56

Nerve injury, repair and reconstruction

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Introduction

The future of peripheral nerve surgery holds exciting possibilities. The advancements in the last 25 years have included an understanding of the internal neural topography facilitating the development of nerve transfers and neural substitutes for short-gap/small-diameter injuries. Despite these innovations, the major limitation to peripheral nerve recovery is still the time it takes for the nerve to regenerate. This remains true regardless of the technique used for the repair. Without prompt motor nerve input, denervated muscle after a prolonged period of time becomes resistant to nerve regeneration. Although avoidance of tension at the neurorrhaphy site and improvements in suturing techniques have improved the process to some extent, the ultimate success is dependent on innervation time, number of neurorrhaphy sites, supply and type of donor nerves, and the condition of the surrounding tissue. At every coaptation site, a percentage of nerve fibres are lost.

Tension is harmful to a repair site and, in the case of a large gap, a nerve graft is often used to fill in the deficit. Autologous nerve grafts are limited and the sural nerve remains one of the most common sources. Alternatives to standard treatment for short, small-diameter nerve gaps include commercially available nerve conduits and acellular human processed nerve allografts. Schwann cell-lined nerve conduits and tissue-engineered substitutions are still experimental, but may offer the solution for enhanced nerve regeneration in the future. It is important, however, to remember that even the 'gold standard' nerve autograft gives functional results typically deserving of a 'bronze' award, and no nerve substitute to date equals the results of an autograft. Nerve transfer techniques have largely replaced nerve grafts for many proximal level nerve injuries in our practice.

Types of nerve injury

Nerve injury

Peripheral nerve injuries can be classified in several ways. Sir Herbert Seddon's classification system was the first and was based on gross and histologic anatomical changes rather than the mechanism of injury.¹ He described three types of nerve injuries: (a) *Neurapraxia* involves a local conduction block at a discrete area along the course of the nerve. Wallerian degeneration does not occur and the recovery is excellent within a 'short' time period. (b) *Axonotmesis* implies axonal damage, while (c) *neurotmesis* is the equivalent of a peripheral nerve transection. In both axonotmesis and neurotmesis, Wallerian degeneration occurs distal to the site of injury. Although the former will recover, the latter cannot.

Within axonotmesis, Seddon described a degree of injury associated with scarring and less complete recovery. Within neurotmesis, he described an 'in continuity' total scar and a non-recovery injury. However it was Sunderland who emphasized Seddon's expanded description of axonotmesis and neurotmesis, with a classification that emphasized five degrees of nerve injury.² Mackinnon later emphasized a sixth-degree injury that could include normal fascicles with various degrees of injury to other fascicles (Table 56.1 and Figure 56.1).^{3,4} First-degree (neurapraxia) and second-degree (axonotmesis) injuries recover spontaneously, the latter at the classic rate of 1–1.5 mm/day or 1 inch/month (2.5 cm/month).⁵ Fourth- and fifth-degree injuries do not recover, while third- and sixth-degree injuries have partial recovery for different reasons. The difficulty associated with surgical correction of a fourth-degree injury is limiting the repair to the fascicles affected by fourth- and fifth-degree damage and not damaging fascicles normal fascicles with the potential for spontaneous recovery. The sixth-degree injury is the surgeons most technically challenging injury. We also use the simpler classification of 'favourable' and

Table 56.1 Classification of nerve injury

Mackinnon	Sunderland	Seddon	Injury	Recovery	
				Outcome	Rate
Degree I	Degree I	Neurapraxia	Conduction block resolves spontaneously	Favourable	Fast/excellent
Degree II	Degree II	Axonotmesis	Axonal rupture without interruption of the basal lamina tubes	Favorable	Slow/excellent
Degree III	Degree III		Rupture of both axons and basal lamina tubes, some scar	Favourable	slow/incomplete
Degree IV	Degree IV		Complete scar block	Unfavourable	None
Degree V	Degree V	Neurotmesis	Complete transection	Unfavourable	None
Degree VI			Combination of I–V ± normal fascicles	Mixed	Mixed

Source: Weber et al. Mackinnon Repair and grafting of peripheral nerve. In Gurtner & Neligan (eds), Plastic Surgery Volume 1: Principles; Elsevier, 2013. Reproduced with permission of Elsevier.

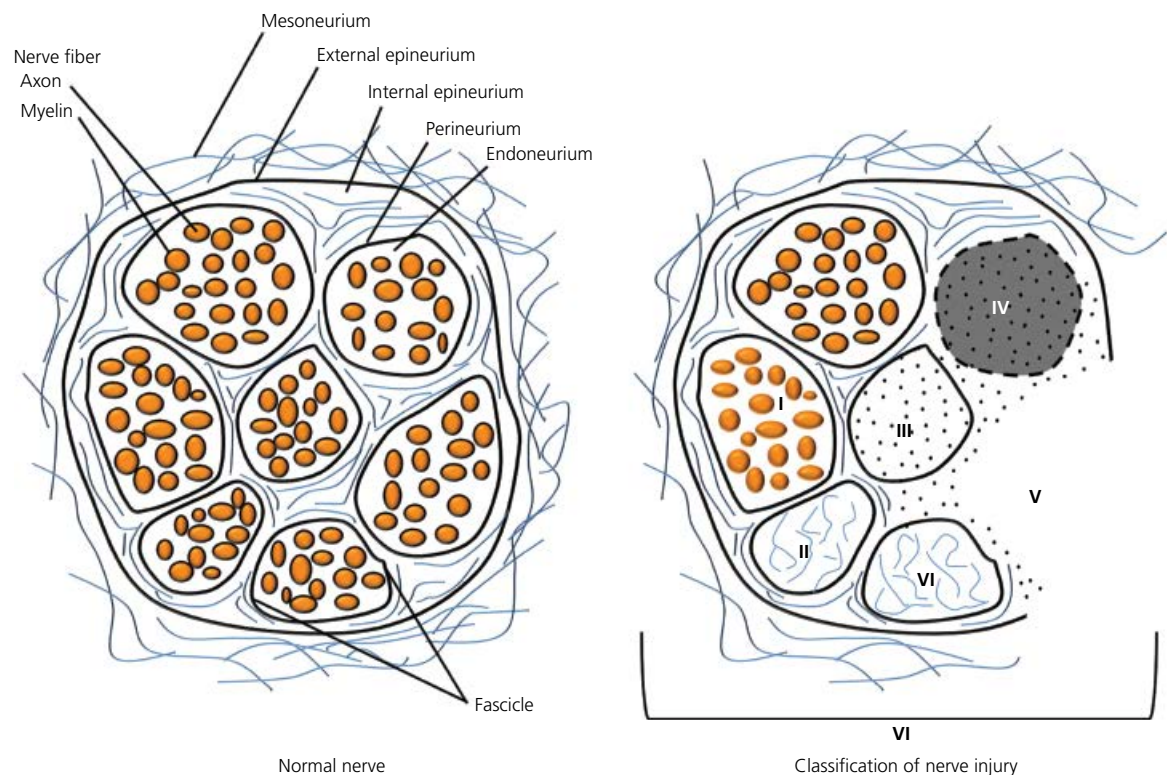


Figure 56.1 Schematic representation of the cross-section of a normal peripheral nerve showing the connective tissue and nerve tissue components. The cross-section of the peripheral nerve on the right demonstrates a mixed, or sixth-degree, injury pattern. The fascicle at the top left is normal. Moving in a counter-clockwise direction, fascicle I is a first-degree injury (neurapraxia) with segmental demyelination. Fascicle II is a second-degree injury (axonotmesis). The second degree involves both the axon and the myelin. The endoneurial tissue is not damaged. Fascicle III demonstrates a third-degree injury, with injury to the axon, myelin and endoneurium. The perineurium is intact and normal. Fascicle IV demonstrates a fourth-degree injury, with injury to the axon, myelin, endoneurium and perineurium. The fascicle is marked by scarring across the nerve, with only the epineurium being intact. Fascicle V is a fifth-degree injury in which the nerve is not in continuity and is transected. The surgeon will separate the fourth- and fifth-degree injury patterns, which will require reconstruction from the normal fascicles and the fascicles demonstrating first-, second- and third-degree injury patterns. These latter patterns of injury require, at most, neurolysis. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

‘unfavourable’ injuries. Favourable injuries recover fully and quickly (neurapraxia, first degree) or slowly (axonotmesis, second degree) and injuries that partially recover slowly (axonotmesis, third degree). ‘Unfavourable’ injuries will not recover (neurotmesis, fourth and fifth degree).

Nerve injuries may alternatively be classified by mechanism of injury and by open versus closed injury. This clinical

classification is useful in determining the likelihood for spontaneous recovery, and provides an algorithm for managing these injuries. Table 56.2 lists the typical nerve injury patterns and their significance with respect to assessment and treatment. For simplicity, nerve injuries can be grouped as: (a) stretch and avulsion injuries; (b) crush and compression injuries; and (c) penetrating injuries. Although generally, avulsion and crush

Table 56.2 Classification of nerve injury by mechanism

Type	May be injured	Significance
Stretch/avulsion injury		
Avulsion	Nerve roots Nerves exiting Foramen, bony fracture	Unable to be repaired primarily Indication for nerve transfer
Stretch	Any nerve	Mixed nerve injury (degree VI)
Crush and compression injury		
Complex crush	Skin, subcutaneous tissue, muscle, nerve $\pm$ bone	Varying degree of depth, loss of function is related to amount of tissue destruction
Chronic compression	Nerve	Slow onset, reversible
Acute compression	Nerve $\pm$ muscle	Quick onset, reversible muscle ischaemia, variable recovery of both muscle and nerve
Compartment syndrome	Nerve + muscle	Quick onset, reversible muscle ischaemia if ischaemia less than 6 h; no or variable recovery of muscle if released after 6 h
Penetrating injury		
Sharp	Skin, subcutaneous tissue, muscle, nerve $\pm$ bone. All levels	Needs surgical exploration because of high probability for nerve disruption
Blunt	Variable	Injury may extend further than expected
Blast	Variable	Injury pattern depends on ballistic makeup and velocity
Electrical	Variable	Neuropathy is from damage to myelin sheath and ranges from neuropathy to causalgia

Source: Weber et al. Mackinnon Repair and grafting of peripheral nerve. In Gurtner & Neligan (eds), Plastic Surgery Volume 1: Principles; Elsevier, 2013. Reproduced with permission of Elsevier.

injuries tend to be closed injuries, they may be open. When the skin is violated, a significant force was exerted to the body and thus it is not unusual to have associated soft tissue, vascular and orthopaedic injuries. The extent of soft tissue loss, especially muscle and skin, will ultimately influence the outcome of the nerve repair. More extensive soft tissue damage, with or without concomitant bony injuries, raises the complexity and the likelihood for a staged reconstruction. Often, the nerve reconstruction is and should be delayed in light of more acute vascular and orthopaedic injuries.

Traction and avulsion injuries

Stretch or traction injuries to a nerve occur when the strain exerted on a particular nerve exceeds the maximum limit that nerve diameter can accept. The internal structure of the nerve becomes injured, usually without any appreciable external evidence of injury. High-velocity or high-impact injury can often lead to an avulsion of the nerve or nerve root. Soft-tissue attachments around joint or bony foramina act as tethering points along the nerve's path. If arteries and veins run in parallel to the nerves, which is often the case, a severe injury can compromise the circulation. Unless the surgeon involved is skilled at vascular as well as nerve and tendon repair, the nerve injuries are often ignored while the trauma team deals with the limb-threatening injury. Even at major medical centres with unlimited resources and personnel it is rare to see a multi-specialty surgery occur in the emergency setting. The nerve injury is usually addressed at a later date.

Nerve injuries in which the proximal nerve is accessible are repaired directly or with a graft. When warranted, nerve transfer

and possible tendon transfers can be performed to complement and augment the nerve reconstruction.⁶

Nerves avulsed distally at the neuromuscular junction present a different problem, as there is no distal end unavailable for repair. For motor nerves, the proximal nerve can be directly implanted into the muscle belly. Some studies show as good as M4 motor recovery 1–2 years after direct nerve-to-muscle neurotization;⁷ however, experimental studies do not support these findings; rather, recovery is much less than a nerve coaptation would produce⁸ and we rarely use this technique in preference to tendons or muscle transfers.

Crush injuries

The most common peripheral nerve injury in the extremities is from external forces crushing the tissue. In the case of light compressive forces, a self-limiting neurapraxia occurs, but with increased force, the higher the probability of irreparable damage. In addition to external force, increased internal pressure from a haematoma, fracture and local tissue oedema may complicate matters. Compartment syndrome is the ultimate extreme of the spectrum and constitutes a surgical emergency. An early sign of impending compartment syndrome is a decrease in vibration sensibility.⁹ The complications of a missed compartment syndrome is so devastating that the standard of care is to err on the side of caution and release the muscle compartment if clinical index of suspicion is high.

Although nerves are fairly resistant to injury, especially when the surrounding tissue is not significantly damaged, a mixed nerve injury can often occur. The connective tissue surrounding the

larger nerves can contract as it heals, causing an additional compression of the nerve as well as a tethering effect to the surrounding tissue. In addition, local soft-tissue damage, especially to muscle, which is much less resilient to pressure than skin, nerve and tendon, can lead to fibrosis. Thus a relatively minor nerve injury may none the less have enough muscle swelling to cause irreparable damage without clinical evidence of a compartment syndrome leading to a more extensive permanent damage than one would anticipate.

This mechanism of nerve injury is usually treated conservatively and exploration is warranted if the nerve recovery does not follow an expected pattern. Along with traction injuries, crush injuries are treated conservatively with serial exams and electromyograms (EMGs) at three months if no clinical evidence of neurological recovery is noted.

Penetrating injuries

Penetrating trauma can be a result of sharp or blunt penetration, and will often have concomitant vascular structures and tendons injured in addition to the nerve injury. A sharp laceration, such as a hand laceration from a knife or piece of glass, necessitates exploration if a nerve deficit is present. The likelihood that the nerve is partially or completely transected is high. It is recommended to explore these injuries semi-electively within the first week. The more time that passes from the injury date, the less likely the two ends of the nerve can be mobilized to perform a primary repair. More than likely a gap will result after the traumatized nerve is trimmed to healthy nerve and a graft will be needed. In our practice, however, acute nerve grafting is performed if there is concern about a degree of injury that would make it difficult to reoperate in the future.

In the event of a penetrating trauma with an associated vascular injury, immediate exploration is warranted. Occasionally in proximal injuries with large arterial injuries with or without underlying fractures, the nerve injury is overlooked in the face of more urgent vascular and orthopaedic injuries. Rather, the deficit is noticed postoperatively, when it is unclear if the nerve injury is from the inciting event, iatrogenic during the repair of the vascular injury, or secondary to oedema or haematoma. Although a CT scan or MRI may be helpful to evaluate for the latter, internal scarring of the nerve may not always be clearly determined.

Blunt penetrating and blast injuries are usually treated conservatively, similar to closed crush and stretch injuries, because they may recover spontaneously. The local tissue trauma often causes a neurapraxia that resolves; however, electrodiagnostic studies and the algorithm for traction or crush injury followed should evaluate those that do not recover after three months.

Evaluation of nerve injuries

One must first start with a complete history and thorough physical exam. Sensation may be tested using the two-point static and moving discrimination, the Semmes-Weinstein filament test, or the '10 test'. This quick and easy test uses the

patient's own subjective perception to moving light touch to elicit differences in sensation between the right and left sides of the mirror digit.¹⁰

Both index fingers are stroked at the same time and patients are asked if the sensation is the same or different between the two digits. We find this technique to be particularly useful in young children and in patients who have some underlying neuropathy.

Determining spontaneous motor or sensory nerve recovery in closed injuries may be difficult, but the mechanism of injury can offer insight into the likelihood for early surgical management. For sensory recovery, serial physical exam is done looking for an advancing Tinel. In motor nerve injuries, the serial physical exam is accompanied by electrodiagnostic testing, as EMG changes precede clinical recovery. If by 3 months there is no evidence of reinnervation by way of motor unit potentials, surgical exploration and reconstruction is recommended. By evaluating and following the patient soon after the injury, we are in control of ordering any electrodiagnostic testing needed and thus avoid delay in recognizing a nerve deficit that will not resolve spontaneously.

There are clinical investigations using newer ultrasound machines that can detect nerve pathology; however these studies are still investigational and are highly operator-dependent.¹¹

An open injury is easier to assess, as surgical exploration is most often justified except in the case of a blast injury. Typically, we recommend that a sharp injury with associated motor or sensory deficits be explored within 7 days if possible. Conventionally, it is accepted that primary neurorrhaphy should be possible up to 2 weeks after an injury. With injuries that present later than 3 weeks, the surgeon should be prepared to do a graft repair.

Nerve repair

Timing of repair

In the case of a nerve transection, patients who are clinically stable should be taken to the operating room for exploration and repair of the nerve within the first 7–10 days. Nerves repaired acutely within the first 2 days are considered repaired primarily. A delayed primary repair occurs between 2 and 7 days. In either case, the proximal and distal ends of the nerve are freshened and an end-to-end coaptation performed. Nerves repaired after the first week are considered repaired secondarily. The cut-off days of 2 and 7 are arbitrary because even at 2–3 weeks from injury, the nerve can occasionally be coapted without need for a graft; however, the longer the time from injury, the less likely it is that a primary neurorrhaphy can be achieved, even with significant mobilization of the proximal and distal nerve ends.

Tension and nerve repair

Impaired regeneration as a result from scarring at the neurorrhaphy occurs when there is excessive tension at the repair site. Studies on an uninjured nerve show that a 15% stretch of

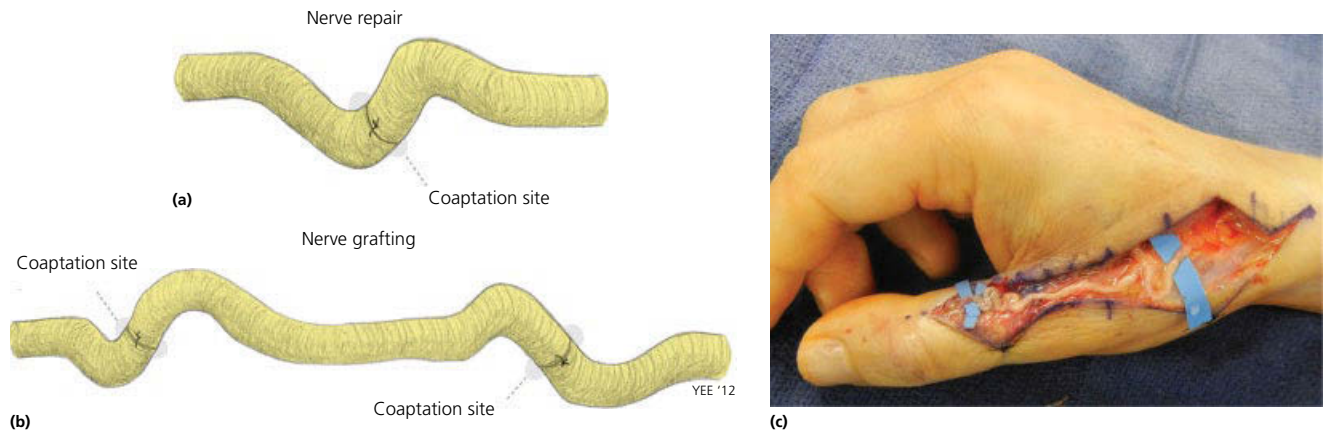


Figure 56.2 Tension-free nerve repair. A nerve repair without tension is important for optimal recovery, as tension on the nerve repair will significantly downgrade the functional results. In addition, it is important to have redundancy at the coaptation sites. Tension is checked by performing range-of-movement on the extremity. (a) Nerve repair without tension at single coaptation site. (b) Nerve grafting without tension at two coaptation sites. (c) This patient had a radial sensory neuroma that was grafted with a lateral antebrachial cutaneous nerve graft in the hand. The tension-free repair sites with redundancy occurred at both ends of the nerve graft. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

the nerve causes a reduction in microvascular flow. For the hour after relaxation the peak velocity is decreased to 66% of its normal velocity.¹²

In clinical practice we do not formally measure the tension across a neurorrhaphy; however we prefer to have a completely tension-free repair (Figure 56.2). Each nerve end is mobilized in order to re-approximate the two ends in a reasonable manner. A single 8–0 or 9–0 nylon suture is used to bring the newly fresh nerve ends together. If the nerve remains together after the limb is manipulated in its normal range of motion, the tension is deemed to be appropriate and the remainder of the repair completed with 9–0 nylon.

Strain on the nerve decreases the microcirculation and excessive tension will cause the repair to break down. Additional tricks such as positioning the arm in adduction or placing the wrist and fingers in flexion will significantly decrease the tension across the repair site. Depending on the location of the injury, this may increase the total length of available nerve; however, we do not recommend using postural manipulation in order to force primary repair. Not only can dehiscence occur when the joint near the repair is moved too early, but it may create significant stiffness from prolonged immobilization.¹³ An immobilized nerve also results in scarring at the repair site. If the nerve ends cannot be brought into reasonable proximity, it is recommended that the resulting gap is reconstructed with a nerve graft technique.

Type of repair: epineural versus fascicular

Any repair that withstands gentle range of motion is considered a 'tension-free' repair. Giddins *et al.*¹⁴ showed in a cadaveric study comparing suture size to the strength of an acute repair of the median nerve that 9–0 nylon withstood the greatest distractive forces. At a lower tension 10–0 snapped, and 8–0 sutures pulled out of the nerve tissue. However, in clinical practice, 10–0

and 8–0 sutures are often used based on the size of the nerve, the thickness of the epineurium and the amount of inflammation.

Fibrin glue may be used in place of suture repair when there is no tension. Experimentally lasers have been used to coapt the epineurium; however, the heat produced causes tissue damage and a suboptimal tensile strength at the repair site. Microsuture on the order of 8–0, 9–0 or 10–0 nylon placed with the use of loupe or microscopic magnification remains the gold standard. Approximately two-thirds of surgeons use a combination of microsutures and glue, whereas about one-third use one or the other. A personal preference is not to use a nerve wrap.

External markers, such as a vessel on the surface and matching the fascicular patterns, assist in aligning the two nerve ends (Figure 56.3). Overlaps should be avoided. By placing the sutures in the epineurium after the fascicles have been trimmed back behind the level of the epineurium, a sleeve is created with the external connective tissue. For larger nerves, a fascicular repair is possible, where the perineurium acts as the epineurium. As long as the fascicles do not overlap, both techniques give similar results.¹⁵ We feel that neither repair is superior to the other. The more critical issues are tension and alignment to avoid fibrosis.¹⁶

Our preferred method for repair is a group fascicular repair with the aim of aligning the sensory/motor groups appropriately. The least number of epineural sutures, using 9–0 nylon to approximate the ends and keep the repair intact through a full range of motion, is used. If there is a slight mismatch in size, as in the setting of a nerve transfer, we can improve that size mismatch by going more proximally on the recipient nerve to make the diameter smaller and more distally on the donor nerve to make a cross-sectional area greater. We will occasionally also use fibrin glue, but do not use nerve wraps as we have seen these remain present years after placement, causing neural ischaemia and pain.

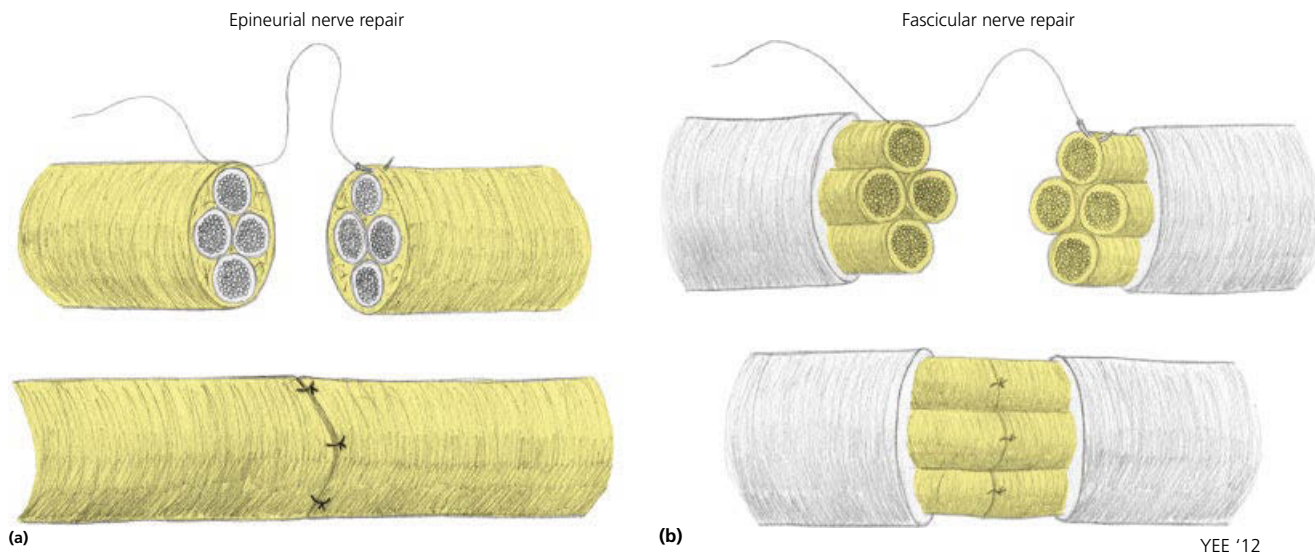


Figure 56.3 Epineurial and fascicular nerve repair. (a) Epineurial nerve repair. (b) Fascicular nerve repair. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

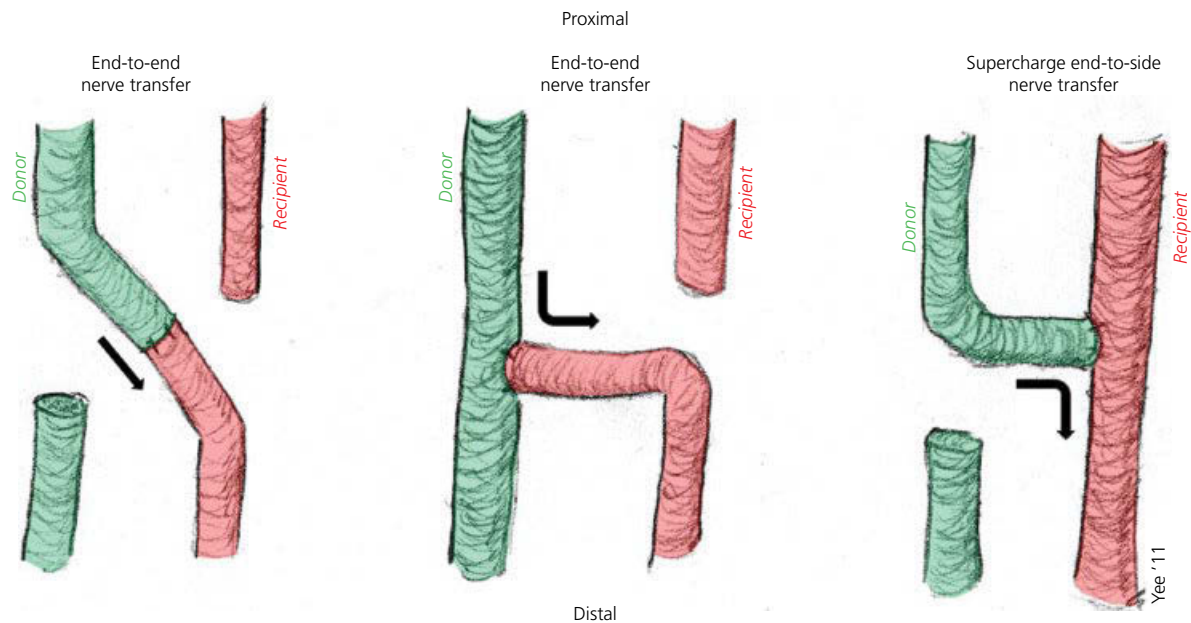


Figure 56.4 Types of nerve repairs. Primary nerve repairs and nerve grafting occur in a traditional end-to-end fashion with its native nerve. When a nerve is transposed to another nerve, it is described as a nerve transfer. Nerve transfers can have a coaptation of an end-to-end, end-to-side or supercharge end-to-side fashion. Traditional nerve transfers occur from donor to recipient in an end-to-end fashion. End-to-side nerve transfers is described as the recipient nerve end being transferred to the side of the donor nerve. Opposite of an end-to-side, supercharge end-to-side nerve transfers is described as the donor nerve end being transferred to the side of a recipient nerve. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

Type of repair: end-to-end versus end-to-side

Most nerve repairs, whether primary or with a graft repair, are performed end-to-end. We recommend end-to-end repair for all motor, mixed nerves and sensory nerves in critical distributions. We use sensory end-to-side repair for non-critical sensory nerves (Figure 56.4) such as the dorsal cutaneous branch of the ulnar nerve. We also use end-to-side to restore sensation to a donor sensory nerve graft territory, such as

transferring the distal end of the medial antebrachial cutaneous to the sensory (lateral) side of the median nerve.

When we do sensory end-to-end nerve transfers (for example, the third webspace median to ulnar sensory), we do an end-to-side of the distal end of the donor nerve (third webspace median) to an adjacent normal sensory nerve (ulnar nerve). This transfer is conducted to restore sensation in the donor nerve distribution (third webspace median).

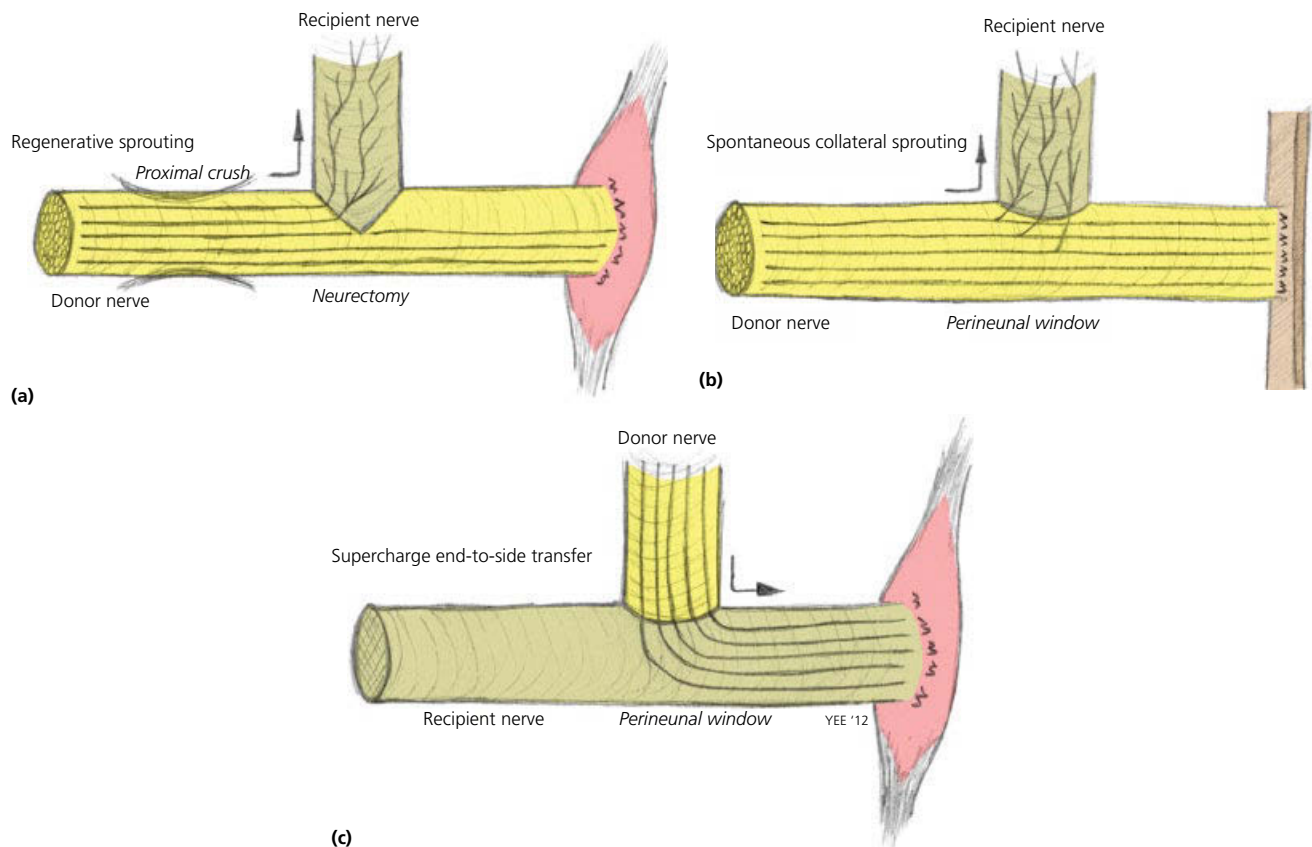


Figure 56.5 Nerve regeneration in end-to-side nerve repairs. The two types of end-to-side nerve repairs or transfers are the traditional end-to-side and supercharge end-to-side. End-to-side nerve transfers have two sprouting mechanisms that are characteristic of either motor or sensory nerves. **(a)** Regenerative sprouting occurs in motor nerves in response to an injury. In order to elicit regenerative sprouting for a nerve transfer, a partial neurectomy and a proximal crush is required. The proximal crush causes an axonometric injury and creates a proximal regenerative front that regenerates into the recipient nerve and backfills the donor nerve. **(b)** Spontaneous collateral sprouting occurs in sensory nerves. Due to the spontaneous sprouting, no injury is required except for a perineurial window for the nerve fibres to regenerate through. **(c)** Supercharge end-to-side nerve transfer is where the donor nerve end is transferred into the side of a recipient nerve. This allows nerve fibres to regenerate into the recipient nerve through a perineurial window without sacrificing the integrity of the donor nerve. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

While sensory axons can spontaneously sprout collateral axons with respect to end-to-side repairs, motor axons need to have a proximal injury in order for 'traumatic' sprouting to occur (Figure 56.5).¹⁷ One setting where we employ this technique is for the spinal accessory to suprascapular nerve transfer. The suprascapular recipient nerve is transferred end-to-side to the spinal accessory donor nerve via a partial neurectomy in the donor nerve. An area of the spinal accessory nerve proximal to the repair is crushed with micro forceps to initiate sprouting into both the suprascapular recipient nerve and the spinal accessory donor nerve to minimize deficit of the donor nerve.

Based on our experimental studies, we had used a reverse end-to-side nerve transfer to augment recovery in 60 patients with second- and third-degree injuries. The normal anterior interosseous motor nerve branch to the pronator quadratus is transferred to the side of the motor component of the ulnar nerve, about 8–9 cm proximal to the wrist crease. Each of the 2–3 distal anterior interosseous nerve branches are sewn to the side of the ulnar motor fascicular group, which in general

includes 2–3 fascicles. We believe these anterior interosseous nerve axons both innervate and 'babysit' the ulnar intrinsic muscles, in essence creating a Martin–Gruber anastomosis. Results have been very encouraging.

Intraoperative nerve stimulation

In our practice, a detailed clinical examination, combined with preoperative electromyogram/nerve conduction studies (EMG/NCS) completed by our skilled and experienced neurologists have made intraoperative nerve monitoring unnecessary. We do routinely, however, use a handheld nerve stimulator in the operating room. It provides muscle contraction, but no information about sensory recovery and is used when motor nerve transfers are performed. It allows us to pick out redundant motor nerve fascicles, which will be transferred into non-functioning motor nerves to restore function (Figure 56.6). For routine acute lacerations that require grafting or primary repair, there is no additional benefit to

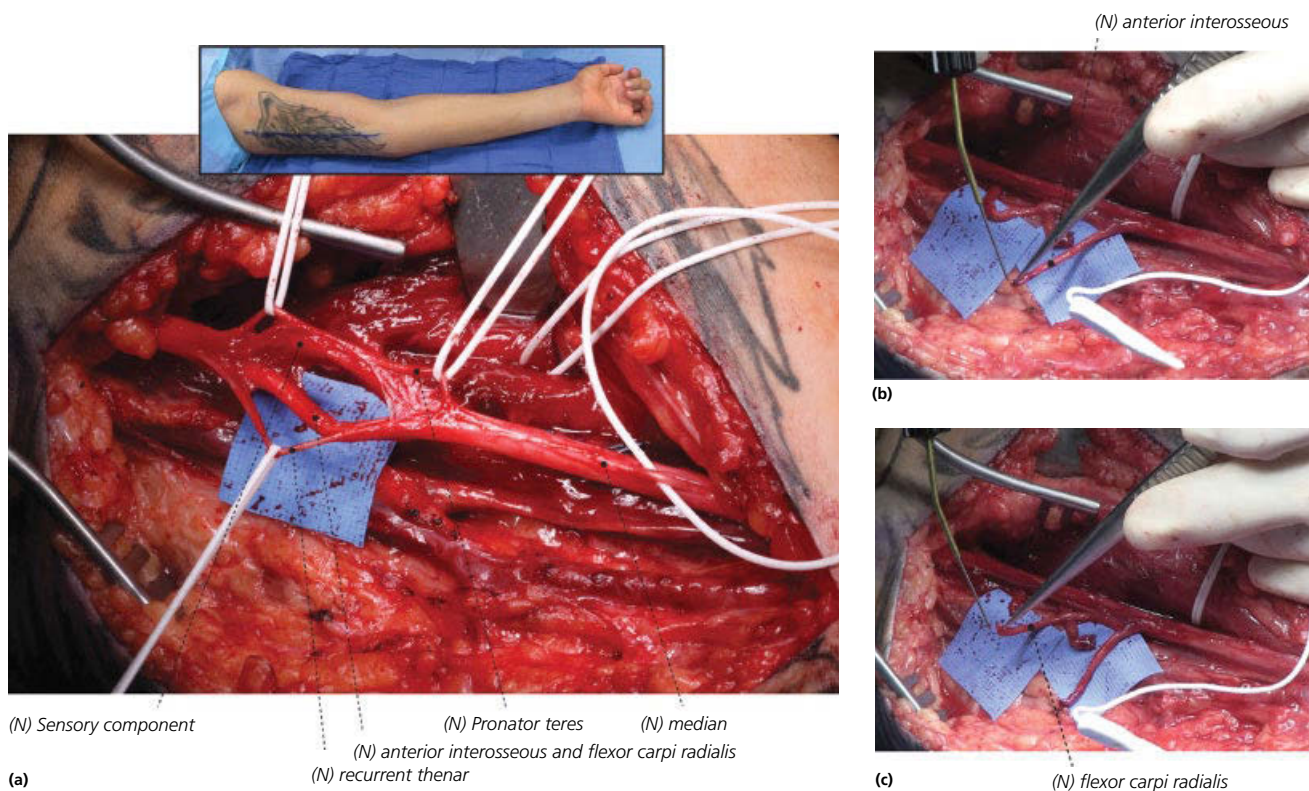


Figure 56.6 Intraoperative electrical stimulation of nerve for identification. Intraoperative electrical stimulation allows for identifying motor and sensory components of a normal nerve, in addition to determining whether a motor nerve has innervated a muscle following an injury. Evaluation within 72 h post injury will allow electrical stimulation of the distal injured nerve for identification. After 72 h, the distal injured nerve will not respond to electrical stimulation due to the degeneration process. (a) This patient had a C7 spinal cord injury, where the peripheral neurons were intact and the central neurons were compromised, and underwent a nerve transfer. Interfascicular neurolysis of the median nerve revealed its fascicular component that were identified through electrical stimulation. (b, c) After proximal transection of the flexor carpi radialis and anterior interosseous nerve fascicles, the nerve fascicles continued to respond to electrical stimulation and are expected to respond for another 72 h post transection. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

the use of nerve stimulation, unless it is performed in motor and mixed motor nerves transected and repaired within 72 h. For a partial nerve injury with neuroma involving only a portion of the nerve's cross-sectional area, a nerve stimulator can help determine which motor nerve fascicles to preserve, while resecting the damaged tissue. In nerves that are purely or mostly sensory fibres, a handheld nerve stimulator is not useful.

Factors affecting outcome

Many factors beside technique affect nerve repair and age of the patient seems to be the most important factor. From historical data, we know that nerve repair in children and young adults yields better results than in adults. One reason is that the nerve has a shorter distance to travel. Nerves regenerate at 1–1.5 mm/day and in a child, the nerve recovery would be expected to reach the intended target organ earlier than in an adult and thus have a quicker recovery. Secondly, a child's brain has greater plasticity and more neural activity than an adult, thus the child has an enhanced ability to process the reinnervated motor and sensory within the brain cortex.

The type of injury, the location and the degree or depth of the injury affect the outcome. A sharp laceration will have less associated soft-tissue trauma as a crush or avulsion injuries at the same level. Proximal injuries contain may contain mixed nerves and may affect larger muscle or sensory domains. The reconstruction can be more challenging and a minor misalignment of fascicles has greater consequences than a more distal injury. Sensory nerve lacerations in the finger repaired a few days after the injury usually have excellent outcomes while sensory nerve neuromas requiring reconstructed with grafts because of a finger crush months after the injury do not recover as well. Often the internal damage is underestimated and if care is not taken to be outside the zone of injury, the repair may be suboptimal.

When considering the surgical intervention, timing, type of repair, tension and alignment of fascicles have an equally important role in the outcome of nerve recovery. Earlier repair usually results in better outcomes and the best results occur if the repair can be performed within 3 weeks. A good result can still be expected if the repair occurs before 6 months, although a nerve graft will most likely be needed. Functional recovery (FR) is directly proportional to the number of motor axons reaching the target endplate and inversely proportional to the time of denervation. 'Time=muscle.'

$$FR \propto \frac{\text{Number of motor axons reaching target endplate}}{\text{Time of denervation}}$$

Bridging the gap: current techniques

The gold standard for a nerve injury repair is a primary neurorrhaphy within the first few days. The belief that a primary nerve repair with a single coaptation site is superior to a nerve reconstruction with a graft involving two neurorrhaphy sites is supported by multiple animal studies (Figure 56.7).¹⁸ A certain

percentage of fibres may be lost at each neurorrhaphy site, thus limiting the number of repair sites to one should be the goal. Some studies suggest that as much as 50% of regenerating sensory or motor axons may never reach the correct end organ.¹⁹ Tension at the repair is to be avoided as mentioned earlier and a primary repair should never be forced together. Rather, a graft repair should be performed. Making sure to be outside the zone of injury is essential (Figure 56.8) as well as using meticulous microneurosurgical technique, accurately matching the sensory and motor nerve fascicles, and begin early protected motion to allow neural gliding.

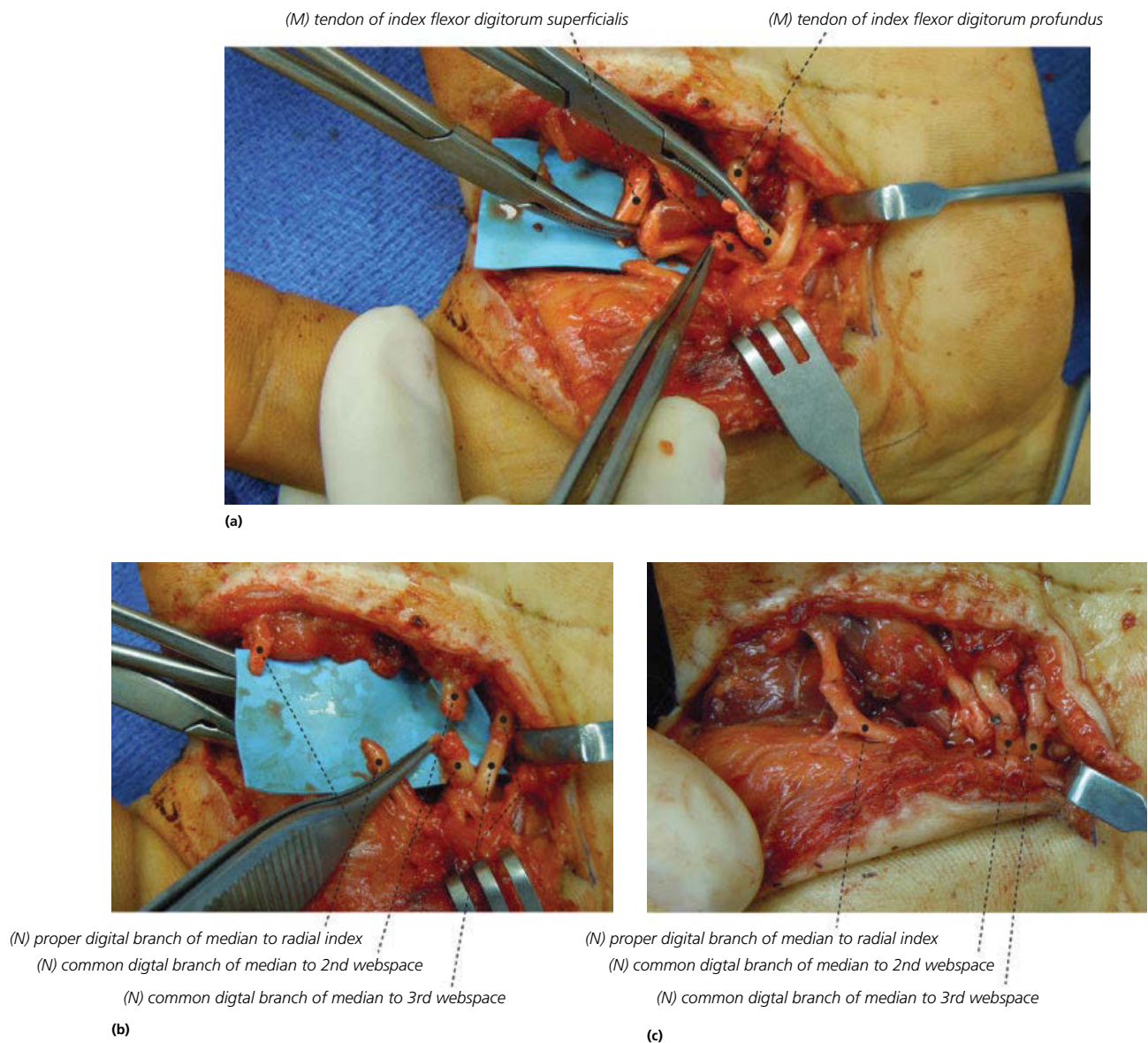


Figure 56.7 Primary nerve repair of a sharp lacerating nerve injury. Nerve injuries that are sharp and lacerating induce a small zone of injury, which can allow for a tension-free primary repair. (a) This patient had a sharp lacerating injury to the right palm, which involved the digital nerves and tendons of the flexor digitorum superficialis and profundus. These tendons were repaired in zone 3. (b) Digital nerves of the median nerve were identified and transected. (c) The proximal and distal ends of the digital nerves were trimmed by evenly transecting the ends for healthy nerve fibres. Primary repairs were performed without tension in all range of motion of the fingers, wrist and hand. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

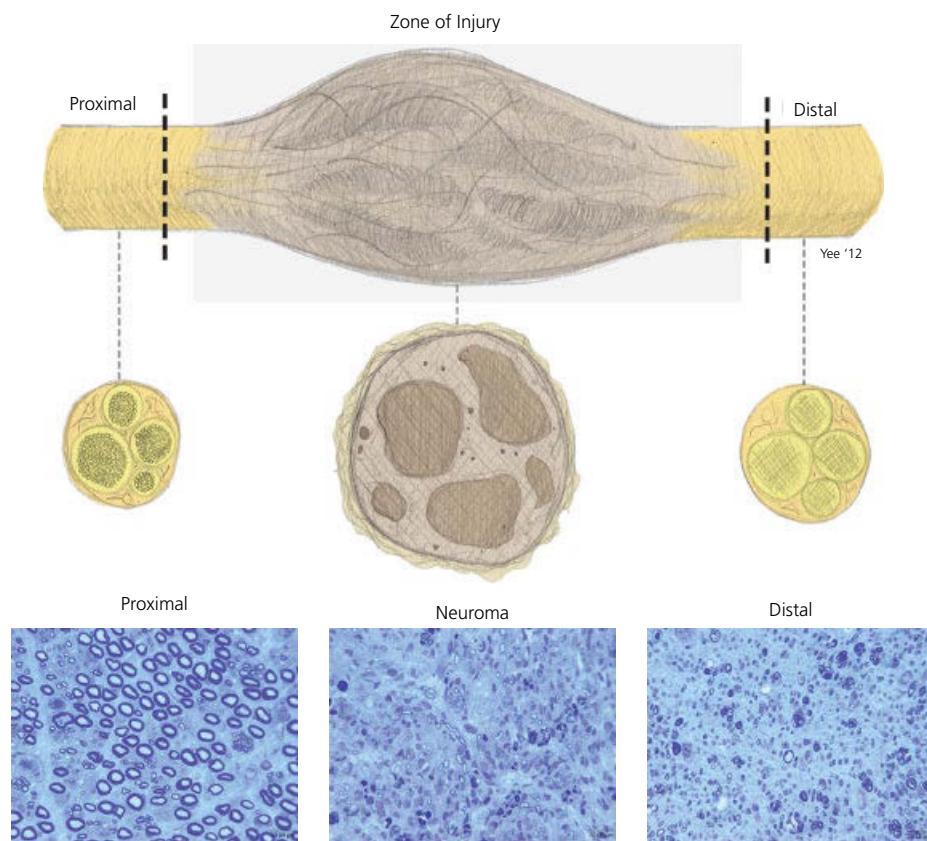


Figure 56.8 Zone of injury within a nerve. The zone of injury is dependent on the mechanism of injury. A sharp lacerating injury will have a small zone of injury, while blunt, stretch, gunshot and traumatic injuries will have large zones of injury. It is important to excise the injured nerve outside the zone of injury (dotted lines) to allow proximal nerve fibres to regenerate distal without inhibitory factors from the injury. Histological section of proximal nerve exhibits normal nerve fibres. Histological section of neuroma exhibits disorganized patterns of fibres in a dense stroma of connective tissue. Histological section of distal nerve, distal to the injury, exhibits degenerating nerve fibres. Magnification 400 $\times$, Toluidine Blue. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

When primary neurorrhaphy is not possible, mobilization and elongation of both nerve ends can bridge small gaps usually less than 5 mm in size because of the inherent elasticity of a nerve.²⁰ When there is extreme tension and contamination, scarring of the repair site will invariably occur and a proximal neuroma develop. We recommend reconstructing the nerve with a graft and accepting two neurorrhaphy sites than perform a single neurorrhaphy under unfavourable conditions.^{13,21} Several options for gap reconstruction are listed below with the pros and cons for each option (Table 56.3).²² An autologous nerve graft remains the most common technique for repairing defects in peripheral nerves, although an acellular nerve allograft can be used for short nerve gaps ≤ 3 cm for small diameter/non-critical nerves.^{23–26} In the senior author's opinion, if an acellularized allograft ≥ 4 cm is used, it will not consistently drive axons across the allograft but will help to 'dwindle' nerve regeneration such that a painful neuroma will not develop. In a non-critical digital nerve this would be an acceptable result, as collateral sprouting of an adjacent sensory nerve will occur to provide some sensation and the long ≥ 4 cm allograft will 'dwindle' regeneration to prevent painful neuroma formation.

The purpose of the nerve graft is to act as guide for the regenerating axon. The most common harvested nerve graft is the sural nerve; however, when working in the upper limb we prefer to use an expendable nerve in the field such as the lateral or medial antebrachial cutaneous nerve or the distal anterior or posterior interosseous nerve.^{27–29} Experimental studies comparing motor, mixed and sensory nerve grafts to repair a motor nerve defect shows a motor or mixed nerve grafts is a superior graft choice than a sensory nerve graft.^{30,31} However, in clinical practice, expendable motor nerve grafts are extremely limited. The gracilis muscle nerve and the distal anterior interosseous nerve are the two motor nerve grafts that cause the least morbidity.

Nerve grafts typically are less than 3 mm in diameter, which is a consequence of the historically poor results achieved when full-thickness nerve trunks were used as grafts.³² When necessary, the grafts may be cabled together. It is believed that smaller grafts will revascularize more easily, ultimately contributing to the success of the graft. There is no consensus on what is the maximum length a graft may be and still achieve regeneration of the nerve, although there are a few studies that have shown success with

Table 56.3 Options for management of a nerve gap

Type	Advantage	Disadvantage
Autograft	Gold standard for reconstruction Schwann cells in extracellular matrix	Second operative site Results in donor sensory loss Potential for neuroma formation/pain Sensory nerve autograft do not support motor regeneration as well as motor or mixed sensory nerves Limited available length
Acellularized allograft	Functional recovery is almost equivalent to autograft No donor site morbidity Non-immunogenic	Length limitation (<7 cm) No Schwann cells
Allograft	Functional recovery is almost equivalent to autograft No donor site morbidity	Requires patient systemic immunosuppression (about 18 months) Patient vulnerable to opportunistic infections
Conduit	Guides regenerating nerve to intended target No donor site morbidity	Length limitation Only for small-diameter sensory nerves
End-to-side	No length limitation	Poor sensory results Motor requires donor neurectomy
Reverse end-to-side	Augment partial motor/sensory nerve injury	Requires knowledge of topography Requires knowledge of expendable donors May need nerve autograft or acellular graft
Nerve transfer	Earlier motor/sensory target recovery	Requires expendable donor Requires motor (sensory) re-education Requires knowledge of topography

Source: Weber et al. Mackinnon Repair and grafting of peripheral nerve. In Gurtner & Neligan (eds), Plastic Surgery Volume 1: Principles; Elsevier, 2013. Reproduced with permission of Elsevier.

grafts as long as 20 cm.^{33,34} However, in general nerve autografts ≤ 6 cm seem to be more reliably successful if close to the target, compared with longer nerve autografts. Failure of the long nerve autografts was the stimulus to move towards nerve transfer techniques in the 1990s, just as failure with nerve repair done under tension to overcome longer gaps was the stimulus to the shift towards the nerve grafts by Millesi in the 1970s.

Free vascularized nerve grafts were introduced in 1976 by Taylor and Ham in order to treat these longer gaps.³⁵ For smaller defects, a vascularized graft and conventional graft do not appear to differ in clinical outcome; however, Doi *et al.*³⁶ recommend using free vascularized nerve grafts when the gap distance is greater than 6 cm with associated soft tissue loss over the repaired area. Currently the indication for a free vascularized nerve graft is for reconstruction of large-diameter nerve grafts, such as the ulnar nerve in brachial plexus avulsion surgery.^{37,38}

The gold standard for repairing a nerve gap injury is with an autologous nerve graft. (Figures 56.9 and 56.10) Because the major disadvantage of autografts is the donor site morbidity and the limited number of donor nerves available, new approaches to repairing the gap defect have been created. This has led to the use of vein grafts, biological and synthetic conduits, and nerve allograft as substitutes for short gap injuries. The current consensus is that for a sensory deficit in a non-critical, small-diameter nerve less than 3 cm, any of these options is equally reliable,³⁹ although more recent literature supports the fact that while nerve allografts offer excellent results in this gap range,

the conduit results can vary greatly depending on type of material when the gap is greater than 15 mm.^{40,41} Neither conduits nor allografts are currently recommended for gaps longer than 3 cm or for gaps in motor and mixed motor defects, although clinical trials are in progress assessing the role of allografts in some of these situations.⁴⁰

Acellular human processed nerve allografts have been available for clinical use since 2008 as an alternative to conduits (Figure 56.11). In an animal study, Whitlock²⁴ showed that the acellular allograft was superior to an empty conduit because of its extracellular matrix, but inferior to an autograft, which provides Schwann cells in addition to the extracellular matrix. Initial clinical trials using allografts for short sensory gaps show recovery comparable to autografts and better than a conduit.⁴² Because this is human tissue, it retains its three-dimensional structure, including epineurium, fascicles, endoneural tubes and laminin, and it is believed that the scaffold allows for repopulation of the host cells, as in autologous nerve tissue. The product is expensive; however, since it shortens operative time, it allows the surgeon to be more efficient if working without assistance and may save overall operating room costs.

The patient benefit is no morbidity of an additional scar and associated sensory loss. It handles similarly to autologous nerve and comes in various widths ranging from 1 to 5 mm in diameter and up to 70 mm in length. Interestingly, research studies have not shown successful regeneration at a distance of 40 or 60 mm (Figure 56.12).²³

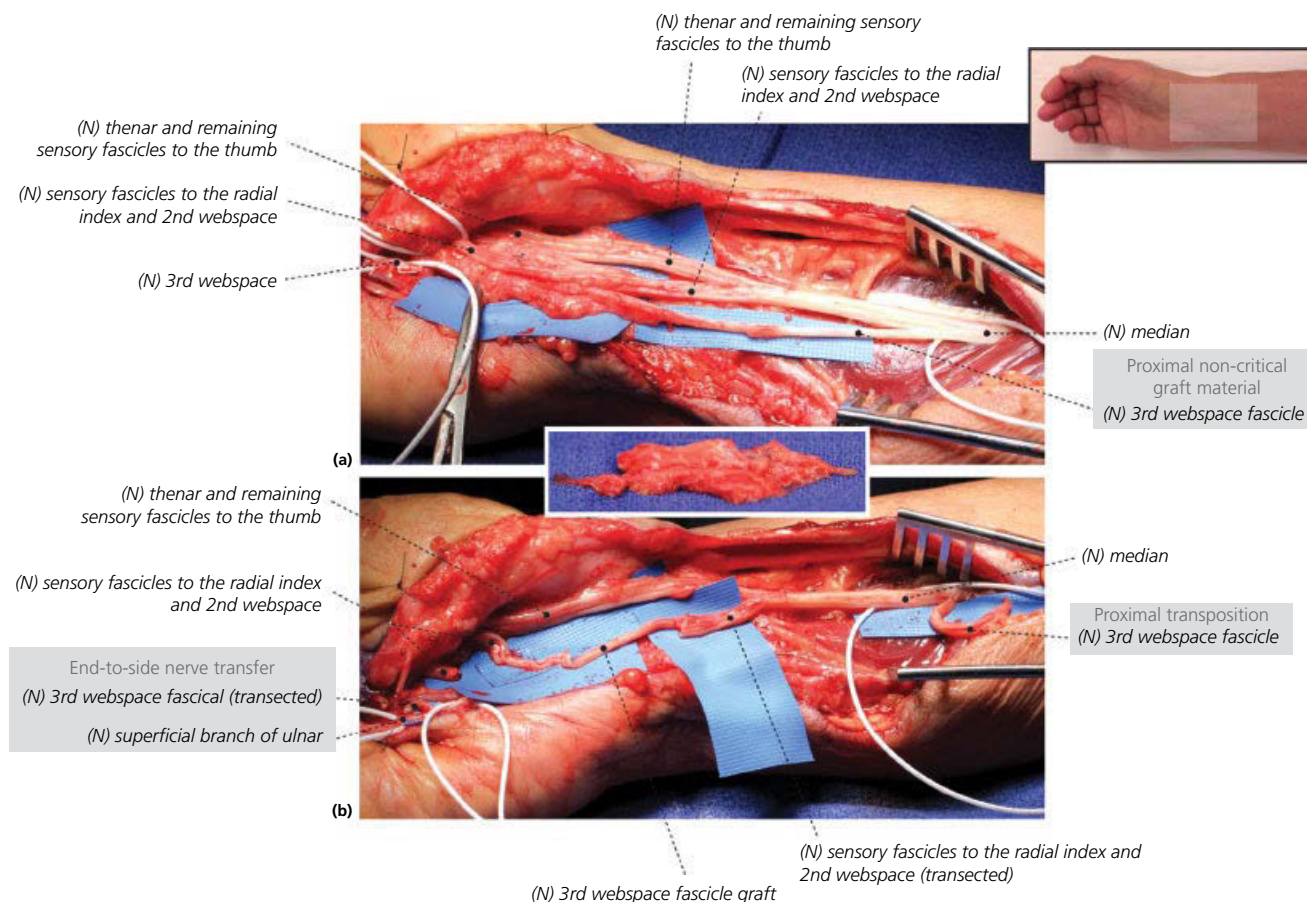


Figure 56.9 Case of median nerve reconstruction at the wrist with third webspace graft. This patient had a significant injury to the median nerve at the wrist that involved the sensory components. **(a)** The zone of injury was identified and the neuroma was resected with proximal and distal median nerve components identified. The third webspace was further neurolysed proximally to mobilize graft material. **(b)** The proximal end of the third webspace fascicle was transected and used as a nerve graft to repair a portion of the median nerve. The proximal remainder of the third webspace was transposed proximally to prevent a painful neuroma. The distal third webspace was end-to-side transferred to the sensory component of the ulnar nerve to provide rudimentary sensation for donor deficit. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

Autologous vein grafts are the oldest conduit and are still used both as a primary conduit and as a wrap.⁴³ Biological conduits, including bone, artery and muscle⁴⁴ have been used, but not as conventionally as vein, collagen⁴⁵ or small intestine submucosa (SIS).⁴⁶ Biodegradable synthetic conduits such as polyglycolic acid are currently used, while non-degradable nerve guides made from silicone have fallen out of favour.⁴⁷ Their major disadvantage is leaving foreign material that potentially causes a chronic reaction with excessive scarring (Figure 56.13).

Of the commercial conduits in use today, collagen and polyglycolic acid are biodegradable, while the SIS conduits are resorbed and replaced by the body's natural tissue. Like the original vein graft, they are used as primary conduits or as nerve wraps. They provide at least protective sensation and, in some cases, good sensory recovery.⁴⁸ Since the conduit is essentially a hollow tube with no structure, experimental studies have shown that placing morselized nerve into the tube can help to augment nerve growth.⁴⁹ When using a conduit, we recommend placing a

small portion of the proximal nerve into the conduit to provide a source of Schwann cells.

Nerve transfer: alternative to conduits

The introduction of the nerve transfers in the early 1990s has resulted in a paradigm shift in how nerve injuries are managed and for the severe proximal injuries nerve transfers have resulted in marked improved functional results.

Tendon transfers set the stage for the development of motor nerve transfers. The principles are similar expect that a single nerve transfer can innervate more than one muscle unlike a tendon transfer. The type of muscle fibre unit and the insertion of the tendon will influence the ultimate effectiveness of that muscle's contraction in its new position.^{50,51} Excursion and vectors do not need to be considered in nerve transfers, although the number of axons in the donor nerve will affect the overall

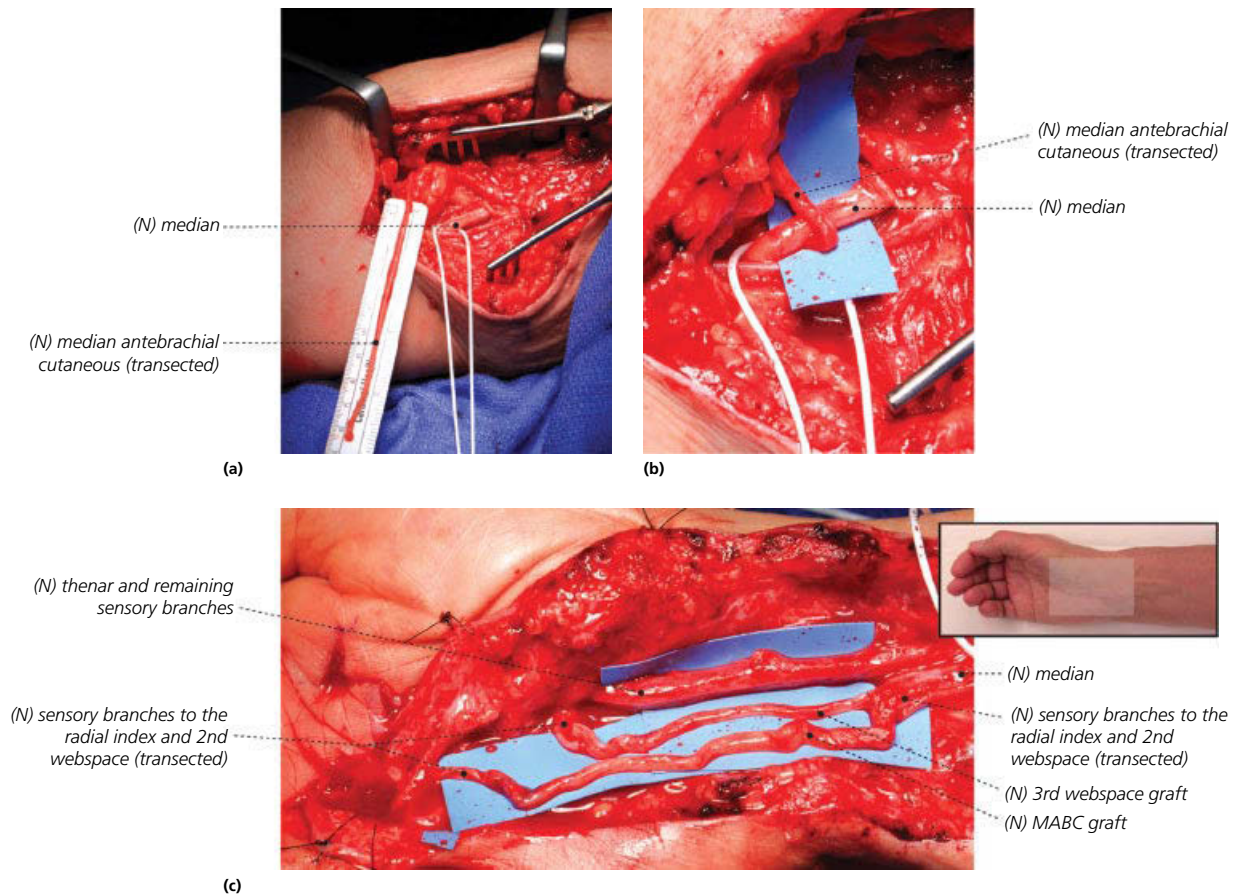


Figure 56.10 Second webspace grafted with a medial antebrachial cutaneous nerve graft. (a) The medial antebrachial cutaneous nerve (MABC) was isolated within the arm for donor material. (b) The MABC was then transected with the distal end transferred to the sensory component of the median nerve through and end-to-side epineurial window fashion. The sensory component of the median nerve is located on the superior aspect of the median nerve. Note that in this image, the median nerve has been rotated so that it appears to be on the inferior portion. (c) The MABC graft was used to repair the remaining portion of the median nerve. The thenar branch and remaining sensory branches to the thumb were protected and were found to be not injured. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

strength of the recovery. We like to use a donor nerve with a similar number of motor axons as the recipient nerve we are targeting. Expendable nerves and redundant fascicles or branches are used, avoiding nerves that innervate critical functions.

The major advantages of a nerve transfer over that of a tendon transfer are that: (a) sensory nerve transfers can restore sensibility; (b) a nerve that innervates multiple muscle groups can be restored with a single nerve transfer; and (c) the insertion and attachments of the muscle(s) in question are not disrupted, thus the original muscle function and tension are maintained. When choosing donor nerves for transfer, a nerve that innervates a synergistic muscle group is preferable, as it facilitates postoperative re-education. Although a nerve supplying a non-synergistic, or even antagonistic, muscle group may be used, more motor retraining may be necessary to learn to contract the newly reinnervated muscle. Although a synergistic nerve transfer is ideal, antagonistic nerve transfers can be successfully utilized in some cases, such as branches of radial nerve used to restore median nerve function. In median to

radial nerve transfers we are careful to transfer flexor carpi radialis to the posterior interosseous nerve and flexor digitorum sublimis nerve to the extensor carpi radialis brevis nerve (Figure 56.14). Similarly, when we do a double fascicular transfer (DFT) for elbow flexion we will try to always use the median fascicle to transfer to the brachialis nerve and the ulnar fascicle to transfer to the biceps nerve for faster motor re-education (biceps supinates and median nerve pronates, so it is a bit harder to re-educate if done the opposite way).

Nerve transfers offer the advantage of converting a proximal high-level nerve injury to a low-level nerve injury by moving the reconstruction closer to the target muscle or sensory territory of the affected nerve. In high ulnar and high median nerve injuries this is critical. A donor nerve which is close to the injured nerve is used in nerve transfers and usually nerve grafts are not needed. The donor and recipient nerves are neurolysed from the main nerve so that an end-to-end repair between donor and recipient fascicles can be done. Nerve transfers are discussed in greater detail elsewhere.

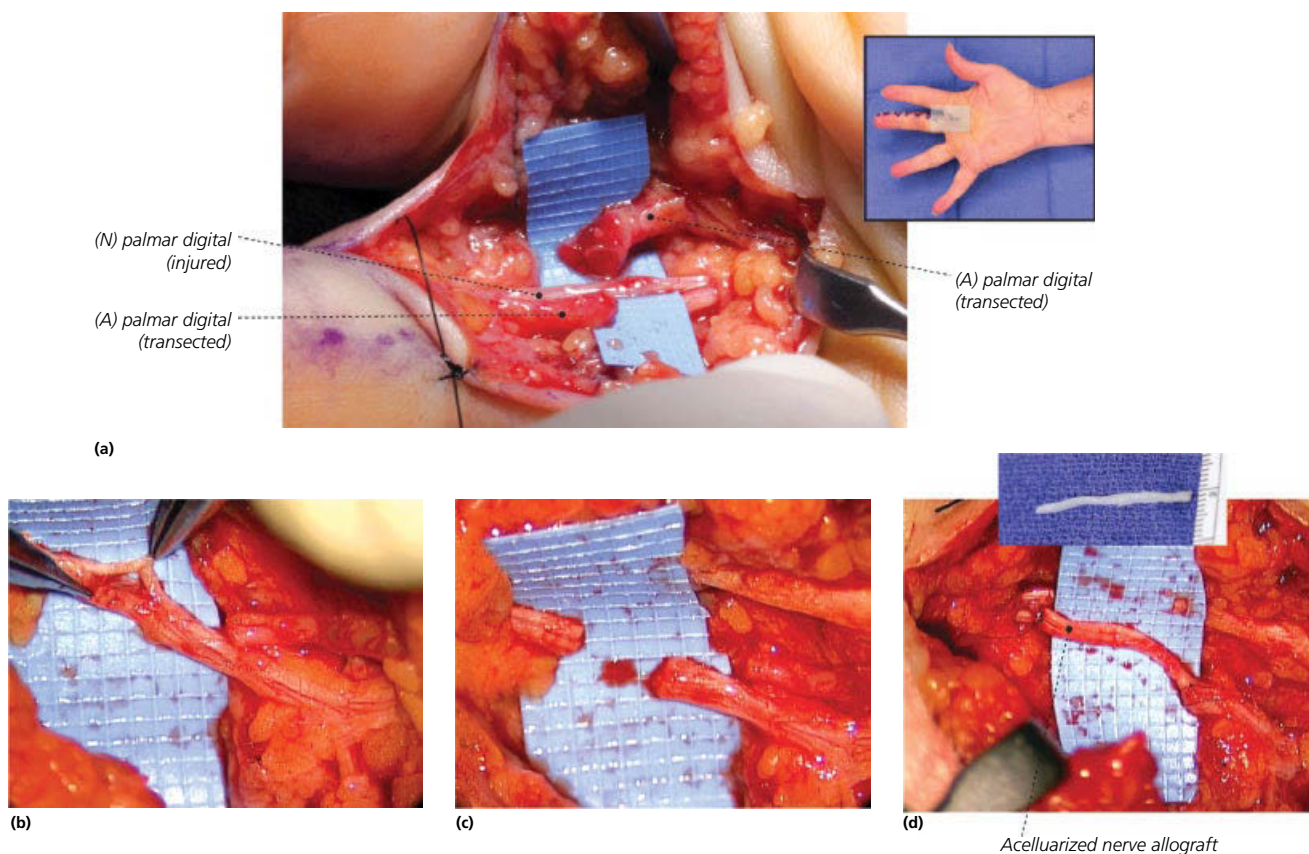


Figure 56.11 Reconstruction of a non-critical sensory nerve with acellularized nerve allograft. This patient had a lacerating injury at the base of the long finger. (a) Upon exploration, the palmar digital artery was found to be transected. The palmar digital nerve to the radial aspect of the long finger was found to be intact but injured. (b) The digital nerve had been longitudinally injured by the sharp object. (c) The zone of injury was identified and resected until healthy nerve was seen at both proximal and distal ends. (d) A 2 cm acellularized allograft was used to bridge the nerve gap for a tension-free repair. The nerve repair was tested for tension by putting the long finger through full flexion and extension. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

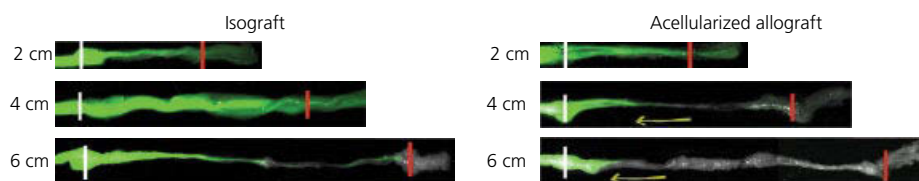


Figure 56.12 Nerve regeneration through various lengths of isografts and acellularized nerve allografts (ANA) in a fluorescent GFP rat model. Regeneration through a short 2 cm graft were similar between an isograft and ANA, however as the graft length increased, the regeneration distance of the ANA decreased in comparison to an isograft. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

Most nerve transfers are done with an end-to-end repair. As mentioned, the circumstances in which an end-to-side neurorrhaphy is utilized continues to evolve.^{52–54} The majority of these end-to-side transfers are for sensory restoration; however, when a motor nerve transfers is performed with this technique, a partial neurectomy to facilitate end-to-side regeneration is required.¹⁷

Nerve transfers have evolved because of our understanding of internal topography and the redundancy built into the human nerve. Intraoperative electrical stimulation with a hand held nerve stimulator identifies the motor donor fascicles, allowing for selection of the donor nerve without extensive

neurolysis. Fascicle groups can easily be cleaved from the main nerve even in the proximal extremity without injuring unintended fascicle groups because the nerves do not merge distally as once thought. Instead, the fibres course parallel to each other via an extensive plexus pathway.⁵⁵

Postoperative management

We recommend immobilizing for 1–2 weeks after a simple nerve repair; however, gentle protected range of motion can be started as early as 2–3 days. No consensus exists on what length of time

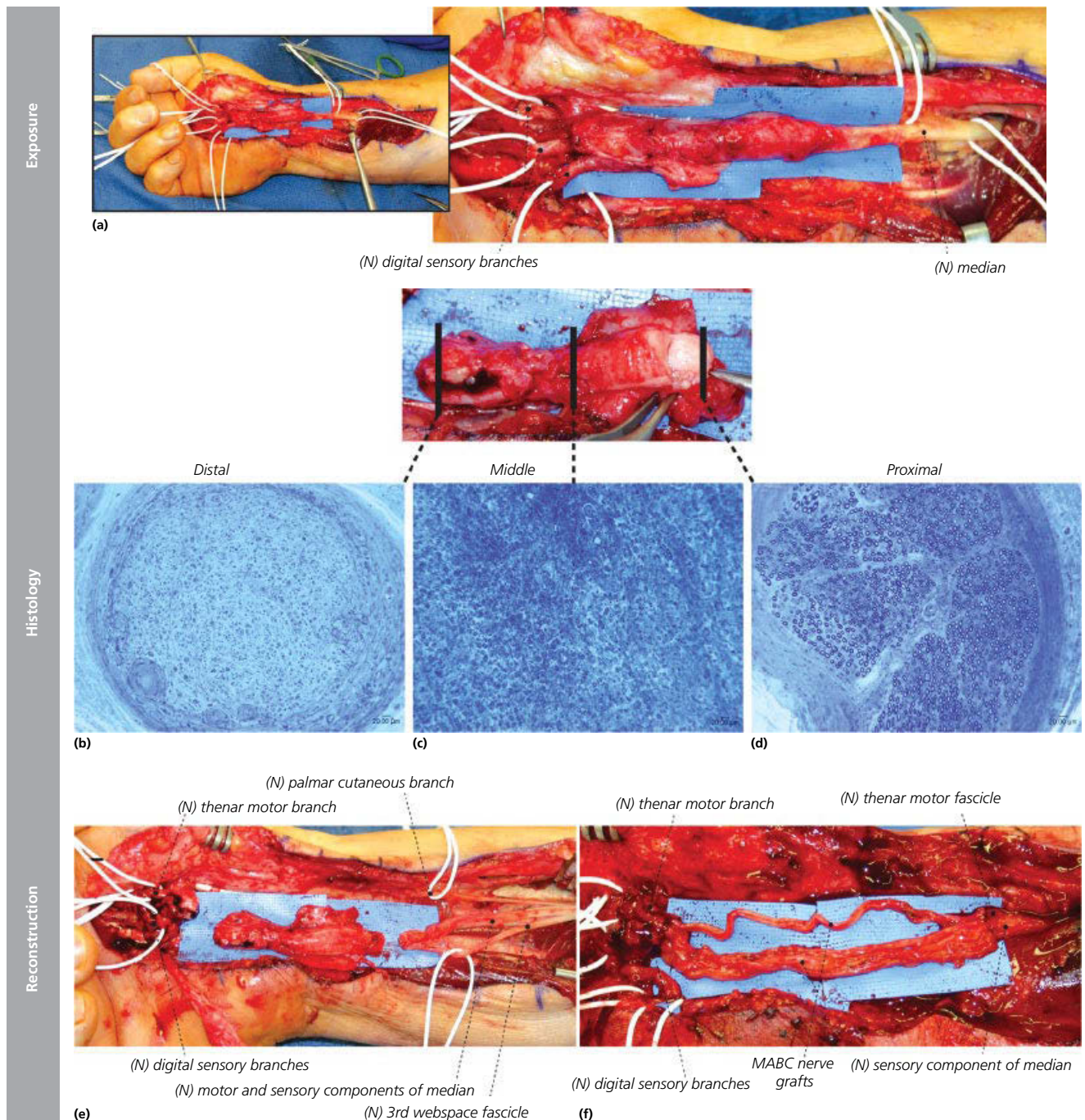


Figure 56.13 Failed nerve wrap case. Patient presented with a right median nerve injury following a carpal tunnel release that was repaired with a nerve wrap 2 years postoperatively. The sensory component of the median nerve was injured and repaired with a nerve wrap. **(a)** Upon exposure, the median nerve was identified with a large neuroma. **(b)** Histological evaluation revealed scarce nerve fibres in the distal segment. **(c)** The middle segment displayed significant disorganized architecture with no axonal organization, consistent with neuroma. **(d)** The proximal segment revealed healthy nerve fibres. **(e, f)** The neuroma was resected and the motor and sensory components of the median nerve identified. The gap was reconstructed with several medial antebrachial cutaneous nerve cable grafts with an individual graft for repair of the motor component. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

for immobilization is necessary. However we believe that early, protected range of motion is critical for neural gliding and an excellent result. In patients with concomitant tendon or bony injuries, the postoperative protocol is guided by the injury most

difficult to rehabilitate. For example, in finger lacerations in which both tendon and nerves are transected, a flexor tendon protocol is followed postoperatively in order to maximize tendon gliding. The nerve repair, while important, is easier to reconstruct

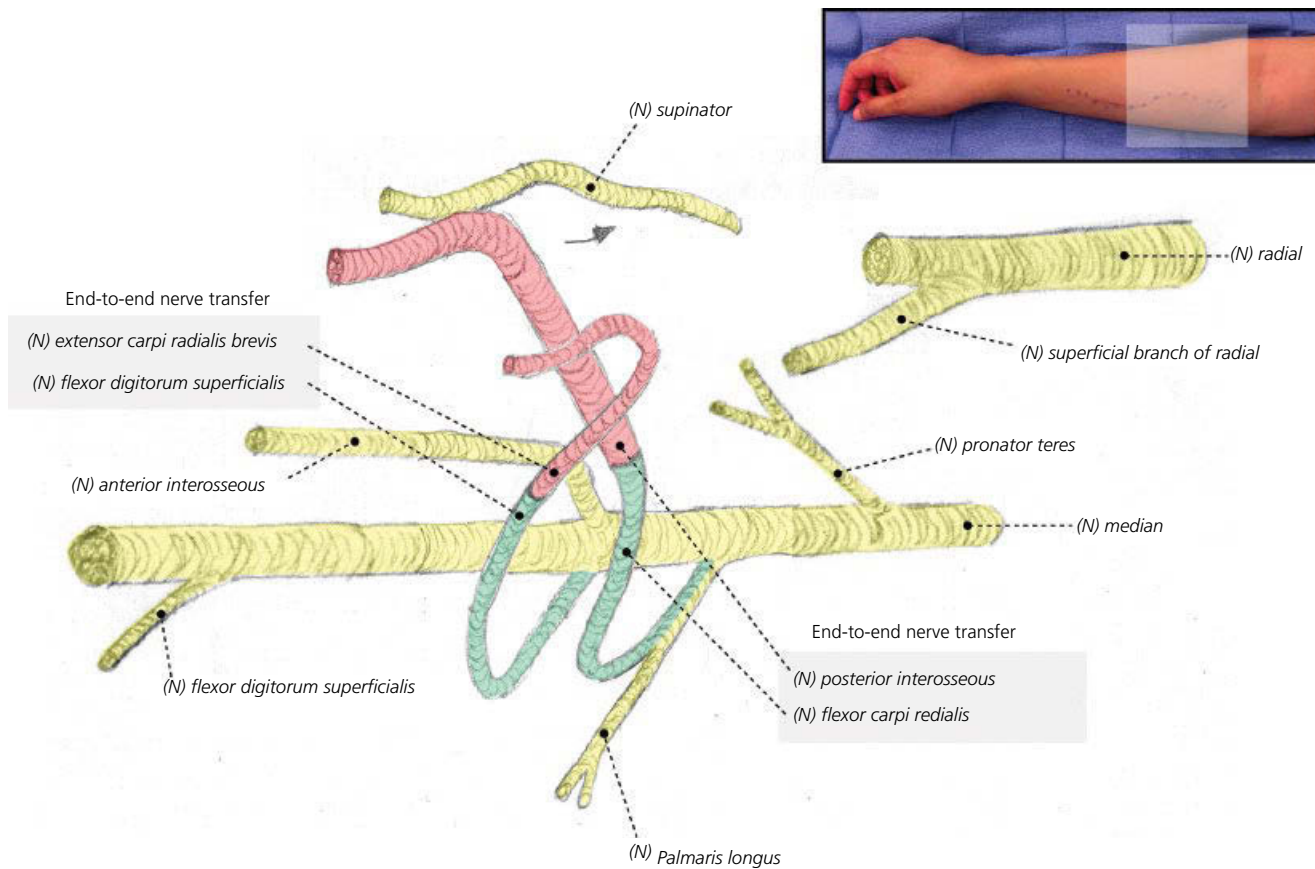


Figure 56.14 Illustrative median to radial nerve transfer. Two nerve transfers occur in the median to radial nerve transfer for restoration of wrist/finger extension. The donors (green) and recipients (red) occur in the following specific sets for optimal results with postoperative rehabilitation: (1) flexor carpi radialis to posterior interosseous (PIN) and (2) flexor digitorum superficialis (FDS) to extensor carpi radialis brevis. PIN and FDS are antagonistic. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

if necessary later. In the case of fractures, in addition to nerve and soft tissue injuries, the bony fixation takes precedence.

Most patients complain in the postoperative period of paraesthesia and electrical shocks, which extend beyond the area of the injury. Most of these can be controlled with neurotropic medications such as nortriptyline or pregabalin. In patients with neuropathic pains, prompt involvement of a pain specialist is critical. Physical therapy and occupational therapy continue for an extended rehabilitative period to prevent joint contractures while the nerve recovery is in progress, to fabricate and adjust protective splints, and to assist in motor and sensory re-education as the recovery process is underway. Whenever possible, our hand and nerve therapists evaluate and advise patients pre-operatively and re-education begins within the first month of surgery, long before results of regeneration are anticipated. Similarly mirror training is used to keep the 'brain open' and receptive for eventual recovery.

Complex regional pain syndrome

Complex regional pain syndrome (CRPS) was documented as early as 1864 during the American Civil War by Silas Weir

Mitchell in his book titled *Gunshot Wounds and Other Injuries of Nerves*, where he describes 'long after the trace of the effects of a wound has gone . . . neuralgic symptoms are apt to linger, and too many carry with them throughout long years this final reminder of the battlefield'.⁵⁶ The term *causalgia* was coined at that time after the Greek words *kausis*, meaning burning, and *algos*, meaning pain. In 1900, Sudeck noted muscle atrophy and demineralization of bone described as 'patchy osteoporosis of the small bones of the hands or feet and the distal metaphysis of the forearm or tibial bones'.⁵⁷ This gave rise to the term *Sudeck's dystrophy*. Almost 50 years later, the term *reflex sympathetic dystrophy* was used to describe what Evans assumed to be sympathetic nervous system involvement with the abnormal activity observed in the affected extremities.⁵⁸ However, the signs and symptoms of this disease did not consistently show sympathetic involvement, a reflex mechanism or the presence of dystrophy. For this reason, the Special Consensus Group of the International Association for the Study of Pain (IASP) created the term *complex regional pain syndrome* in 1994 to allow for a more broad inclusion of patients that showed varying levels of the disease process.

Clinical features and diagnosis

Although there is currently no gold standard to test the diagnosis of CRPS, it is imperative to perform a thorough history and physical examination. The clinical diagnostic criteria developed in 1994 by the IASP consisted of the following:

- 1 Presence of an initiating noxious event or reason for immobilization.
- 2 Disproportional pain, allodynia, or hyperalgesia from a known inciting event.
- 3 Signs or symptoms of any evidence showing oedema, skin changes, blood flow, or abnormal sudomotor activity in the region of the pain.
- 4 No other condition that would otherwise explain the degree of pain or dysfunction.⁵⁹

The sensitivity for this set of diagnostic criteria was high at 0.98 but was unfortunately met with a low specificity of 0.36, leading to an overdiagnosis of the pain syndrome.⁶⁰

In 2007, the Budapest criteria was developed to refine and create stricter guidelines for both research studies and clinical diagnosis.⁶¹

- 1 Presence of continued disproportional pain from the known inciting event.
- 2 Must report at least one symptom in three of the following four categories:
 - Sensory: hyperaesthesia, allodynia
 - Vasomotor: temperature asymmetry, changes in skin colour
 - Sudomotor/oedema: oedema, changes in sweating, sweating asymmetry
 - Motor/trophic: decreased range of motion, motor dysfunction (tremor, weakness, dystonia), trophic changes (hair, nail, skin).
- 3 Must report at least one sign in two or more of the following categories at the time of evaluation:
 - Sensory: hyperalgesia to pinprick, allodynia to touch or joint movement
 - Vasomotor: temperature asymmetry, colour asymmetry
 - Sudomotor/oedema: oedema, asymmetrical sweating, sweating changes
 - Motor/trophic: decreased range of motion, motor dysfunction, trophic changes.
- 4 No other condition that would otherwise explain the degree of pain or dysfunction.

In comparison, the Budapest criteria retained the exceptional sensitivity of the IASP criteria (0.99), but greatly improved upon the specificity (0.68).⁶² CRPS is further divided into two groups (CRPS 1 and CRPS 2), with the distinction being that CRPS 2 has the presence of a definable nerve lesion.

Other common diagnostic tests to aid in the diagnosis of CRPS include three-phase bone scintigraphy which can detect alterations in bone metabolism, quantitative sensory testing which can show impaired mechanical pain thresholds, and infrared thermography which can show differences in temperature in the affected region.

Epidemiology

CRPS is a rare disease with a prevalence of less than 2%.⁶³ The incidence of CRPS (CRPS 1 and 2) is conservatively estimated to be 6.28 per 100 000.⁶⁴ It is more common in younger adults, with a mean age of 41.8 years and with greater frequency in females than males (2.3–3:1).⁶⁵ A survey by Sharma *et al.*, revealed that CRPS more commonly occurs as a result from work-related injuries or surgery and is associated with functional impairment, sleep disturbance and suicidal ideation.⁶⁶

Pathophysiology

Although the majority of patients who develop this syndrome have an initiating inciting event such as trauma, surgery, ischaemia or nerve compression, it is unclear why only some patients develop CRPS. It is becoming increasingly apparent that multiple mechanisms may be involved in the development of this disease. Some of these mechanisms include an alteration to cutaneous sensory innervation with a reduction in C and A-delta fibre density, central and peripheral sensitization, an increase in the release of proinflammatory cytokines and neuropeptides, alterations in sympathetic nervous system function, and altered processing of nociceptive stimuli from cortical reorganization.⁶⁷

Treatment

Unfortunately, there is limited evidence as to the most effective treatment regimens for CRPS. This is partly due to our inadequate understanding of the disease, as well as the greater need for more well-designed, randomized controlled trials. As such, current recommendations are based on treatment modalities for other neuropathic pain conditions, but is largely accepted to be part of a multimodal, multidisciplinary approach. Of utmost importance is that earlier recognition/diagnosis, and thus earlier initiation of targeted therapy, typically achieves greater results.

Pharmacological therapy includes antidepressants such as tricyclic antidepressants (TCAs) and serotonin noradrenaline reuptake inhibitors (SNRIs), which, although not specifically studied for CRPS, have been shown to be quite effective in neuropathic pain. Anticonvulsants and membrane stabilizers such as gabapentin and pregabalin have been shown to be effective in diabetic neuropathy and postherpetic neuralgia. Corticosteroids have been used to help decrease the acute inflammatory process that occurs during CRPS. NMDA receptor antagonists, such as ketamine, showed short-term pain relief, thought to be due to modulation of central sensitization. Bone resorption inhibitors, namely bisphosphonates, has been advocated for use in patients with CRPS as there is often the accompaniment of bone demineralization. Several studies using either intravenous or oral bisphosphonates have shown improvement in both reported pain scores as well as functional assessment in both short-term as well as medium-term outcomes.⁶⁸ More novel pharmacological approaches include the use of free radical scavengers such as dimethyl sulfoxide (DMSO) 50% and *N*-acetylcysteine (NAC), which have shown medium-term benefit.

Interventional strategies include sympathetic nerve blocks, stellate ganglion or lumbar sympathetic, for both diagnostic and treatment purposes. As a diagnostic tool, these blocks to help differentiate sympathetically maintained pain from sympathetically independent pain. Should these blocks provide relief, they typically do not last greater than 7 days. As such, these injections should not be performed as the sole form of treatment, but as part of a multidisciplinary regimen that includes physical therapy where these interventions allow patients to progress further with physical therapy and with modalities such as desensitization to a more restorative functional goal. A small study showed that intrathecal baclofen can be useful for otherwise treatment-resistant dystonia that may develop as a result of CRPS.⁶⁹ Spinal cord stimulation (SCS) has been shown to be beneficial in patients with CRPS who have failed conventional therapy. In a 5-year follow-up study, SCS plus physical therapy (PT) showed a greater reduction in pain than PT alone at 2 years, though at the 5-year mark, both groups had similar results.⁷⁰

Physical and occupational therapy with the goal of functional restoration continues to be the benchmark for successful CRPS treatment. Exercises that target myofascial release, isometric strengthening and increased range of motion as well as desensitization techniques should be instituted as soon as possible for the best results. As an increasing amount of evidence supports cortical reorganization as a component of CRPS, there is also an increasing amount of evidence that supports the beneficial effects of graded motor imagery (GMI). Graded motor imagery is a three-stage programme consisting of implicit motor imagery (left/right handedness), explicit motor imagery (imagined movements) and mirror therapy. A randomized controlled study showed that at 6 and 12 weeks, those patients who underwent GMI had significantly less pain and swelling. There is definitely a role for nerve surgery in the management of CRPS. We have had success in improving pain relief in patients with CRPS 1 when a nerve injury is the inciting factor (e.g. carpal tunnel syndrome following wrist fracture) and of course in CRPS 2 when pain is localized to a specific nerve injury.

Summary

Primary neurorrhaphy remains the gold standard for all nerve repair techniques, whereas an autologous nerve autograft is the gold standard for bridging a nerve defect. Since the first implementation of a nerve allograft in 1870 by Philipeaux and Vulpian, significant contributions regarding suturing technique, neural topography and the biology of nerve regeneration have transformed the way we approach nerve reconstruction. Although primary neurorrhaphy and autografts are the most common methods for repair, several newer options are at our disposal. Nerve transfers have revolutionized our approach for treating devastating brachial plexus injuries, converting them to highly selected upper and lower motor and sensory nerve injuries.

Nerve allografts and conduits offer alternatives to autologous nerve grafting when needed. However, after decades of research towards defining the best nerve substitutes, we still struggle to find an alternative as good as a nerve autograft even for small diameter/short gap injuries. For these short injuries, autograft material is readily available. Intuitively, culturing recipient Schwann cells to repopulate a conduit or acellularized nerve allograft is appealing. This concept has, however, been explored for decades in the laboratory without clinical translation. It is unlikely that modifications of nerve conduits or acellularized allografts are going to provide a nerve graft alternative in the near future for injuries where the need is greatest (long-gap/large-diameter nerve injury). It is likely that other avenues of basic research will lead to the 'next' meaningful advance in this field.⁷¹

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CHAPTER 57

Brachial plexus injuries and reanimation

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Anatomy

The brachial plexus comprises a complex grouping of nerve fascicles, giving function and sensation to the upper extremity. Progressing from proximally to distally, the plexus is divided into regions termed roots, trunks, divisions, cords and branches. Within these nerves, there is a clearly defined internal topography that will guide future reconstructive efforts (Figure 57.1).

Roots

The traditional brachial plexus has contributions from cervical roots C5–C8 and thoracic root T1. Anatomic variations of this included a prefixed plexus, which has additional contribution from C4, and a postfixed plexus, which has additional contribution from T2. The spinal roots pass between the anterior and middle scalene muscles. There are three nerve branches exiting from the root level. The dorsal scapular nerve arises from the C5 nerve root and supplies the rhomboid muscles and the levator scapulae muscle. The long thoracic nerve arises from roots C5, C6 and C7 and supplies the serratus anterior muscle, which stabilizes the scapula. The nerve to subclavius arises from the distal C5 and C6 nerve roots. This nerve supplies the subclavius muscle, which is unable to be tested on clinical examination.

Trunks

In the inferior portion of the posterior cervical triangle, the roots combine to form trunks. The upper trunk is formed from the combination of the C5 and C6 nerve roots. The middle trunk is a continuation of the C7 root. The lower trunk is formed from C8 and T1. There is a single branch off the upper trunk that is critical for shoulder function. The suprascapular nerve departs from the upper trunk, travels deep to the trapezius muscle and runs through the suprascapular notch of the scapula to supply the supraspinatus and infraspinatus muscles.

Divisions

Passing deep to the clavicle, each trunk divides into an anterior and posterior division. There are no extraneous branches at the level of the divisions.

Cords

The divisions pass deep to the pectoralis minor muscle and combine in a predictable fashion to form the cords. The cords are named in relation to their anatomic location around the axillary artery. The anterior divisions of the upper and middle trunks combine to form the lateral cord. The medial cord is the continuation of the anterior division of the lower trunk. All of the posterior divisions combine to form the posterior cord.

Branches

There are several branches that arise from the cords. The lateral cord gives off the lateral pectoral nerve, which innervates the clavicular head of the pectoralis major. The medial cord gives off the medial pectoral nerve, which innervates the sternocostal head of the pectoralis major muscle. The medial brachial cutaneous and medial antebrachial cutaneous nerves also branch from the medial cord supplying sensation to the medial aspect of the upper arm and forearm. More distally, the lateral and medial cords each contribute to the median nerve, supplying the sensory and motor functions of the nerve respectively. This relationship continues distally and is useful for understanding the internal topography of the median nerve, where motor fascicles are located on the medial side of the nerve and sensory fascicles remain more lateral. At the level of the wrist, the most radial fascicles of the median nerve will supply the first webspace. Following its contribution to the median nerve, the lateral cord becomes the musculocutaneous nerve. This nerve innervates the coracobrachialis, biceps brachii and brachialis. The terminal portion of this nerve is the lateral cutaneous nerve of the forearm. After contributing to the median nerve, the medial cord becomes the ulnar nerve, which, after innervating the flexor carpi ulnaris and the small and ring flexor digitorum profundus muscles, is primarily responsible for all of the intrinsic musculature of the hand.

The internal topography of the ulnar nerve can best be remembered as sensory–motor–sensory, where the most medial portion of the nerve will eventually contribute to the dorsal cutaneous ulnar branch, exiting the main portion of the ulnar

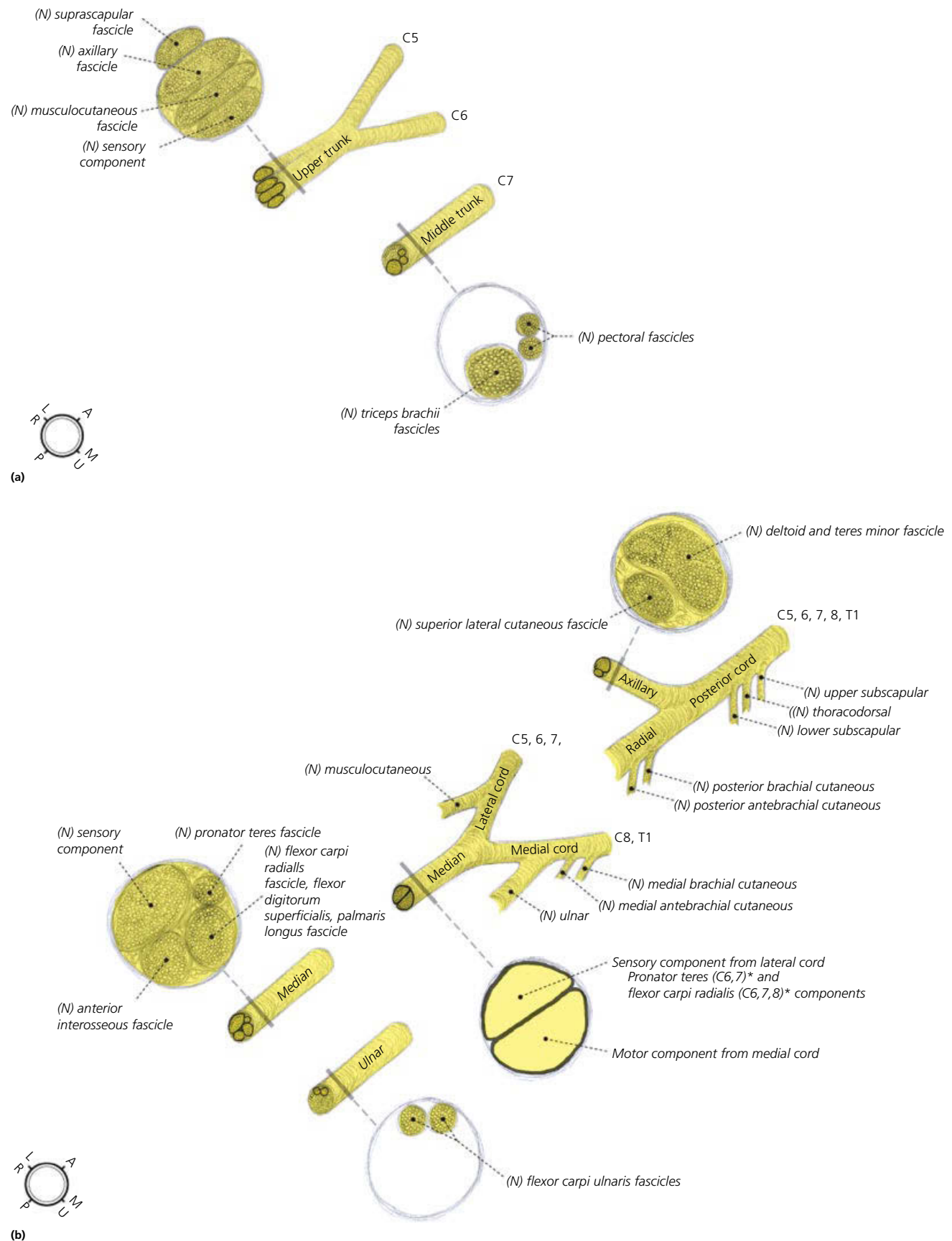


Figure 57.1 Internal topography of the brachial plexus. **(a)** Cross-sections of the upper and middle trunks delineating fascicular grouping and **(b)** internal topography at the level of the cords and branches. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

nerve approximately 9 cm proximal to the wrist crease and providing sensation to the dorsal ulnar aspect of the hand. After this division, the more medial 40% of the ulnar nerve will pass through Guyon's canal to become the deep motor branch and the more radial 60% will contribute to the superficial sensory branch (Figure 57.2).

The posterior cord gives off the upper subscapular nerve, the thoracodorsal nerve and the lower subscapular nerve. These branches innervate the superior portion of the subscapularis muscle, the latissimus dorsi muscle and the lower subscapularis/teres major muscles, respectively. The two most terminal branches of the posterior cord are the axillary nerve and the radial nerve. The axillary nerve travels through the quadrangular space (bounded by the humerus, the long head of triceps brachii, teres minor and teres major) with the posterior circumflex humeral artery. It innervates the deltoid and the teres minor, but also provides sensation to a patch of skin overlying the deltoid, sometimes known as the 'soldier's patch'. This sensory component is located at the most inferior portion of the axillary nerve. The radial nerve travels through the triangular interval (bounded by the teres major and the lateral and long heads of triceps brachii) with the profunda brachii artery. Wrapping around the spiral groove, the radial nerve goes on to supply the elbow, wrist, finger and thumb extensors.

Clinical history and assessment

Brachial plexus injuries occur in approximately 1% of multi-trauma victims, most frequently in young males.¹ The vast majority are due to motor vehicle collisions and of these, up to 84% may involve motorcycles.^{2,3} Due to these circumstances, many patients present with multisystem trauma requiring resuscitative procedures. Associated closed head injuries including loss of consciousness can occur in almost three quarters of patients, which can often result in a delay of recognition of a brachial plexus injury.¹ It is not uncommon for the patient to be the first to identify the injury. In the obstetrical population, approximately 1.6% of deliveries have a shoulder dystocia and of these, almost 20% experience a brachial plexus palsy.⁴

Acquiring a thorough history is critical in the investigation of a patient with a brachial plexus injury. The mechanism of injury may provide information to recognize a pattern of injury or to estimate the degree of injury. In a brachial plexus injury, information about birth weight, gestational age, shoulder dystocia, delivery aides such as forceps and duration of labour are useful. For a trauma, details about a motor vehicle collision, including the speed, direction of impact, use of safety equipment such as seatbelts or helmets, and concomitant injury may

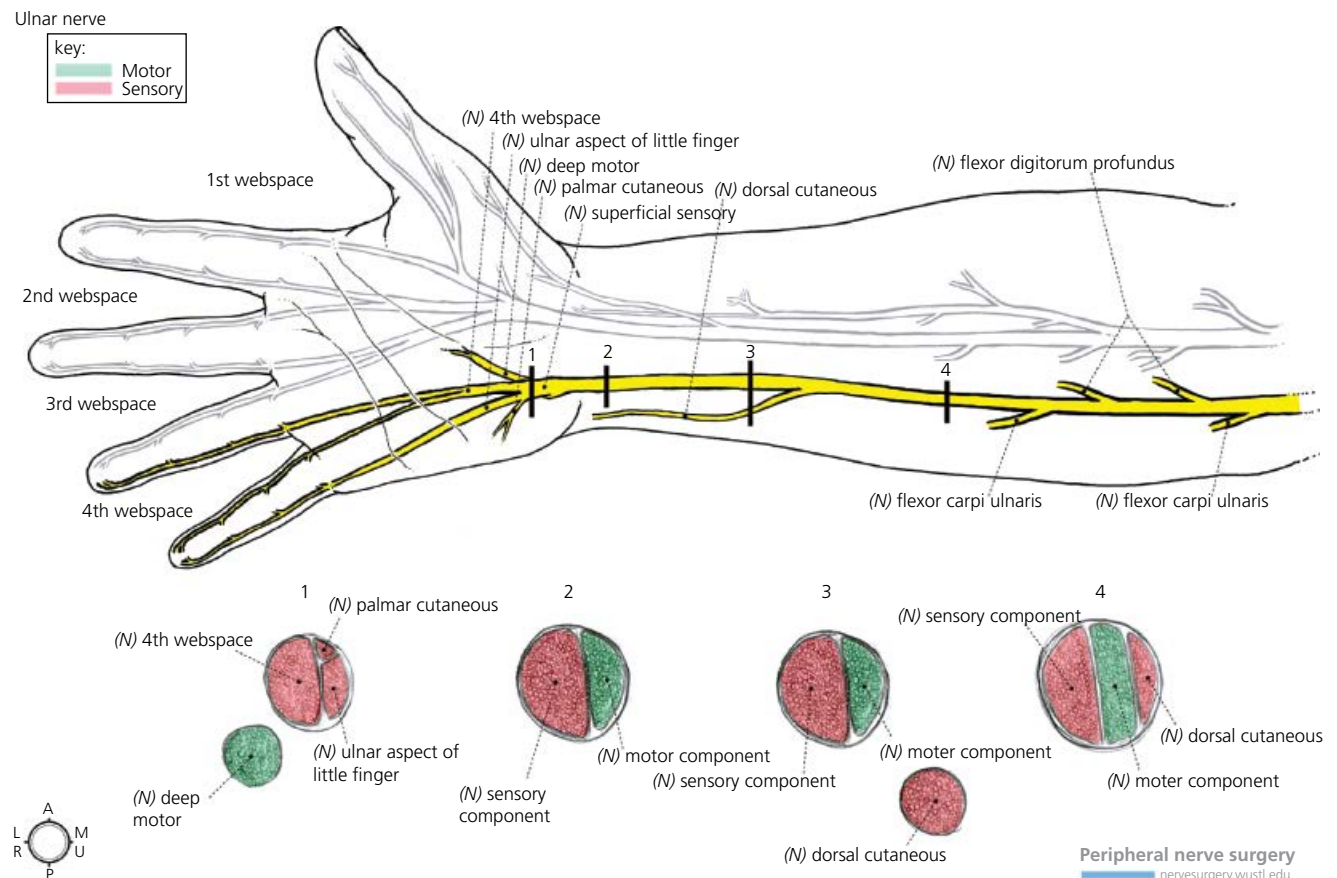


Figure 57.2 The internal topography of the ulnar nerve. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

be useful in ascertaining risk to certain structures. In the event of a blow or a fall, knowing the direction and weight of impact, location of impact and arm position can contribute valuable information. Knowledge of pre-existing upper extremity injuries and medical comorbidities is also useful. The patient's general medical status may contribute to candidacy for surgical intervention.

An important component of this history is related to pain. Patients should be questioned regarding the severity, location and nature of their pain to assist in determining musculoskeletal versus neuropathic components. Severe pain can be suggestive of root avulsions. Inclusion of a pain questionnaire at the initial assessment and then at subsequent visits can provide both quantitative and qualitative assessment of pain. Having a low threshold for starting neuropathic pain medications or involving a pain specialist is crucial for these patients, as pain management can be a significant problem.⁵ In addition, patients should be screened for depression and mood disorders, as the incidence in this patient population can be as high as 40%.⁶

In this patient population, serial clinical examination is crucial to determine spontaneous recovery. At the initial physical examination, a complete upper extremity sensory and motor examination should be performed with care to document both peripheral and central nervous system examination. Atrophy may not initially be present but will later mark denervated muscle groups. Having a systematic approach to the brachial plexus examination will allow for determination of patterns of injury and more specifically about level of injury. A thorough knowledge of the anatomy of the plexus can help to localize lesions simply by physical examination.

The location of scars on the upper extremity, shoulder and neck should be noted, from either trauma or surgical intervention. Joint mobility should be assessed for the entire extremity, and, in the setting of any decreased passive range of motion or stiffness, early intervention with an occupational or physiotherapist should be arranged to optimize overall outcome.⁷ Assessment should be carried out for concomitant findings such as a Horner's sign (ptosis, miosis and anhydrosis) which occurs due to injury to the cervical sympathetic fibres during avulsion of the C8 and T1 roots.⁸

Perhaps the most critical part of the physical examination is accurate documentation. Careful recording of the Medical Research Council (MRC) scale for muscle strength for each muscle group in an organized fashion will allow comparison over time. Given the known rate of recovery following nerve injury of 1 mm/day, this allows for assessment of whether spontaneous recovery is progressing at the expected rate, or whether surgical intervention may be appropriate. It should be remembered that examination for potential donors is as important as identifying non-functioning muscle groups. The preparation for surgical reconstruction of brachial plexus injuries is the biggest determinant of success.

Aetiologies

The aetiology of brachial plexopathy can be traumatic, iatrogenic, compressive, neuralgic amyotrophy or radiation.

Traumatic

The majority of brachial plexopathies secondary to trauma occur in young males, with up to 90% of cases comprising this patient population.¹ Traumatic injuries to the brachial plexus can be divided into open and closed injuries. Open injuries are those secondary to penetrating injury, such as knives or other sharp objects. These necessitate immediate surgical exploration. The majority of traumatic brachial plexus injuries are closed, and these can be composed of crush injuries or traction injuries. Crush injuries tend to occur after motor vehicle accidents, whereas traction injuries are more likely to be related to motorcycle accidents, skiing, falls and sports injuries.¹ In traction injuries, displacement of the upper extremity inferiorly combined with neck flexion contralaterally causes an increase in the neck-shoulder angle, which results in injury to the upper roots and trunks.⁹ This commonly occurs in both high-velocity injuries such as motorcycle collisions and also in lower velocity accidents such as sports injuries. The opposite can occur with strong upward force or traction on the upper extremity increasing the scapula-humeral angle and causing injury to the lower plexus. The brachial plexus is susceptible to these injuries due to limited mobility secondary to prevertebral fascia and attachment of the cervical roots to the vertebrae. Traction injuries are also responsible for obstetrical brachial plexopathies, most commonly following shoulder dystocia.⁴ Risk factors for obstetrical plexus injury include high birth weight, fetal acidosis, posterior arm involvement and narrow pelvis.¹⁰

Iatrogenic

During long operations, positioning of the upper extremity can contribute to a traction palsy.¹¹ Most commonly these injuries are neuropraxic in nature and will recover spontaneously with time, however occasionally a more severe injury will occur. There are patient factors that contribute to the propensity for developing these injuries, including diabetes, hypothyroidism, prior injury, hereditary neuropathy with liability to pressure palsy (HNPP) and obesity.¹¹

Compression

Compression can be secondary to structures adjacent to the plexus in an enclosed space such as malignancy, callous formation, haematoma or pseudoaneurysm. Anatomic variants may be responsible for compression primarily or may increase the propensity for trauma to the brachial plexus. These variants include cervical ribs, prominent transverse processes and congenital fibrous bands.¹² Compression often occurs in the costoclavicular space as the brachial plexus travels between the clavicle and the first rib. Traumatic injuries can result in swelling

or haematoma that cause compression of the brachial plexus. Occupational injuries where a heavy object lands on the shoulder or neck are an example.

Neuralgic amyotrophy

Brachial neuritis, or Parsonage–Turner syndrome, is a brachial plexopathy of unclear aetiology that most commonly involves the lower brachial plexus; the axillary nerve is implicated in up to 70% of patients.^{13–15} Most commonly, these non-traumatic plexopathies present with a history of severe neuropathic pain that resolves and then is followed by patchy nerve paresis.¹⁶ Possible contributory factors are viral disease, infection, immunization, trauma, surgery and autoimmune mechanisms.¹⁷ The majority of these plexopathies will have a full spontaneous recovery within approximately 3 years, however the prognosis is more guarded in the setting of other medical comorbidities such as diabetes.¹⁸

Radiation

Transient or permanent brachial plexopathy can occur as a result of radiation therapy. Approximately 1.2% of women receiving radiation as adjuvant therapy for breast cancer, higher following chemotherapy, will present with clinical manifestations anywhere from six months to 20 years following treatment (median 1.5 years).¹⁹

Investigations

Imaging

Brachial plexopathy and level of injury are primarily a clinical diagnosis supported by electrodiagnostic studies, however there are several circumstances where imaging contributes valuable information.

Plain films are useful for identifying fractures that might be associated with certain patterns of brachial plexopathy. For example, a transverse process fracture is more likely associated with a root avulsion injury because of the deep cervical fascia that tethers cervical root to transverse process.²⁰ Clavicular fractures can cause sharp injuries to the plexus on bony fragments, or can contribute to compression injuries related to either haematoma acutely, or callus formation in a more delayed fashion.

CT myelography is a useful investigation for assessing for root avulsion. The use of contrast material allows for visualization of the dural sheath around the nerve root, which is not pathognomonic for a root avulsion, but can be suggestive.²¹ The absence of a root at a particular level is diagnostic. When obtained at least one month post injury, which allows time for the dural sheath to heal improving distribution of the dye, CT myelography is associated with a sensitivity of 95% and a specificity of 98% for root avulsion injuries.²²

MRI is becoming a more valuable tool for the imaging of the brachial plexus due to newer techniques and protocols. The

advantage of MRI is that it can directly visualize the entire brachial plexus and is non-invasive. Soft tissue injury is more easily assessed and three-dimensional imaging is possible.

Electrodiagnostic studies

It is crucial that all patients presenting with brachial plexopathies receive electrodiagnostic studies, and in many cases serial studies will confer helpful information in the decision to proceed with surgery. Electrodiagnostic studies are not useful before Wallerian degeneration has occurred and fibrillation potentials have appeared, therefore delaying investigation for approximately six weeks is beneficial. There are two components to the electrodiagnostic study: nerve conduction and electromyography. Nerve conduction studies are traditionally more useful for compression neuropathies, although they can portray valuable information in the setting of a preganglionic lesion to the brachial plexus. In these injuries, the dorsal root ganglion remains intact and the peripheral sensory nerve does not undergo Wallerian degeneration. The motor nerve, however, arising more proximally in the spinal cord, will still be affected. The resultant nerve conduction studies will demonstrate intact sensory nerve action potentials (SNAPs) but no volitional activity.

The electromyography portion of the study also has several components. On insertion of the needle, a denervated muscle will exhibit spontaneous discharges or fibrillations and positive sharp waves. In contrast, a normal muscle will not have any waveform on insertion alone. When the patient is asked to voluntarily contract the designated muscle, the presence of motor unit potentials (MUPs) signifies that the action potential is reaching the desired motor endplate. Nascent units are representative of connections to the motor endplates from regenerating injured axons, while MUPs signify distal motor sprouting from uninjured axons. Both are good prognosticators of a spontaneous recovery without surgical intervention.²² Electromyography often precedes clinical signs of recovery by 1–2 months, and the presence of new MUPs and nascent units can be a promising prognostic sign before the patient notices any changes. In addition, electrodiagnostic studies can be valuable in preoperative planning, especially where nerve or tendon transfers are being contemplated.

Patterns of injury

Brachial plexus injuries can occur in any potential combination, however there are several common patterns of injury that warrant mention, as the approach to treatment can be dictated by the predictable deficits.

Upper plexus injuries

These injuries comprise injuries to the C5 and C6 roots or to the upper trunk. This occurs when the shoulder is wrenched away from the head and neck. Classically these patients will present

with deficits of shoulder abduction, external rotation and potentially loss of elbow flexion. There will be a concomitant loss of sensation over the deltoid muscle. A sulcus sign may be present, identifying the inability of the shoulder girdle to support the weight of the arm (Figure 57.3). Priorities are to re-establish elbow flexion and then to restore shoulder stability and function.

Lower plexus injuries

Injuries to the lower plexus typically involve C8 and T1 roots or the lower trunk. This occurs when the arm is forcefully abducted above the head. In this pattern of injury, wrist and hand flexion will be the most greatly affected, with intrinsic muscle paralysis and potential clawing. Loss of sensation in the ulnar nerve distribution accompanies the lower plexus injury. Priorities include restoration of hand function.

Posterior cord injury

Injury to the posterior cord involves contributions from C5–T1. When injured, extension of the elbow, wrist, thumb and fingers are affected, as is shoulder abduction from the axillary nerve, which is a terminal branch of the posterior cord. Priorities are to restore shoulder abduction and wrist, finger, and thumb extension. Restoration of elbow extension is more controversial.

Pan-plexus injuries

This injury involves the entire plexus (C5–T1) and presents with a flail, insensate upper extremity. Associated with a poor prognosis, this extremity will likely be a ‘helper extremity’ at best. Priorities to restore function include elbow flexion, shoulder stability and abduction/external rotation, and hand function. Often associated procedures such as wrist arthrodesis will be considered given the lack of potential donor nerves or tendons for surgical intervention.

Obstetrical brachial plexus injuries

Classically, these patients present with an Erb’s palsy, which is injury to the upper plexus. This leads to loss of shoulder abduction, external rotation and loss of elbow flexion. These patients are often



Figure 57.3 A sulcus sign secondary to denervation of the left axillary and suprascapular nerves. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

described as presenting in the ‘waiter’s tip’ position, where the arm hangs by the child’s side with the shoulder internally rotated, the elbow extended, and the forearm pronated. Alternatively, these children can present with a Klumpke’s palsy, which is a lower plexus lesion. These children present with paralysis of multiple muscle groups, including the hand and wrist flexors and the intrinsic muscles of the hand. They will often have an associated claw deformity and potentially an associated Horner’s syndrome, which presents as ptosis, miosis and anhydrosis. They will have a sensory deficit in the ulnar nerve distribution.

Management principles and operative approach

Timing

The management of brachial plexus injuries is determined by the mechanism, level and degree of injury. Open injuries of the brachial plexus should be explored within 72 h to allow for intraoperative stimulation of the distal nerve segment. Depending on the mechanism, acute nerve repair may be warranted. If, however, the mechanism is other than a sharp, transecting injury, it is important to consider the zone of injury. In the event of a crush or avulsion, identification and intraoperative tagging of the nerve may be prudent, followed by re-exploration approximately three weeks after the injury when the zone of injury is well demarcated. If acute repair is desired, both the distal and proximal nerve ends should be resected to ensure healthy fascicular nerve ends. The authors advocate the use of a bread-loafing technique to ensure that adequate scar is resected while preserving optimum nerve length, by sequentially cutting the nerve in thin slices until clearly past the zone of injury and able to visualize healthy cut fascicular ends.

In the event of resection of the damaged nerve, a nerve gap will be present. Addressing the nerve gap is critical to avoid tension on the nerve repair.²³ The nerve gap can be addressed with nerve grafting. Potential donors include the medial antebrachial cutaneous nerve (MABC) or the sural nerve. Our preference is to use the MABC given that it is located within the injured extremity, is expendable, has a larger calibre than the sural nerve, and sensation can potentially be restored into the donor deficit by transferring the distal end to an intact sensory donor in the upper arm. Although nerve grafts can be used to span the distance of a nerve gap, such as following neuroma resection, debridement of nerve ends, or for nerve transfers where the donor and recipient are not in close enough proximity, they involve two coaptations, which reduces the number of successfully traversing axons.^{24,25}

Another potential option for addressing the nerve gap is to use distal nerve transfers. There are several advantages conferred by this technique, including avoiding operating within the zone of injury, conversion of a proximal injury to a more distal one with axon regeneration closer to the motor endplates, and a single coaptation.^{23,24} Tendon transfers and

functional muscle transfers are options for dealing with injuries that present in a delayed fashion or that have minimal donor options.

Closed injuries, such as traction or crush injuries, are significantly more common than open injuries to the brachial plexus. These injuries are not immediately explored, but are instead followed with serial clinical examination and electrodiagnostic studies to evaluate for any spontaneous functional recovery. With these injuries, it is imperative to be cognizant of timing. Peripheral nerve regeneration occurs at a rate of approximately 1 mm/day.²⁶ The distance from injury to the first expected nerve branches to show recovery in an injured nerve should be documented and serially examined for signs of recovery within the anticipated time period. Permanent atrophy and fibrosis of denervated muscle occurs between 12 and 18 months following nerve injury and therefore it is necessary to intervene surgically in a timely manner to achieve reinnervation to the motor endplates prior to this occurring.²³

Priorities to establishing function

The first priority in a complete brachial plexus injury is to restore elbow flexion. This is followed by shoulder stability/range of motion and subsequently restoration of hand and wrist function.

Exploration of the brachial plexus

Exploration of the proximal brachial plexus takes place through a hockey stick shaped incision along the posterior border of the lower sternocleidomastoid muscle and extending transversely above the clavicle. This allows access to the roots with careful dissection and mobilization of the supraclavicular fat pad. The roots travel between the anterior and middle scalene muscles. Caution to avoid the phrenic nerve, which runs longitudinally along the anterior surface of the anterior scalene muscle, is imperative. To explore the more distal plexus, and infraclavicular exposure is performed, with an incision extending from the clavicle to the anterior axillary fold in the deltopectoral groove. We perform this incision in a zig-zag fashion to allow for improved exposure and to prevent scar contracture. The pectoralis major muscle is temporarily divided and reflected medially, as is the pectoralis minor. The patient should be positioned supine with the chest and ipsilateral upper extremity prepped and the arm abducted to 90° and the head turned gently to the contralateral side. Paralytic agents are avoided on induction to allow for intraoperative nerve stimulation. Traditionally, exploration and grafting at the level of the plexus was the gold standard for these injuries, but more recently, there has been a momentum to more distal nerve transfers.²⁷ The authors' preference is for distal nerve transfer.

Restoring elbow flexion

There are a number of different potential donors to re-establish elbow flexion. This necessitates exposing the musculocutaneous nerve in the upper arm. A longitudinal incision is made

along the brachial sulcus on the medial upper arm. The musculocutaneous nerve travels deep to this sulcus in between the biceps brachii and brachialis muscles and can often be palpated against the humerus in the proximal incision. Once this nerve is identified, the branch to the biceps brachii, which branches laterally into the muscle at approximately the upper third, lower two thirds junction of the upper arm, and the brachialis branch, which branches medially closer to the mid-upper arm can be localized and stimulated. The continuation of the musculocutaneous nerve becomes the lateral antebrachial cutaneous nerve, which can be confirmed by the 'tug test', which will put traction on the skin of the proximal forearm when the nerve is gently tugged.

When it has been confirmed that the musculocutaneous nerve does not stimulate, appropriate donors can be selected for nerve transfer. Our preference would be to perform the double fascicular nerve transfer (DFT), which restores function to both the brachialis and biceps brachii branches of the musculocutaneous nerve (Figure 57.4). This procedure involves borrowing redundant fascicles from the flexor carpi radialis or palmaris longus from the median nerve and the flexor carpi ulnaris from the ulnar nerve as donors.²⁸ If the median and ulnar nerves are involved distally, other potential donors include the medial pectoral nerve or the thoracodorsal nerve. In patients presenting with a pan-plexus injury, extraplexal donors are required. The most commonly used extraplexal donor to restore elbow flexion is the intercostals, which can be harvested through a curvilinear incision in the inframammary fold. Approximately three levels are selected as donors and often the nerve to the rectus abdominis is taken as an additional source of motor fascicles. With meticulous dissection distally, the use of a nerve graft can be prevented as the donor nerves are passed through a subcutaneous tunnel in the axilla to the recipient musculocutaneous nerve in the upper arm. Remembering the mantra 'donor distal, recipient proximal' to maximize length and avoid tension on the transfer is essential.

The use of other extraplexal donors has been described, including the phrenic nerve and the contralateral C7, however these are less favourable options due to distance from the recipient nerve and donor deficit.^{29–31}

Restoring shoulder function

Restoration of shoulder function is the next priority and involves regaining shoulder stability, abduction and external rotation. In order to accomplish this, reinnervation of the axillary nerve distribution (deltoid, teres minor) and suprascapular nerve (supraspinatus, infraspinatus) are required. Our preference for addressing the suprascapular nerve is the spinal accessory to suprascapular nerve transfer (Figure 57.5). This transfer is possible from either an anterior or posterior approach. The anterior approach has the advantages of supine patient positioning and the ability to perform the transfer in an end-to-side manner, which preserves spinal accessory nerve function. With the posterior approach, release at the

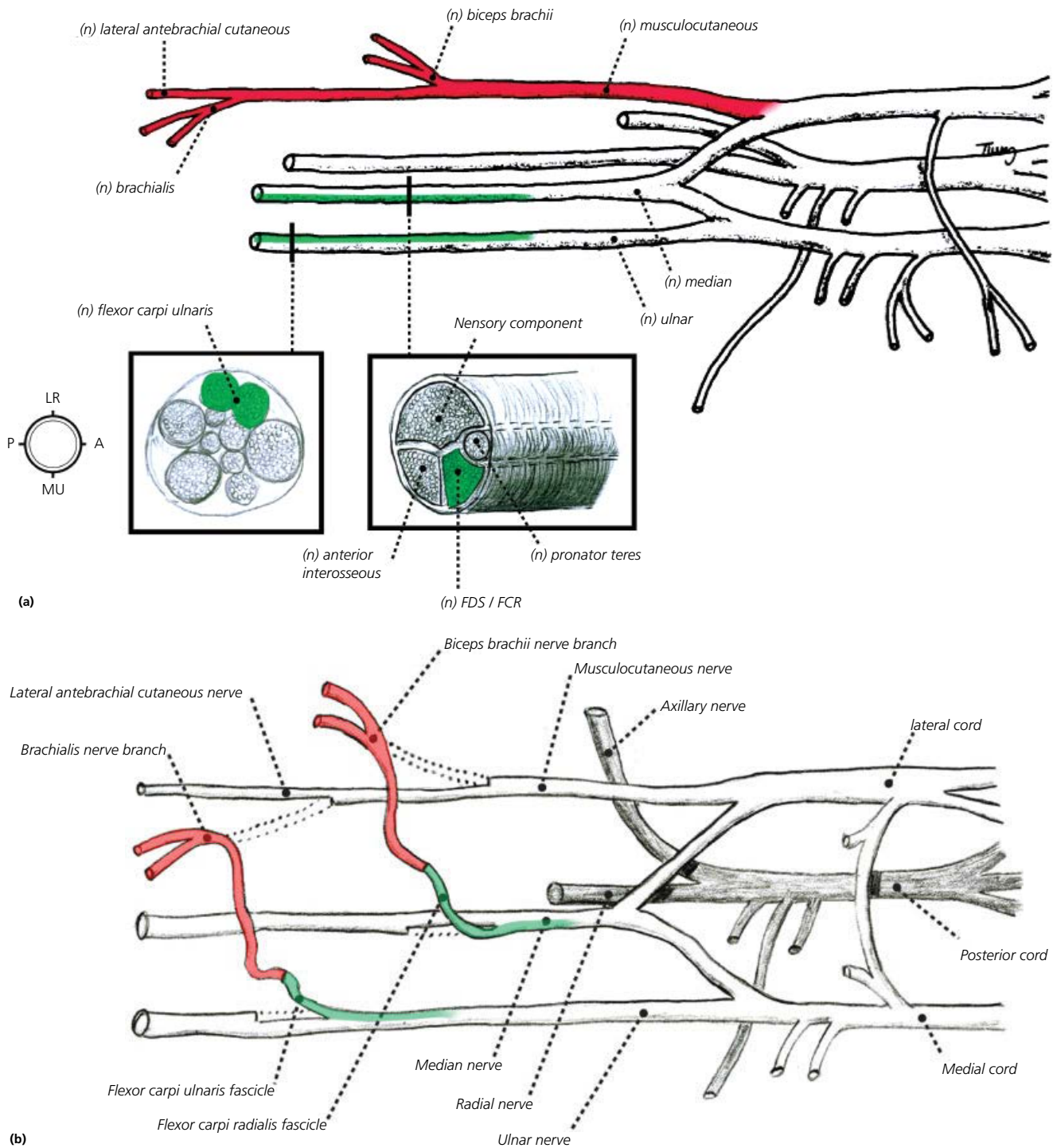


Figure 57.4 The double fascicular nerve transfer (DFT). (a) The internal topography of the median and ulnar nerves delineating the desired motor donors (green) and the injured musculocutaneous nerve recipients (red). (b) A schematic illustrating the donor and recipient pairings of the DFT. (c) Intraoperative photo illustrating the isolated motor donor fascicles from the median and ulnar nerve and the divided musculocutaneous recipients draped over to facilitate deciding on location of coaptation. (d) Intraoperative photo illustrating the two transfers coapted with no tension. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

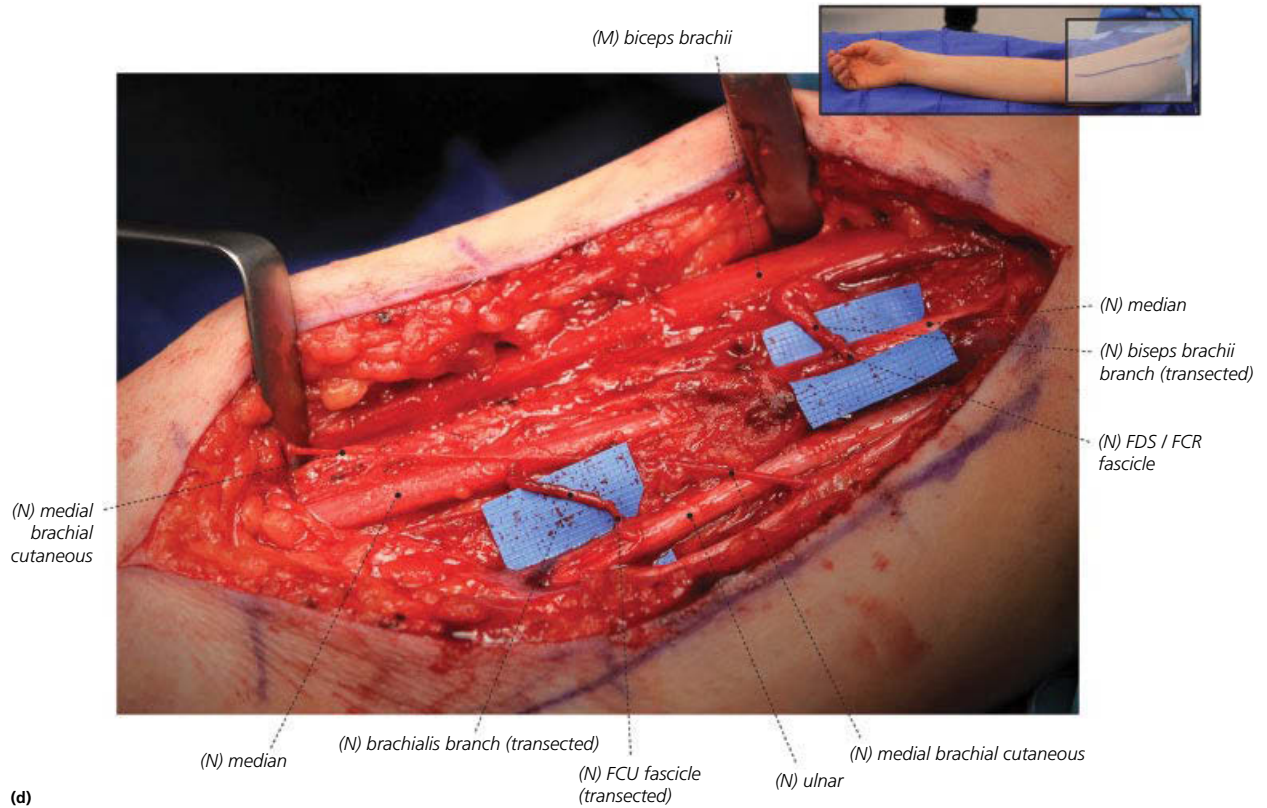
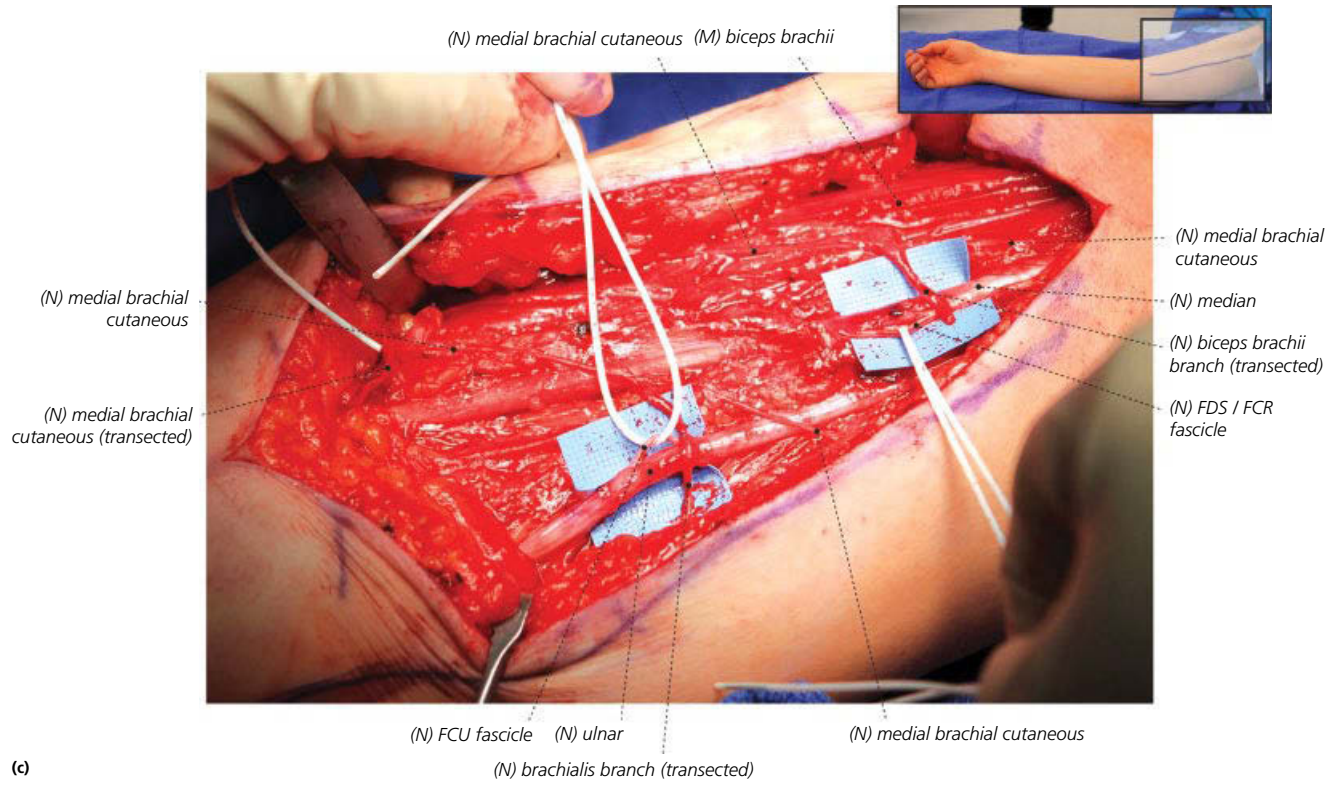
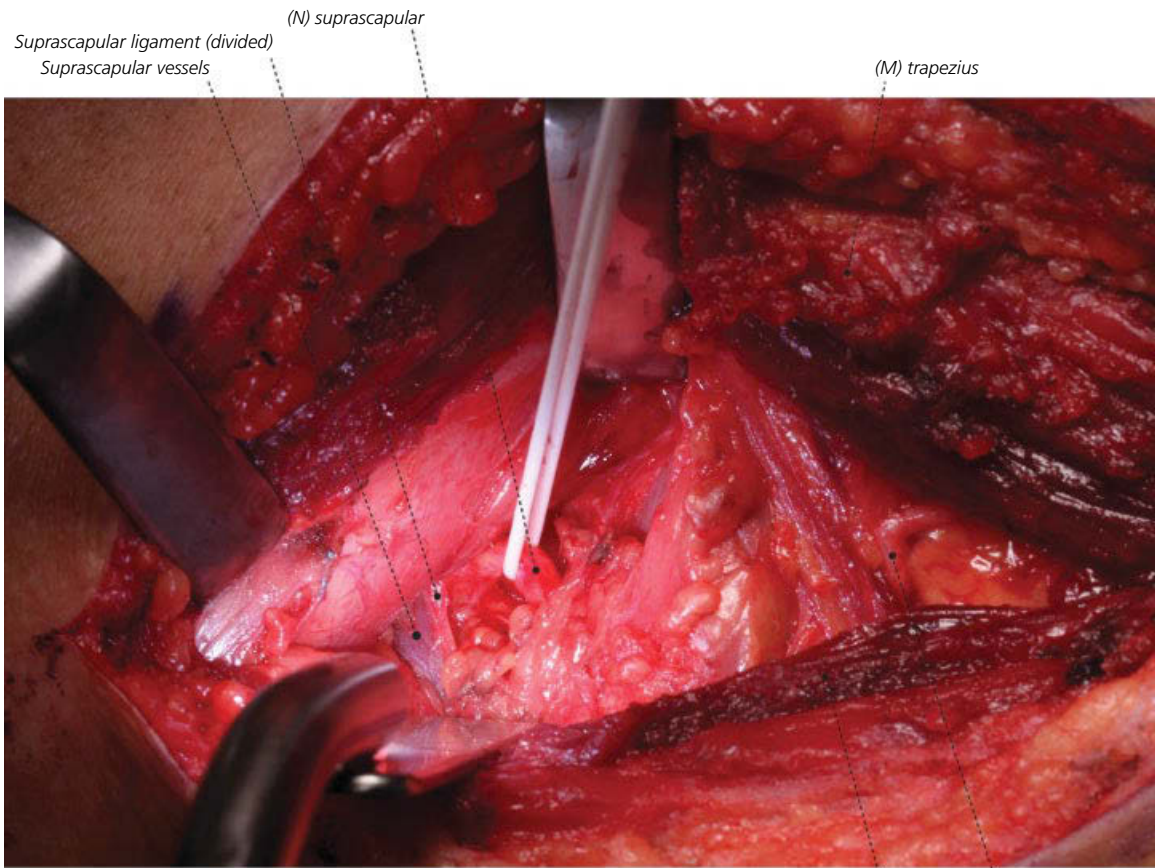


Figure 57.4 (Continued)

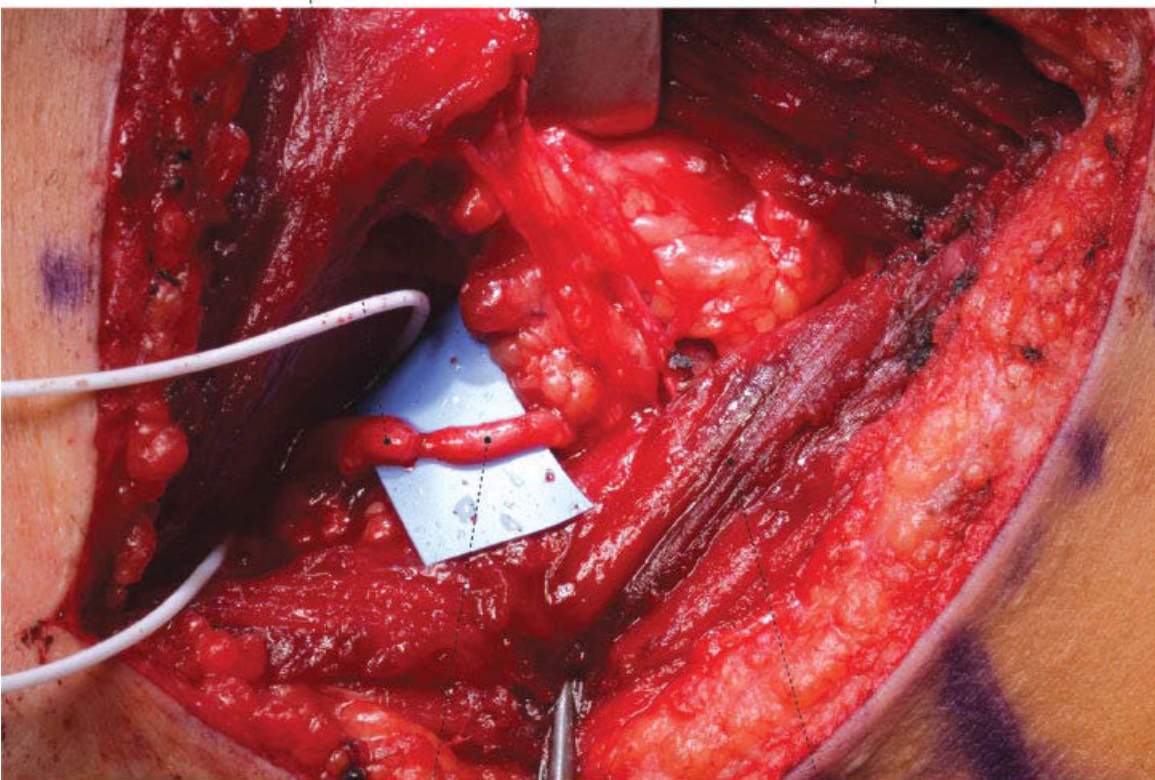


(a)

(N) suprascapular (proximally transected)

(N) spinal accessory
(M) trapezius

(M) trapezius



(b)

(N) spinal accessory (distally transected)

(M) trapezius

Figure 57.5 Spinal accessory to suprascapular nerve transfer from posterior approach. (a) Intraoperative photo documenting the location of both donor spinal accessory and recipient suprascapular nerves and (b) coaptation under no tension. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

suprascapular notch, preserving more innervation to the trapezius, and a wider exposure are advantages.

The spinal accessory nerve is located on the deep surface of the trapezius muscle approximately 40% of the way along a line drawn from the spine to the acromion at the level of the superior scapular border.²³ The suprascapular nerve travels through the suprascapular notch at the midway point along the superior scapular border and is most easily accessed by release of the ligament that travels over the notch.

Shoulder abduction is addressed by reinnervation of the deltoid and teres minor. Our preference is to transfer the medial branch of triceps, which travels along the superficial surface of the radial nerve as its own fascicular group, to the axillary nerve (Figure 57.6). This can be performed by an incision on the posterior arm that extends from the quadrangular space, where the axillary nerve can be located, to the distal upper arm. It is easily performed in conjunction with a spinal accessory to suprascapular nerve transfer as a double nerve transfer. Other potential donors include the contralateral C7, the medial pectoral nerve and the thoracodorsal nerve, although the thoracodorsal nerve is harder to rehabilitate.

Restoring hand function

In a pan-plexus injury, restoring hand function is a challenging problem. With limited donors and with priority going to restoring elbow and shoulder function, there are few options for donors to the hand. Although use of the contralateral C7 with a long nerve graft to median nerve transfer has been described, the long distance from the motor endplates and the multiple coaptations results in suboptimal restoration of function.^{29,31} The authors' preference in that setting is to use a free functioning muscle transfer, most commonly gracilis, to restore extrinsic hand function. The first stage of this transfer can be performed at the time of the original plexus reconstruction with inset of a long graft from the intercostal donors. Once enough time has passed to allow sufficient regeneration, the second stage transfer of the muscle to restore finger flexion can be performed.

In lower plexus injuries, where patients present with isolated nerve palsies, more distal nerve transfer is accomplished using synergistic donors. For recovery of a median nerve palsy, establishing anterior interosseous nerve function is a priority and can be performed with a number of potential donors. Options

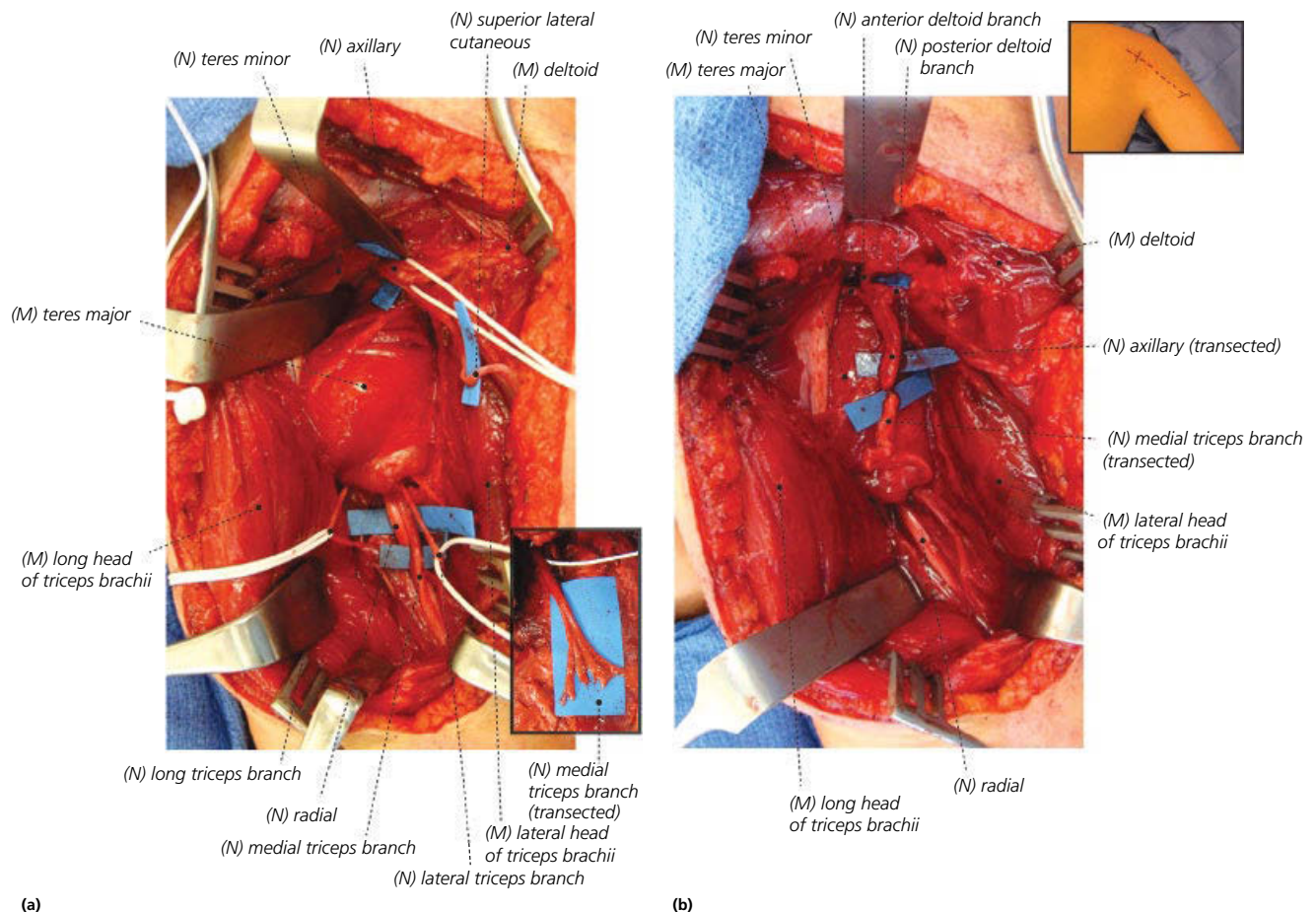


Figure 57.6 Medial triceps to axillary nerve transfer. (a) Intraoperative photo documenting the location of both the donor medial triceps branch and the recipient axillary nerve and (b) nerves prepared for coaptation under no tension. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

include the brachialis branch,³² and expendable branches of the radial nerve.³³ The internal topography of the median nerve is now well understood. In the proximal arm, the anterior interosseous nerve fascicles can be found located on the posterior medial aspect of the nerve. More distally, the fascicles wrap under the nerve and become the only branch to exit from the radial side of the median nerve, distal to the branches to flexor carpi radialis, palmaris longus and flexor digitorum superficialis. For exposure, the nerve can be isolated in the upper arm through the same brachial sulcus incision. More distally, the anterior interosseous nerve can be exposed through a curvilinear incision on the volar forearm.

Following ulnar nerve paralysis, recovery of intrinsic muscle function is a priority and is often accomplished by making use of the terminal branch of the anterior interosseous nerve as a donor to the deep motor branch of the ulnar nerve (Figure 57.7).³⁴ This terminal branch can be located on the anterior surface of the interosseous membrane as it penetrates into the pronator quadratus muscle. Harvesting it at its most terminal branching point will allow for enough length to transfer it to the side of the ulnar nerve, allowing this procedure to be used as a supercharging or babysitting option. The internal topography of the ulnar nerve at the level of the distal forearm is such that after the dorsal cutaneous ulnar branch exits the ulnar aspect approximately 9 cm proximal to the wrist crease, the more ulnar 40% of the nerve is motor and the more radial 60% is sensory. It is important to make the epineurotomy window at the motor aspect of the nerve to allow the anterior interosseous fascicles to reach the intrinsic muscles of the hand. If the anterior interosseous nerve is not available, other options include the extensor digiti quinti or extensor carpi ulnaris, both of which will require a nerve graft.

Radial nerve deficits can be addressed by borrowing redundant fascicles from the median nerve as donors. Priorities are to re-establish posterior interosseous and extensor carpi radialis brevis functions.³⁵

Restoring function after brachial plexus palsy

The approach to the brachial plexus in the obstetrical population is slightly different than in adults. Here there remains a significant role for exploration of the plexus and nerve grafting.³⁶ Neuroma excision and sural nerve grafting to obtain sufficient graft length are standard of care. The recent addition of motor nerve transfers, either in combination with nerve grafting or independently, has potentially augmented clinical recovery by providing a source of motor axons closer to the neuromuscular junction.³⁶

Restoring sensation

Significantly improved clinical recovery is achieved in the sensate hand. Similar to options for motor nerve transfers, the authors advocate for the use of sensory nerve transfers to restore sensation to critical areas such as the first webspace for improving pinch. Unlike the motor nerve transfers, these sensory transfers can be performed independent of time.³⁷

Postoperative care

The majority of brachial plexus reconstructions can be performed on an outpatient or brief hospital stay basis. Postoperative pain control is critical and surgeons should have a low threshold for starting patients on an aggressive postoperative pain regimen including targeting neuropathic pain.

The period of immobilization following surgery is largely dependent on operative procedure, but also factors in patient compliance and age. Given that primary repair or nerve grafting should only be performed in the setting of no tension, a brief period of immobilization less than one week is all that is required postoperatively. Similarly, nerve transfers require minimal immobilization so long as they are taken through a full range of motion for the joint above and below at the time of coaptation and there is no evidence of tension. Tendon transfers require a more prolonged period of immobilization, ranging from 3 to 6 weeks depending on the transfer. Early gentle passive range of motion is encouraged to prevent adhesion formation and joint stiffness. Where the plexus has been fully exposed by dividing the pectoralis major tendinous insertion, the patient should be kept in a sling for 4 weeks to avoid tension on the repair. Elbow, wrist and hand range of motion within the sling are encouraged. Hand therapy and physical therapy are critical for optimizing recovery and the role of the therapist cannot be overstated. In addition, cortical retraining, motor re-education, and strengthening will be part of the longer term rehabilitation.

Complications

As with any surgery, the complications associated with brachial plexus surgery can be divided into acute, short term and long term. The majority of these patients are young and otherwise healthy, making their anesthetic risks significantly less, however given that they often require multiple surgical procedures, the risk cannot be completely discounted. Other acute complications include injury to adjacent structures, bleeding and inability to complete the procedure. Locating a paralysed nerve can be humbling, given that the nerve does not function, cannot be stimulated and often the surrounding musculature is denervated and abnormal in appearance. Having an excellent knowledge of nerve anatomy is critical to these transfers and it is not unusual for the authors to bring an anatomy atlas into the operating theatre.

Short-term complications include infection, wound healing problems or dehiscence, seroma and haematoma. Long-term complications include scarring, joint stiffness, pain and ineffective or unsuccessful results from the surgery. For tendon transfers, additional complications would include tendon rupture or adhesion, although using a Pulvertaft weave should make rupture less probable.

Donor deficit, for either nerve or tendon transfer, is a consideration. For the majority of nerve transfers, a transient

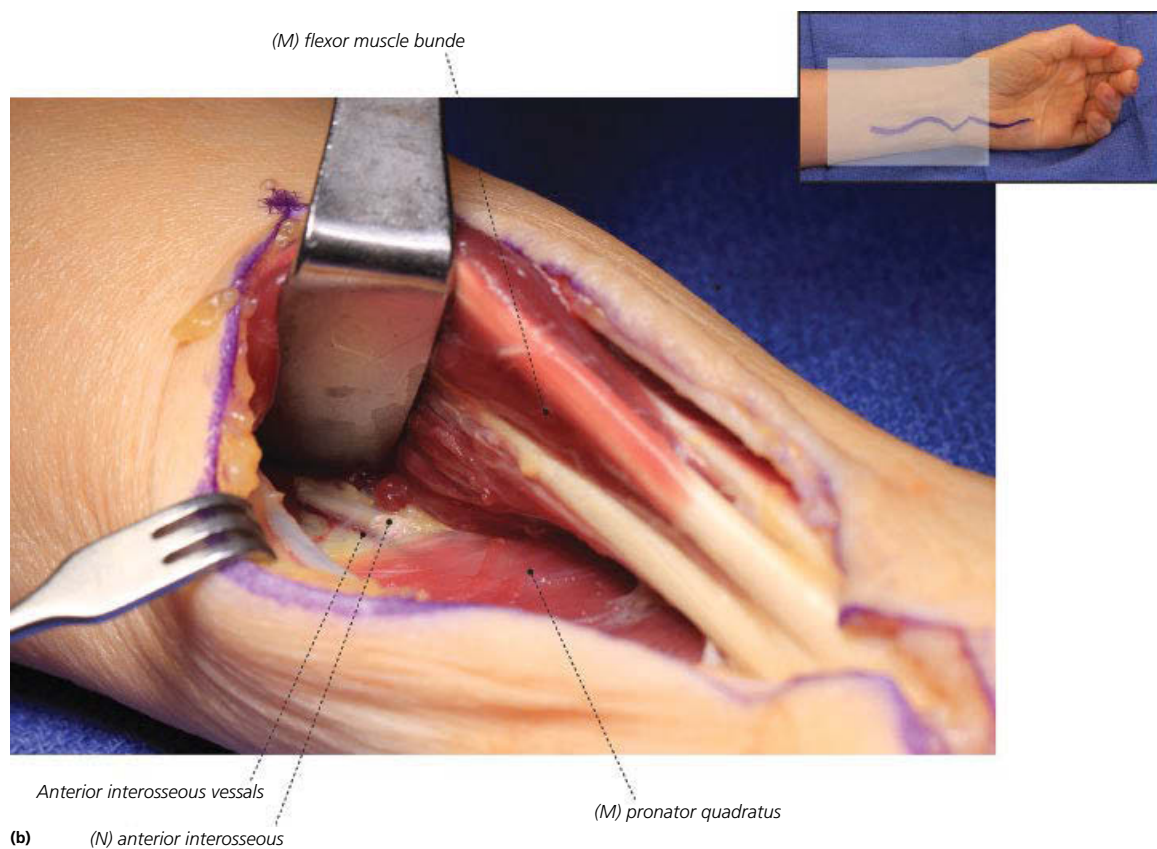
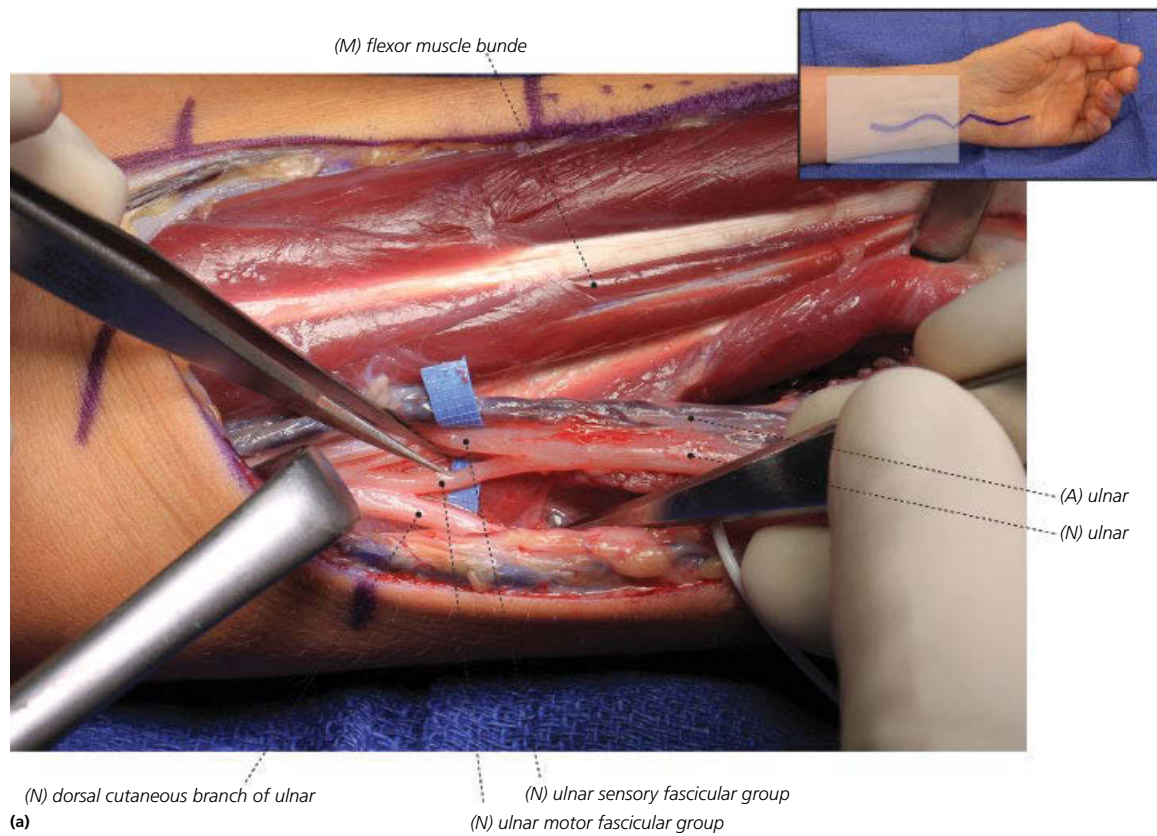


Figure 57.7 Anterior interosseous to deep motor branch ulnar nerve transfer. (a) Intraoperative photo illustrating the topography of the ulnar nerve at the level of the distal forearm and wrist with isolation of the motor fascicles. (b) Intraoperative photo of the anterior interosseous nerve lying on the interosseous membrane just deep to pronator quadratus. (c) Recipient ulnar nerve divided proximally (donor distal, recipient proximal). (d) Donor and recipient nerves prepared for coaptation under no tension. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

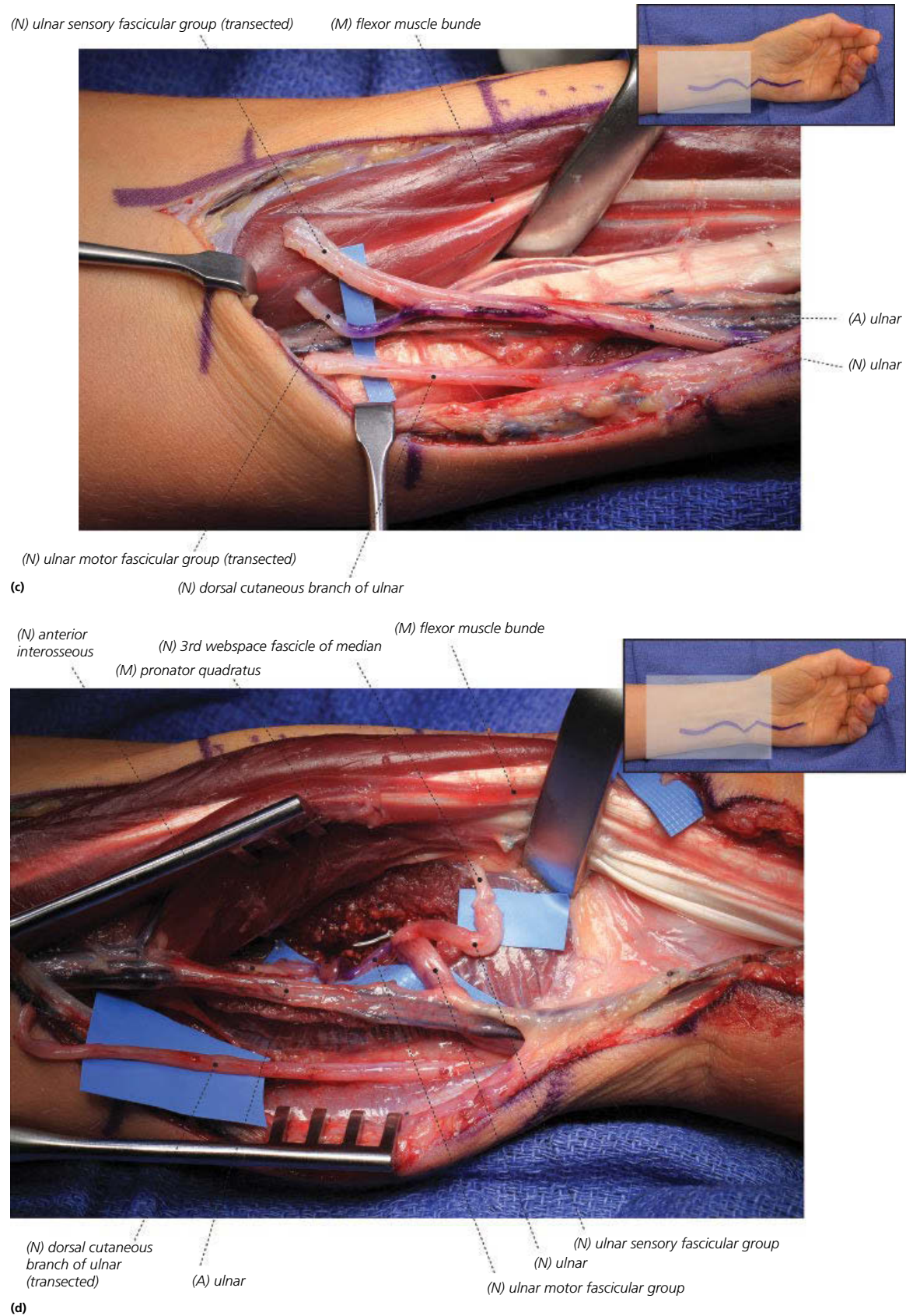


Figure 57.7 (Continued)

donor deficit is a possibility, but almost no long-term deficits are reported. Similarly, for tendon transfers, the selection of an expendable donor means that overall function should not be significantly affected.

Outcomes

Outcomes for brachial plexus injuries are related to the level and severity of the injury, the method and timing of reconstruction, and the patient's degree of compliance with postoperative rehabilitation. Due to the proximal level of these injuries, outcomes have been notoriously poor and prior to the advent of nerve transfers, when results were dependent on plexus exploration and grafting 'good' outcomes were only achieved in 20–33% of patients.³⁸ Many of these earlier studies, however, lack an objective outcome measure, and thus it is difficult to compare degree of recovery. A recent publication comparing almost 300 patients with upper trunk lesions found significantly better outcomes following dual nerve transfers than traditional nerve grafting in establishing shoulder function.²⁷

The Medical Research Council (MRC) grading system for strength, along with other objective measures such as grip strength, pinch strength and potentially maximum weight lifted, provides a more acceptable way to compare outcomes.

For recovery of elbow flexion, the results following nerve transfer are generally quite good. Using a double fascicular nerve transfer, 28 of 29 patients regained elbow flexion and of these, 23 regained MRC 4/5 strength or greater.³⁹ Liverneaux *et al.* reported MRC 4/5 recovery of elbow flexion in all patients using the same procedure.⁴⁰ There was no permanent donor deficit in either of these studies. In the obstetrical brachial plexus population, 67% of patients showed good or excellent recovery following nerve reconstruction for the elbow and the results were significantly better for those patients with a C5 to C7 palsy than those with a pan-plexus palsy.⁴¹

When comparing extraplexal donors, Terzis and Kostopoulos found better success when three or more intercostal nerves were harvested as compared to two.²¹ In addition, results were better when the lower intercostal nerves T7–T10 were used compared to the upper intercostals of T3–T6.²¹ They report good to excellent results in almost three quarters of patients undergoing intercostal to musculocutaneous nerve transfer.

Recovery of shoulder function is not as successful as elbow flexion, however is significantly better than results achieved with shoulder arthrodesis. Songcharoen reported on 577 patients undergoing spinal accessory to suprascapular and/or axillary nerve transfer and reported that 80% of patients regained at least MRC 3/5 motor recovery.²⁹ Reinnervation of both the suprascapular and axillary nerves results in improved results compared to transfer to a single nerve.⁴² Leechavengvongs described seven cases where a triceps branch was transferred to the axillary nerve and all patients regained MRC 4/5 deltoid function and 5 of 7 had an excellent result.⁴³

Recovery of hand function has been less successful. Gu reported on 32 patients undergoing contralateral C7 root transfer. Fourteen of the patients had median nerve recipients and half regained MRC 3/5 motor function and 12 regained MRC 3/5 sensory function.⁴⁴ Ray and Mackinnon report on 12 of 19 patients with good to excellent (MRC 4/5 or greater) recovery of finger and thumb extension following median to radial nerve transfers.³⁵ In the same population 18/19 had meaningful recovery of wrist extension.

Regardless of the clinical recovery, patient satisfaction and return to work are important determinants of the success of brachial plexus reconstruction. Of 32 patients with brachial plexus injuries, over three quarters reported at least moderate overall life satisfaction and no patients reported extreme dissatisfaction.³ Ten of the 32 patients reported that the injury greatly affected their overall quality of life. Approximately half returned to work following their injury and of these, 70% reported moderate to high satisfaction with their job. This compares to 85% of the general population.

In a more recent study, Kretschmer *et al.* revealed that 87% of patients undergoing brachial plexus reconstruction were satisfied with their results and 83% would undergo surgery again. This was interesting in light of the fact that 52% of patients who had been gainfully employed prior to injury returned to their former occupation, pain since the injury was expressed in 86% of patients, and 21% suffered from depression and anxiety.⁴⁵ The group did find that occupational retraining was successful for 70% of patients, suggesting that this is an effective option in the patient with a brachial plexus injury.

Summary

Brachial plexopathies are devastating and have a significant impact on quality of life and upper extremity function. Although traditionally surgical intervention has resulted in relatively poor recovery of function, the implementation of new surgical techniques such as nerve transfers, combined with significantly improved knowledge of internal nerve topography, has dramatically improved outcomes. Although there remains a role for surgical interventions such as nerve grafting and tendon transfer, especially in the obstetrical brachial plexus palsy population, nerve transfers are rapidly emerging as the standard of care for the treatment of these difficult patients. This chapter highlights the anatomy, aetiologies, investigations, patterns of injury and surgical options to manage these patients and discusses the outcomes in this emerging field.

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CHAPTER 58

Nerve compressions

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Anatomy

Median nerve

The median nerve is formed from the medial (motor contribution) and lateral (sensory contribution) cords of the brachial plexus, then crosses over the brachial artery and courses distally along its medial aspect. In the upper arm, the median nerve lies between the medial intermuscular septum and brachialis muscle and then runs distally through the antecubital fossa under the lacertus fibrosis. The median nerve subsequently passes through the pronator teres (PT) muscle, most commonly between its superficial and deep heads. Less commonly, the median nerve will pass deep to both heads of the PT or through the substance of the superficial head.¹

After emerging from PT distally, the median nerve passes under the fibrous edge of the flexor digitorum superficialis (FDS) muscle, travelling between the FDS and flexor digitorum profundus (FDP) muscles. The median nerve gives off four groups of branches in the proximal forearm: branches to PT (proximal, superficial); branches to flexor carpi radialis (FCR) and palmaris longus (PL) (deep, ulnar); branches to FDS (deep, ulnar, more distal); and, the anterior interosseous nerve (AIN) which arises from the radial aspect of the median nerve, approximately 3 cm distal to the intercondylar line, and innervates the FDP to the index and long fingers, the flexor pollicis longus (FPL) and the pronator quadratus.²

In the distal forearm, the median nerve becomes more superficial and courses between and just deep to the FCR and PL tendons. The palmar cutaneous branch (PCM) arises from the volar-radial aspect of the median nerve 5 cm proximal to the wrist flexion crease, and may course distally up to 6 mm ulnar to the thenar crease.^{3,4} The PCM usually crosses the wrist as a single branch and then arborizes in the palm superficial to the palmar fascia.⁵

At the wrist the median nerve enters the carpal tunnel along with nine tendons (FDS, FDP, FPL). The carpal tunnel is bordered dorsally by the carpal bones, ulnarly by the hamate and triquetrum, and radially by the scaphoid, trapezium and FCR

sheath. The volar roof of the carpal tunnel is formed by the transverse carpal ligament (TCL), with contributions from the antebrachial fascia proximally and the thenar/hypothenar aponeuroses distally.

At the distal aspect of the carpal tunnel, the median nerve divides into the proper digital nerves to the thumb and radial border of the index finger, and the common digital nerves to the second and third webspaces. The recurrent motor branch typically arises from the radial border of the median nerve just distal to the flexor retinaculum (extraligamentous course), turning back to innervate the abductor pollicis brevis (APB), opponens pollicis and superficial head of the flexor pollicis brevis. The recurrent branch may also take a subligamentous (31%) or transligamentous course (23%).⁶ Other anatomic variations described by Lanz include an accessory median nerve branch that can arise distal or proximal to the carpal tunnel, and a bifid or duplicated median nerve.⁶

The Riche–Cannieu anastomosis is an anomalous motor connection from the ulnar to median nerve in the palm that contributes on average 28% of the innervation to APB.⁷ When present, thenar function may be preserved in the setting of severe carpal tunnel syndrome.

Points of compression

There are four main compression syndromes associated with the median nerve: carpal tunnel syndrome (CTS), pronator syndrome, AIN syndrome and compression of the PCM.

Carpal tunnel syndrome

CTS is the most common compression neuropathy in the upper extremity, with an annual incidence of 0.5–5.1 per 1000 persons.⁸ The aetiology of CTS is not fully understood, however the final common pathway is compression of the median nerve within the carpal tunnel resulting in impaired microcirculation, subperineurial oedema and eventually fibrosis. Demyelination occurs, which is at first localized then more diffuse, followed by axonal degeneration. Any condition that reduces the volume of the carpal canal or increases the volume of its contents can initiate this process.

Several systemic factors have been proposed as risk factors for the development of CTS, including female gender, advanced age, diabetes, thyroid disease, pregnancy, rheumatoid arthritis, obesity and smoking.^{9,10} Positioning of the wrist in flexion or extension during sleep may present a common causal pathway for many of these risk factors.¹¹ There is also substantial evidence to support an association between CTS and occupational factors, including repetitive hand use and vibration.¹²

Specific anatomic causes for CTS are identified in a minority of cases. Most commonly, this involves a space-occupying lesion within the carpal tunnel but lumbrical incursion into the carpal tunnel can also compress the median nerve, especially when the lumbricals are hypertrophic.¹³

Pronator syndrome

Pronator syndrome is a mainly sensory neuropathy resulting from compression of the median nerve in the proximal forearm, and is much less common than CTS. Risk factors include female gender, advanced age, and repetitive pronation activities (which can result in a short, tight PT).¹⁴

There are several potential compression points along the median nerve that can contribute to pronator syndrome. The most proximal entrapment points are the supracondylar process, present in less than 5% of patients and located 3–6 cm above the medial epicondyle, and the ligament of Struthers, a fibrous band that may span between the medial epicondyle and the supracondylar process.^{15,16} When the supracondylar process and/or ligament of Struthers are present, the median nerve separates from the brachial vessels and passes posterior to these structures, where it can be compressed. More distally, the lacertus fibrosis, fibrous bands between the deep and superficial heads of the PT, a tendinous deep head of the PT and the fibrous edge of the FDS can also compress the median nerve.^{17,18} Other less common causes of compression have been described, including an anomalous head of the PT, a snapping brachialis muscle, an accessory FPL (Gantzer's muscle), an anomalous palmaris profundus and an anomalous FCR brevis.¹⁸

Anterior interosseous nerve syndrome

The AIN can be selectively compressed by the same anatomic structures described above for pronator syndrome. However, unlike pronator syndrome, AIN syndrome is a motor neuropathy and entails no loss of sensory function. The syndrome usually occurs spontaneously (where no inciting event can be identified), but in some cases can result from local trauma or a tumour. It is important to note that most cases of AIN palsy are not due to mechanical compression, but rather to neuritis (i.e. Parsonage–Turner syndrome) and will usually recover spontaneously without operative intervention.^{19,20}

Compression of the palmar cutaneous branch

The PCM pierces the antebrachial fascia 2 cm proximal to the wrist flexion crease and, at this location, travels through a tunnel 3 mm to 2 cm in length. The tunnel may pass through the thickened antebrachial fascia or TCL.²¹ Compression of the

PCM results in altered sensation over the thenar eminence and radial palm.

Ulnar nerve

The ulnar nerve is formed from the medial cord (C8–T1) of the brachial plexus, lying medial to the axillary artery at its origin. In the middle third of the arm, the ulnar nerve pierces the medial intermuscular septum and travels distally between this septum and the medial head of triceps. At the elbow, the ulnar nerve enters the cubital tunnel. This fibro-osseous tunnel is bordered superficially by a fascial band spanning the olecranon and medial epicondyle, and on its deep surface by the ulnar groove of the medial epicondyle proximally, as well as the joint capsule and medial collateral ligament distally.²²

The ulnar nerve then enters the forearm between the two heads of the flexor carpi ulnaris (FCU) muscle, coursing distally between FCU and FDP on the medial aspect of the ulnar artery. The most proximal motor branch innervates the FCU approximately 1.6 cm distal to the medial epicondyle.²³ Subsequent branches to the FCU and ulnar two FDP muscles originate from both the medial and lateral sides of the ulnar nerve.²³ The dorsal cutaneous branch (DCU) of the ulnar nerve, which gives sensation to the dorsal aspect of the ring and small fingers, originates 10 cm proximal to the wrist crease.

At the wrist, the ulnar nerve enters Guyon's canal along with the ulnar vessels. Gross and Gelberman divided this area into three zones according to the internal topography of the ulnar nerve.²⁴ In zone 1 (proximal), the ulnar nerve lies superficial to the TCL, deep to the antebrachial fascia and radial to the FCU tendon. Just distal to the pisiform, the motor and sensory fascicles of the ulnar nerve separate. In zone 2 (distal-motor), the motor branch courses radially around the hook of the hamate and deep to the leading edge of the flexor digiti minimi muscle to innervate the intrinsic muscles of the hand. At this level, the floor of Guyon's canal is formed by the opponens digiti minimi, the medial wall by the abductor digiti minimi, and the roof by the volar carpal ligament and palmaris brevis. In zone 3 (distal sensory), the sensory branch of the ulnar nerve travels superficial to the hypothenar musculature to innervate the volar aspects of the small finger and ulnar half of the ring finger. The ulnar artery and its tributaries remain radial and superficial to the ulnar nerve as it travels distally into the wrist and palm.

The Martin–Gruber anastomosis is an anomalous median-to-ulnar connection in the proximal forearm present in 10–25% of cases.²⁵ The 'anastomotic' nerve branch may arise from the median nerve proper or the AIN and, when present, can result in preserved ulnar function in the hand in the setting of severe proximal ulnar neuropathy.

Points of Compression

Cubital tunnel syndrome

Cubital tunnel syndrome (CuTS), or compression of the ulnar nerve at the elbow, is the second most common compression neuropathy in the upper extremity. Potential sites of entrapment include (Figure 58.1): the arcade of Struthers, a thick fascial

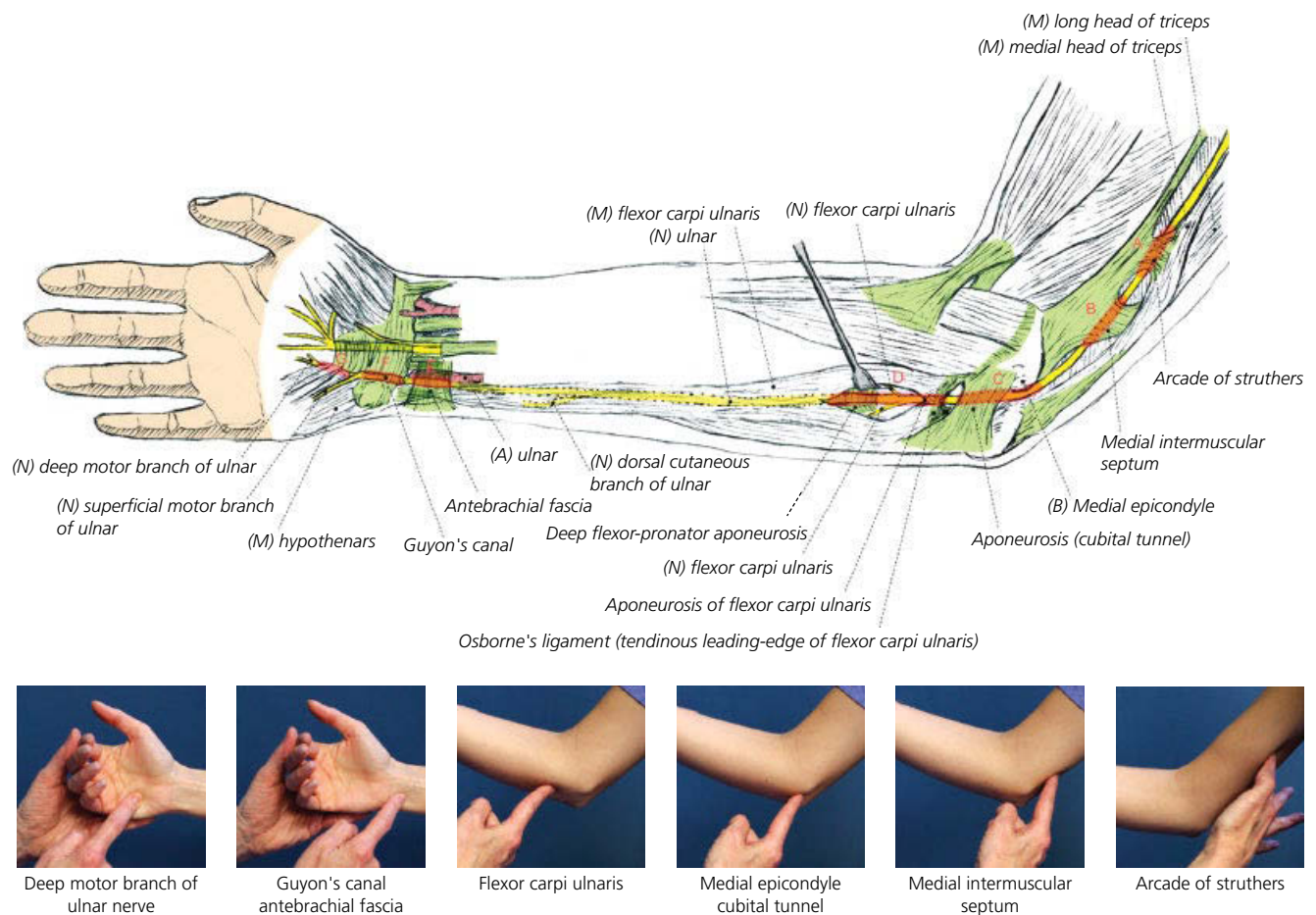


Figure 58.1 Potential sites of compression along the ulnar nerve at the elbow and wrist. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

connection between the medial head of the triceps and the medial intermuscular septum located approximately 8 cm proximal to the medial epicondyle; the bony retrocondylar groove, which may be altered by trauma, arthritis, heterotopic ossification, and soft tissue masses; and Osborne's band, a thickened confluence of fascia between the two heads of FCU.²⁶ More distally, the ulnar nerve may be compressed where it exits the FCU, by fibrovascular bands coursing over the nerve from the ulnar artery to the distal FCU, and by a fascial aponeurosis between the FCU and FDS to the ring finger (ligament of Spinner).^{27–29}

Congenital anatomic variations, such as cubitus valgus, an anconeus epitrochlearis muscle, a low-lying or prominent medial triceps muscle, and a hypoplastic roof of the cubital tunnel (allowing subluxation of the ulnar nerve) may also contribute to proximal ulnar compression.^{30,31} Other associated risk factors include female gender, advanced age, systemic neuropathy (i.e. diabetes), external compression (i.e. above-elbow cast), occupations involving repetitive or persistent elbow flexion and sleeping with the elbows in a flexed position. Several mechanisms have been proposed to explain why elbow flexion contributes to ulnar neuropathy, including dynamic compression, traction, ischaemia, extraneural scar and recurrent nerve subluxation.²⁰

Guyon's canal syndrome

Compression of the ulnar nerve at the wrist is the second most common site of ulnar nerve impingement, but is substantially less common than CuTS.³² A thickened antebrachial fascia can compress the ulnar nerve at the wrist (zone 1), whereas the tendinous leading edge of the hypothenar musculature can cause isolated compression of the deep motor branch (zone 2) (Figure 58.1). Additional aetiologies include compression by extra-anatomical structures (i.e. ganglion cysts, tumours, vascular malformations, anomalous musculature) and trauma (i.e. hypothenar hammer syndrome, hook of hamate fractures, distal radius fractures, carpal dislocations).^{20,32,33}

Radial nerve

The radial nerve is a terminal branch of the posterior cord of the brachial plexus (C5–T1). It lies dorsal to the axillary vessels at its origin then courses beneath the lateral head of the triceps, where it gives off the motor branches to the triceps, the posterior cutaneous nerve of the arm and the posterior cutaneous nerve of the forearm. At the mid-humeral level, the radial nerve travels within the spiral groove of the humerus, crossing from the posterior-medial to posterior-lateral aspect of the upper arm. It pierces the lateral intermuscular septum approximately 10 cm

proximal to the lateral humeral epicondyle and can be found between the brachialis muscle and the origin of the brachioradialis muscle just above the antecubital fossa.³⁴ Here, motor branches to the brachioradialis and extensor carpi radialis longus (ECRL) muscles arise. The radial nerve then bifurcates into the posterior interosseous nerve (PIN; motor component) and the superficial radial nerve (SRN; sensory component).

The PIN courses dorsolaterally over the radiohumeral joint, around the radial head through the substance of the supinator muscle and distally along the dorsal surface of the interosseous membrane. The PIN supplies the majority of the forearm and hand extensors, including the extensor carpi radialis brevis (ECRB), supinator, extensor digitorum communis (EDC), extensor digiti quinti, (EDQ), extensor carpi ulnaris (ECU), abductor pollicis longus (APL), extensor pollicis longus (EPL) and brevis (EPB), and extensor indicis proprius (EIP). Of note, the nerve branch to ECRB arises proximal to the entrance of the PIN into the supinator muscle. The terminal branch of the PIN travels at the base of the fourth extensor compartment to provide sensation to the dorsal wrist capsule and intercarpal ligaments.

The SRN travels distally on the undersurface of the brachioradialis muscle in the volar-radial aspect of the forearm. About 10 cm proximal the radial styloid, the SRN exits between the brachioradialis and ECRL tendons dorsally to become subcutaneous. The SRN provides sensory innervation to the dorsal-radial aspect of the hand.

Points of compression

Proximal radial nerve compression

Proximally, the radial nerve may be compressed by the lateral intermuscular septum or a fibrous arch formed by the lateral head of triceps.²⁰ More commonly, however, the proximal radial nerve is compressed by external forces, such as leaning on a chair resulting in 'Saturday night palsy' or a pneumatic tourniquet; these transient compression neuropathies tend to resolve spontaneously without surgical intervention.³⁵

Radial tunnel syndrome/PIN syndrome

Compression of the radial nerve and its branches in the proximal forearm is usually anatomic in nature, although other aetiologies such as fractures of the radial head have been documented.³⁶ The anatomic structures responsible for radial tunnel syndrome and PIN syndrome are similar, but patient symptomatology differs: radial tunnel syndrome is predominately sensory in nature, whereas PIN syndrome causes motor paralysis.

The radial tunnel is a region of the proximal forearm and elbow approximately 5 cm in length, starting distal to the motor branches to the brachioradialis and ECRL and ending distal to the supinator.²⁰ Here, the radial nerve is bordered medially by the brachialis muscle and biceps tendon, laterally by the brachioradialis muscle, and posteriorly by the lateral epicondyle.

Structures that may compress the radial nerve or PIN include fibrous adhesions at the radiocapitellar joint (i.e. from lateral

epicondylitis), the tendinous margin of the ECRB, the tendinous superficial head of the supinator muscle commonly known as the arcade of Frohse, the fascial border at the distal edge of the supinator and the vascular leash of Henry.^{37–40} Rare anatomic variations, such as fusion of the brachioradialis and brachialis muscles, may also predispose the radial nerve and PIN to compression.^{36,41}

Wartenberg's syndrome

Compression of the SRN is known as Wartenberg's syndrome.⁴² Anatomic compression of the SRN can occur by the brachioradialis tendon, which may be aggravated by activities requiring hyperpronation. However, the superficial location of the SRN distally and its complex branching pattern renders it highly susceptible to traumatic and iatrogenic injury (i.e. DeQuervain's tenosynovitis release, thumb carpometacarpal joint arthroplasty). Other less common aetiologies include compression by space-occupying lesions, haematomas, thrombosis of radial recurrent vessels, muscle anomalies and external compression from a watchband or bracelet.^{43,44}

Clinical history and assessment

A thorough history and physical examination is essential to identify all potential compression points and musculoskeletal disorders contributing to patient symptomatology. The history should explore the nature and time course of symptoms, and address all relevant comorbidities, medications and risk factors for nerve compression. Typical symptoms of compression neuropathy include numbness, paraesthesias, pain and/or weakness in the distribution of the affected nerve(s). A pain evaluation questionnaire is a useful adjunct, especially in patients with diffuse symptomatology.

The physical examination should consist of a complete sensory and motor evaluation of both upper extremities, to localize the site of compression and determine the severity of neuropathy. All potential entrapment sites of a nerve should be evaluated owing to the double crush mechanism, wherein compression of a nerve at one site renders it more susceptible to compression at a second site.⁴⁵ Failure to identify and address all involved compression points can lead to ongoing or residual symptoms following treatment.

Multiple techniques to evaluate sensation have been described, but there is no accepted gold standard. Sensory examination may include: light touch (i.e. the Ten Test⁴⁶); vibration and cutaneous pressure thresholds (i.e. Semmes-Weinstein monofilament testing); and, static and dynamic two-point discrimination. Abnormal sensory thresholds are an early finding in compression neuropathy, whereas abnormal two-point discrimination is a late finding.

Provocative tests using percussion (i.e. Tinel's test), direct pressure or joint movement can elicit patient symptoms by increasing compression on a nerve at known entrapment sites.

The recently described scratch collapse test (SCT) is also a useful tool to identify areas of compression.⁴⁷

Motor testing should include inspection of both upper extremities for atrophy and abnormal posture/positioning, as well as quantification of muscle strength using the Medical Research Council grading system, dynamometers (i.e. for pinch and grip), and special tests specific to each nerve. Motor weakness is usually a late finding in compression neuropathy.

Median nerve

Carpal tunnel syndrome

CTS classically presents with numbness, pain and paraesthesias in the volar aspect of the thumb, index, long and radial borders of the ring fingers. Since the PCM arises proximal to the carpal tunnel, sensation over the thenar area is preserved. Symptoms are often exacerbated by gripping and wrist flexion, and are usually worse at night. With severe or long-standing compression, thenar weakness and atrophy may occur.

Provocative tests for CTS include the SCT, Tinel's test, Phalen's test (wrist flexion to 90° for up to 60 s), the median nerve compression test, and the fist test for lumbrical incursion (gentle flexion of the fingers into the palm). The latter four tests are positive when paraesthesias occur in the median nerve distribution. In general, clinical tests for CTS have high specificity but moderate sensitivity.^{47,48}

Pronator syndrome

Unlike CTS, sensory abnormalities in pronator syndrome include the PCM distribution as well as the radial three and a half digits. Patients frequently describe pain in the volar forearm, exacerbated by pronation against resistance and direct pressure. Less commonly, patients with pronator syndrome will report weakness or difficulty with small object manipulation.

On examination, pain is reproduced by passive supination with digital pressure placed over the median nerve at the leading edge of PT. The PT may also be tender and occasionally a ligament of Struthers may be palpated.^{20,49} A positive Tinel's sign and SCT may be present, with the latter being performed by firmly pressing over the median nerve to elicit a response (owing to the deep location of the median nerve at this location). Additional provocative manoeuvres for pronator syndrome consist of resisted forearm pronation (PT), resisted elbow flexion with the forearm supinated (lacertus fibrosis) and resisted long finger proximal interphalangeal (PIP) joint flexion (FDS). Weakness may also be present in median nerve innervated muscles, but sensory symptoms predominate.¹⁴

AIN syndrome

The hallmark feature of AIN syndrome is atrophy and weakness of the AIN-innervated muscles. Unlike CTS and pronator syndrome, there are no sensory deficits. A history of severe pain for several weeks followed by weakness in the AIN distribution suggests neuritis rather than mechanical compression.²⁰

Ulnar nerve

Cubital tunnel syndrome

Cubital tunnel syndrome presents with sensory changes in the ulnar nerve distribution, as well as hand weakness that manifests as clumsiness, cramping, difficulty opening jars and difficulty manipulating small objects. Symptoms are worse at night and with activities requiring prolonged elbow flexion.

Sensory testing in CuTS will reveal diminished sensation in both the volar and dorsal aspects of the ring and small fingers. Provocative tests include Tinel's sign in the retrocondylar groove, the SCT and the elbow flexion test with or without simultaneous digital pressure over the ulnar nerve. Provocative manoeuvres should also evaluate for compression distal and proximal to the cubital tunnel.

Motor examination may demonstrate wasting and weakness of the interossei, hypothenars, FCU and FDP to the ring and small fingers. Specific findings related to ulnar intrinsic weakness include clawing of the ring and little fingers (Duchenne's sign), an abducted posture of the little finger (Wartenburg's sign), compensatory FPL flexion when attempting key pinch (Froment's sign), inability to cross the long and index fingers, reduced key pinch strength, and weakness of index and small finger abduction against resistance.⁵⁰

When evaluating a patient with CuTS, a systemic sensory neuropathy should be considered if sensory loss is much worse than motor loss. If motor deficits predominate, a C8–T1 cervical radiculopathy or a systemic motor neuropathy should be excluded. Combined thenar and ulnar intrinsic wasting is also indicative of cervical radiculopathy.

Guyon's canal syndrome

Patients may give a history of hand trauma or fullness in the hypothenar region, and often report worse hand function than patients with CuTS. Sensory testing will show loss of sensation on the volar aspect of the ring and small fingers but sensation on the dorsal-ulnar hand (DCU distribution) is spared. Signs and symptoms of ulnar-innervated intrinsic weakness may be present, but FCU and ring/small finger FDP function are intact. The examiner may also elicit a positive Tinel's sign, SCT and pressure provocation test at the wrist, and holding the wrist in extension may aggravate patient symptoms.

Radial nerve

Proximal radial nerve compression

Radial nerve compression in the upper arm will result in loss of sensation in the SRN distribution, as well as weakness of the brachioradialis, ECRL and PIN-innervated muscles. If there is sensory loss in the distribution of the posterior cutaneous nerves of the arm and forearm, and/or weakness in the triceps, the site of compression is even more proximal. A posterior cord lesion should be considered if there is axillary or thoracodorsal nerve involvement, and a C7 radiculopathy should be suspected if altered sensation is present in both the palmar and dorsal aspects of the middle finger and if there is concomitant

weakness in muscles innervated by the median nerve (i.e. PT, FCR, FDS, FPL).

Radial tunnel syndrome

Patients with radial tunnel syndrome typically describe aching pain in the proximal forearm that is worse with activity, and are tender to palpation over the radial tunnel. No distal sensory changes are present and motor dysfunction of the wrist and finger extensors is uncommon.

The main differential diagnosis of radial tunnel syndrome is lateral epicondylitis. In lateral epicondylitis, pain is maximal at the bony lateral epicondyle and is aggravated by resisted wrist extension. In radial tunnel syndrome, tenderness is present over the mobile extensor wad and is worsened by resisted middle finger extension.⁵¹ The SCT is also routinely positive in radial tunnel syndrome.

Provocative tests may help localize the site of radial nerve compression: compression due to fibrous bands at the radial neck will be aggravated by elbow flexion plus forearm supination with the wrist in neutral; compression by the ECRB will be aggravated by forearm pronation, elbow flexion to 45–90° with the wrist in full flexion; and, compression at the arcade of Frohse will be aggravated by isometric active supination from a full pronated position.²⁰

PIN syndrome

PIN syndrome presents as weakness of finger metacarpophalangeal joint extension (EDC function) and inability to lift the thumb off a table when resting flat (EPL function). Finger interphalangeal joint (IPJ) extension is preserved owing to the function of the intrinsics, and weak thumb IPJ extension may be present due to preserved function of the thenar muscles. Wrist extension is present but radially deviated and weak due to preserved ECRL function, but loss of the PIN-innervated ECU and ECRB. No sensory deficits are present, and the same provocative manoeuvres as for radial tunnel syndrome can help to distinguish the exact site of compression.

Wartenberg's syndrome

SRN compression presents as isolated sensory complaints in the dorsal-radial hand and wrist. A Tinel's sign, SCT and digital pressure test may be positive where the SRN exits dorsally between the BR and ECRL tendons. Motor examination is normal. Use of local anaesthesia can help differentiate symptoms related to the lateral antebrachial cutaneous nerve.

Investigations

Imaging

In general, imaging alone cannot establish the diagnosis of compression neuropathy, but may assist in identifying pathology contributing to the development of nerve compression at a specific location. Plain radiographs and/or CT are

useful for any compression neuropathy where trauma or bony abnormalities (i.e. a supracondylar process in pronator syndrome) are suspected as a causative factor. Ultrasonography and MRI can rule out cysts or other space-occupying lesions as a source of compression. Both ultrasound and MRI have also been used for diagnosis of CuTS with reasonable sensitivity and specificity: ultrasound evaluates the cross-sectional area of the ulnar nerve at the elbow, whereas MRI looks at ulnar nerve hyperintensity.⁵²

Electrophysiological studies

Electrodiagnostic studies are a useful adjunct for evaluating upper extremity nerve pathology. They can help to accurately localize a site of compression, assess the severity and progression of neuropathy, and exclude alternate diagnoses (i.e. cervical radiculopathy, myopathy, systemic polyneuropathy, disuse atrophy). However, they should be interpreted with an awareness of their limitations. Nerve conduction studies (NCS) evaluate large, myelinated fibres but not the smaller unmyelinated pain fibres. Since the latter are affected first in compression neuropathy, NCS may be normal early on.⁵³ Similarly, early ischaemia of a nerve resulting from compression cannot be detected on electrophysiologic studies. This accounts for the false-negative rates seen with electrophysiology.^{54,55} History and physical examination therefore remain a critical component to the diagnosis of compression neuropathy. Carpal tunnel syndrome, in particular, remains a clinical diagnosis.⁵⁶

As compression continues, demyelination and focal conduction block occur, resulting in increased latency and decreased conduction velocity on NCS, and fibrillation potentials on electromyography (EMG). Axonal loss often develops late in the pathophysiology of nerve compression, resulting in decreased action potential amplitude on NCS, and increased fibrillation potentials and reduced motor unit potential recruitment on EMG. In the very late stages of compression neuropathy, NCS may show absent sensory or motor action potentials and EMG will show marked fibrillations with no motor unit potentials.²⁰

Management principles and options

Carpal tunnel syndrome

The initial management of mild-to-moderate CTS should be non-operative. Common recommendations include activity modification, wrist splints, oral anti-inflammatory agents and/or corticosteroid injection. Nocturnal splinting with the wrist in neutral has been shown to significantly improve symptoms, and there is no advantage to wearing splints full time versus just at night.⁵⁷ Blocking the metacarpophalangeal joint in extension additionally helps to prevent the lumbricals from entering into the carpal tunnel. Corticosteroid injection into the carpal tunnel is effective at relieving CTS symptoms, although the results tend to be short-lived and is most useful for patients who require

temporary relief (i.e. pregnancy-related CTS).⁵⁸ Steroid injections may also be used for diagnostic and prognostic purposes. A Cochrane review of other non-operative modalities in CTS showed no benefit.⁵⁹

Surgery is indicated when conservative measures fail, and in severe CTS with motor involvement. Carpal tunnel release (CTR) can be performed via endoscopic or open techniques; the latter includes a spectrum of incision lengths from limited-access to full-length incisions. Multiple randomized controlled trials have been performed comparing open versus endoscopic CTR, none of which have demonstrated significant differences in outcomes or complications.⁶⁰ The potential advantages of endoscopic CTR include less scarring and pain in the immediate postoperative period, which may result in earlier return to work. However, there is a substantial learning curve to the endoscopic approach, during which time there is a higher risk of iatrogenic injury to the median nerve and a longer operative time. Limited incision open techniques also carry a higher risk of iatrogenic nerve injury owing to the reduced visualization afforded by the small incisions, and have no proven advantage over full open CTR.⁶⁰ Since even a small injury to the median nerve can be life-altering for a patient, the author's preferred approach remains open CTR with a full-length incision.

Author's preferred technique (Figure 58.2)

The procedure is performed under tourniquet control, with a Bier block or local anaesthesia plus sedation. A 2–3 cm incision is made in the palm, with the proximal extent abutting the wrist crease (except in obese patients, where the incision extends proximal to the wrist crease in a zigzag fashion to allow complete visualization). The incision is placed 5 mm ulnar to the interthenar depression to avoid injury to the PCM and to avoid dividing the TCL directly over the median nerve. The skin is incised and dissection is carried through the subcutaneous tissue to the palmar aponeurosis, which is divided longitudinally with a scalpel. Dissection then proceeds until the TCL is identified, which is divided sharply along its ulnar aspect. The distal extent of the release is the 'V' intersection between the hypothenar and thenar muscles. Proximally, any tight bands of antebrachial fascia are divided. The distal- and proximal-most division of the TCL is best performed with tenotomy scissors under direct visualization. Adequate release is confirmed by visual inspection of the proximal and distal extents of the carpal canal. Neurolysis of the median nerve, epineurotomy, and tenolysis should not be routinely performed in primary CTR.⁶¹ Following tourniquet release, haemostasis is achieved, and the skin closed with simple interrupted sutures. A well-padded wrist splint is applied for comfort and is removed in 2–3 days. Immediate range of motion (ROM) of the digits and light hand use are encouraged. The wrist is splinted at night for 2–3 weeks postoperatively to prevent bowstringing of the flexor tendons. Sutures are removed at 14 days and a gradual return to full activity is permitted 5–6 weeks after surgery.

Pronator syndrome

Non-operative management consists of stretching exercises for PT, activity modification, splinting and anti-inflammatory medications.

Surgery is indicated when conservative measures fail (Figure 58.3). A longitudinal S-shaped incision is made in the proximal third of the volar forearm, extending to the antecubital fossa proximally. Dissection is then carried down to the lacertus fibrosis, which is divided sharply. The tendon of the superficial head of PT is identified just radial to the radial artery, and is lengthened in a step-cut fashion. The superficial head of PT is then retracted ulnarly and the median nerve is identified proximally, medial to the brachial vessels. The deep head of PT is then identified, and its tendinous portion resected. The tendinous leading edge of FDS is divided. Any crossing vessels compressing the median nerve should also be ligated and divided. Proximally, the surgeon should be able to easily run a finger along the median nerve. If any compression is felt or if a ligament of Struthers or supracondylar process is identified preoperatively, the incision should be extended above the antecubital fossa to allow resection of these structures. Following haemostasis and a layered closure, a sterile dressing and a well-padded splint keeping the elbow in slight flexion are applied. The splint is removed in 3 days, and the patient is instructed to perform full ROM exercises.

Cubital tunnel syndrome

A 2- to 4-month trial of non-operative management is recommended for patients with mild to moderate symptoms. Patient education, consistent nocturnal splinting using a soft elbow pad to maintain the elbow in extension, and modifications to reduce elbow flexion during daytime activities can effectively reduce symptomatology.⁶² Cortisone injections are not recommended due to risk of intraneural injection or subcutaneous placement.

Operative management is indicated when conservative measures fail and when advanced motor dysfunction is present (nerve conduction velocities below 40 m/s, fibrillations, abnormal motor unit potentials).²⁰ Multiple surgical techniques have been described for the treatment of CuTS and there is continued controversy as to which technique is best.⁶³ Surgical options include simple decompression, medial epicondylectomy and anterior transposition in a subcutaneous, transmuscular, intramuscular or submuscular position.

In-situ decompression of the ulnar nerve entails releasing all relevant entrapment sites through a 6–10 cm incision posterior to the medial epicondyle, without disturbing the ulnar nerve from its bed and without performing neurolysis. Endoscopic and limited incision techniques have also been described, although the risk of haematoma, nerve injury and inadequate release of nerve compression is increased.⁶⁴ Potential advantages of in-situ decompression over transposition include reduced technical complexity, lower complication rates, and lower risk of nerve devascularization,⁶⁵ yet it does not address tension on the nerve during elbow flexion. A recurrence rate of 7% has been recently reported.⁶⁶

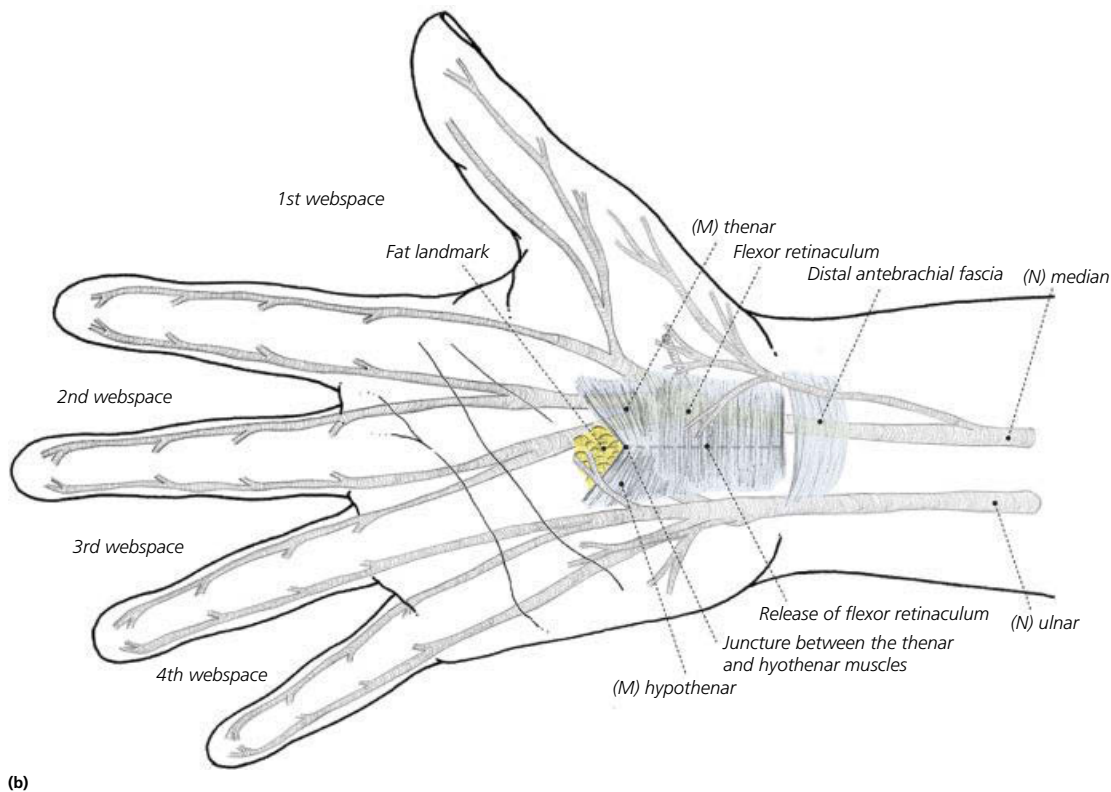
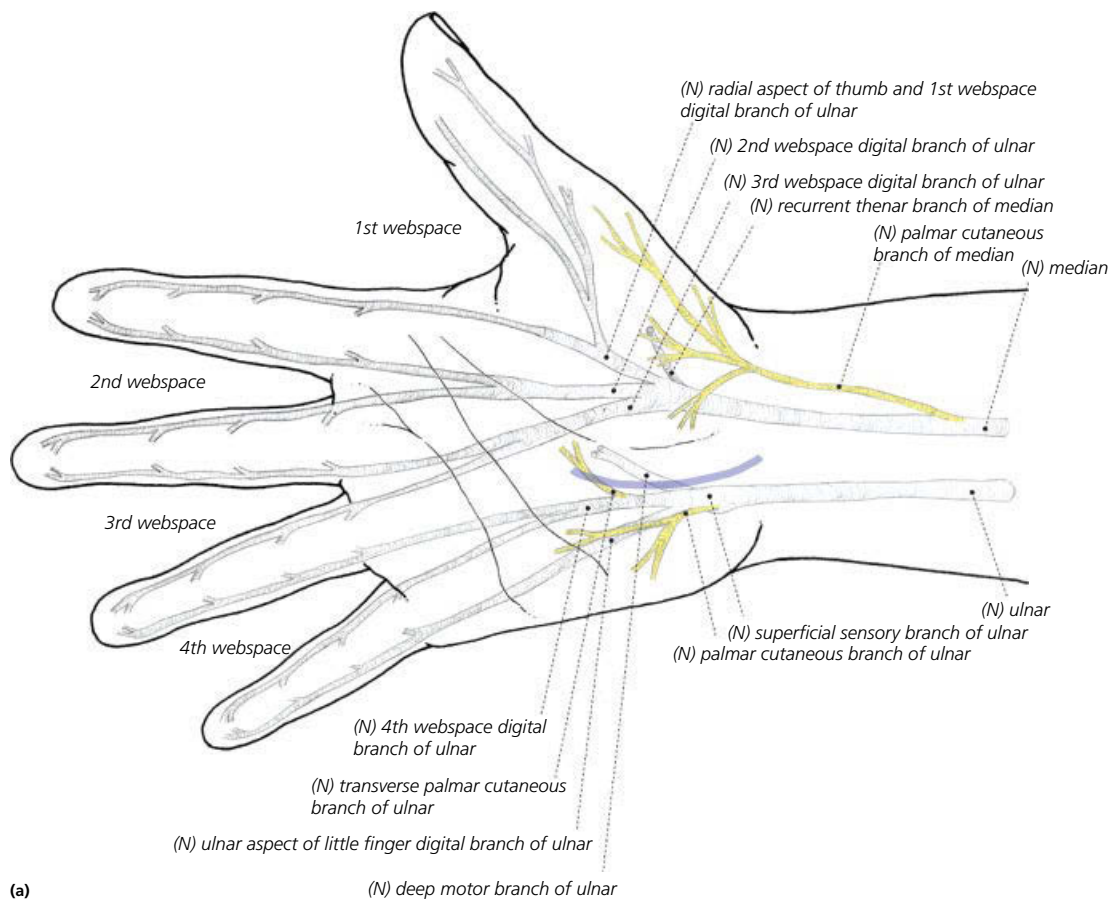
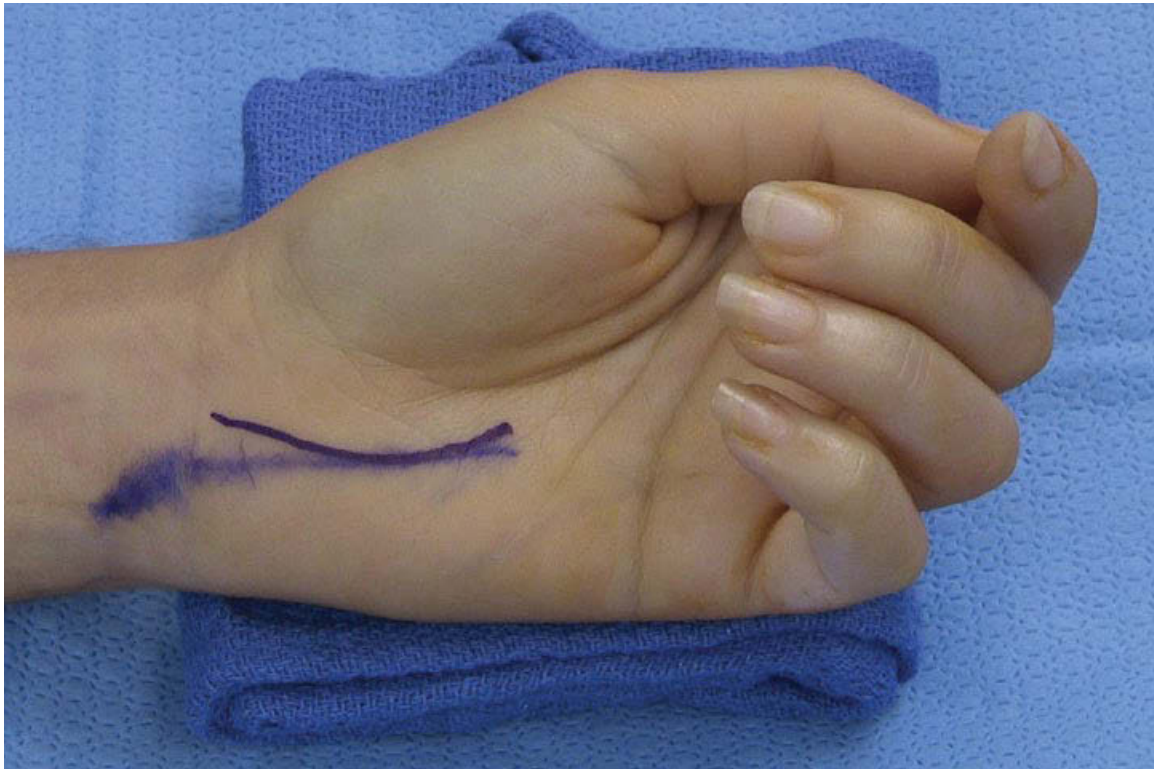
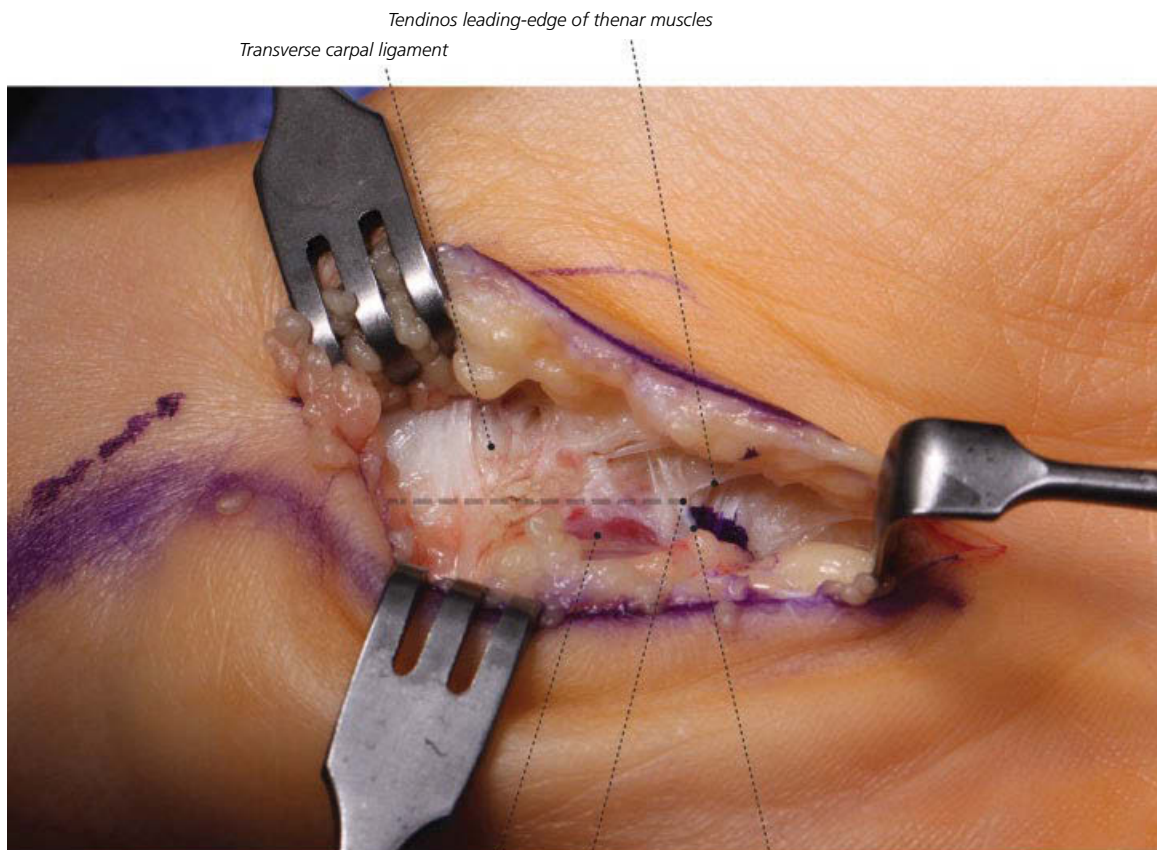


Figure 58.2 Author's preferred technique for open carpal tunnel release. (a) Relationship of the skin incision to the underlying median and ulnar nerve anatomy. (b) Landmarks for complete release of the transverse carpal ligament (TCL) (flexor retinaculum). The distal extent of the TCL is the 'V' intersection of the hypothenar and thenar musculature, distal to which is the mid-palmar fat pad. The proximal extent of the TCL is the antebrachial fascia. (c) Design of the skin incision approximately 5 mm ulnar to the interthenar depression. (d) Longitudinal division of the palmar aponeurosis. The TCL lies immediately dorsal (posterior) to this aponeurosis. (e) Following transection of the TCL, the median nerve and tendons within the carpal canal are visualized. (f) With good retraction, the proximal extent of the TCL should be clearly visualized to ensure an adequate, complete release. The surgeon should be positioned at the end of the hand table for the most ideal view. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

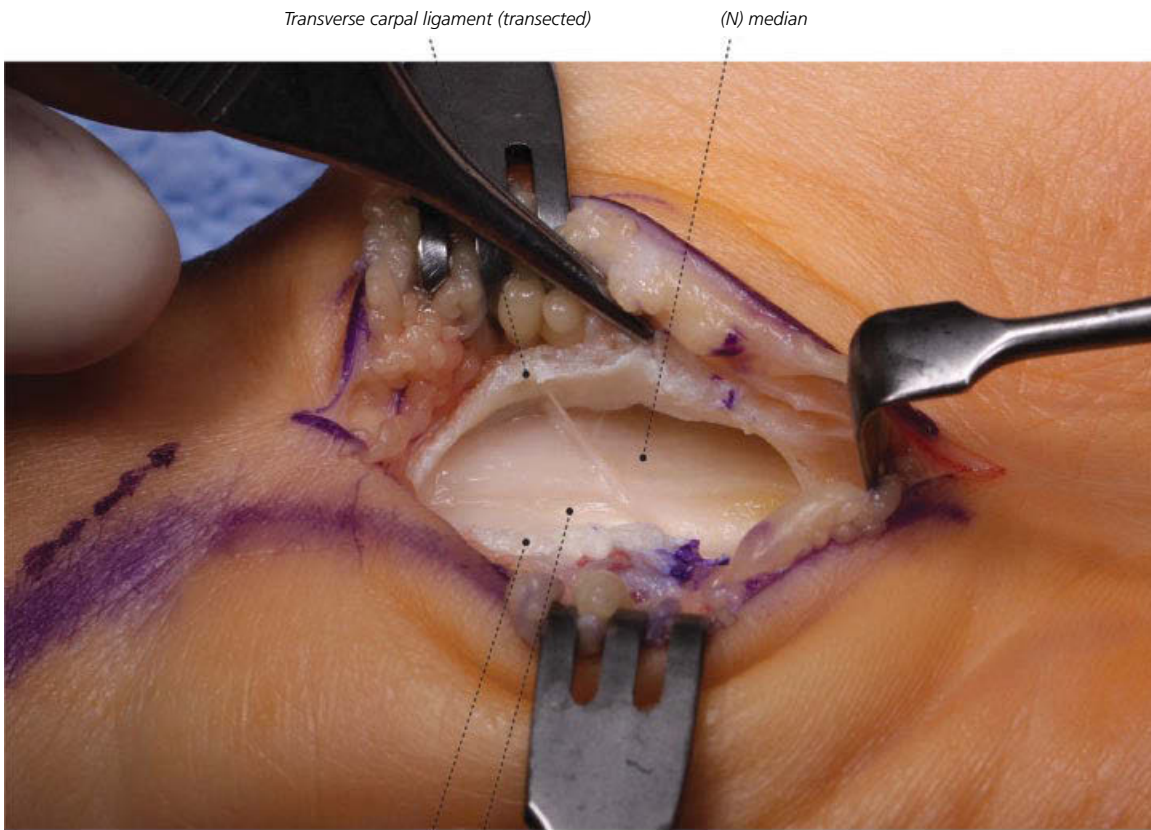


(c)

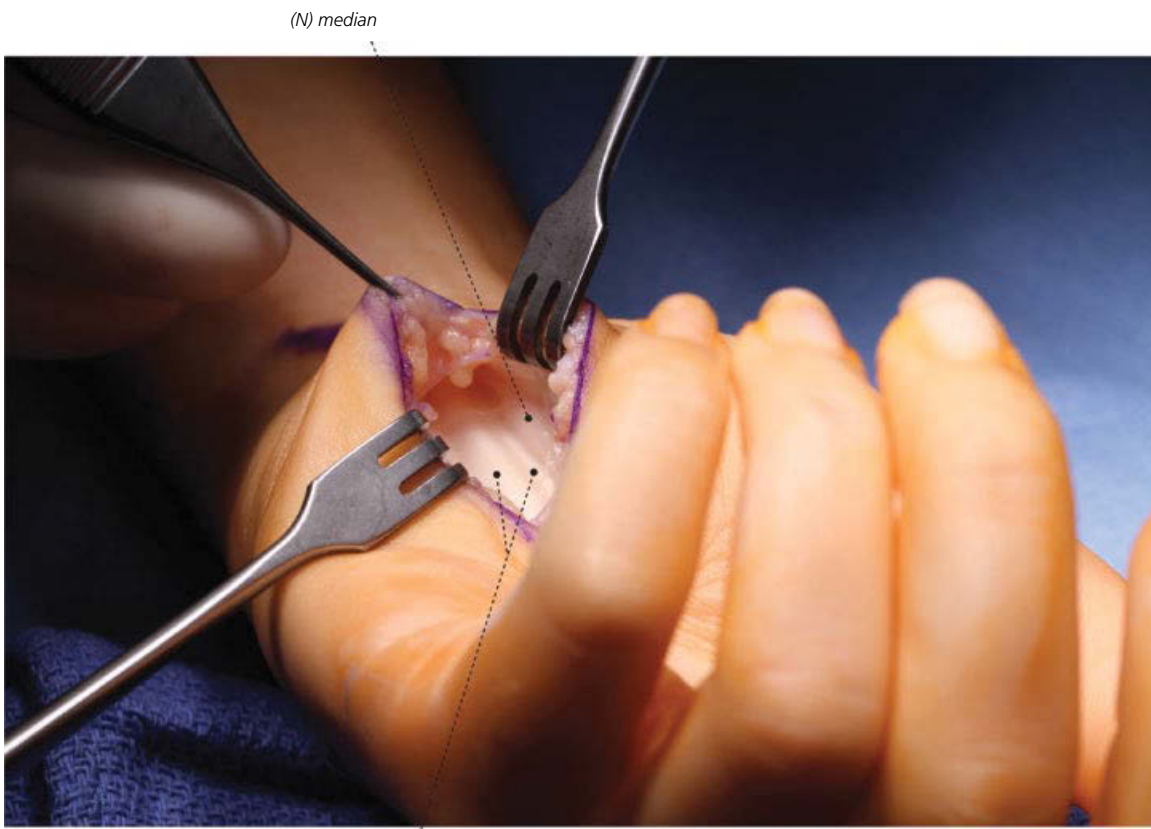


(d)

Figure 58.2 (Continued)



(e) Transverse carpal ligament (transected)
(M) tendons of flexor digitorum superficialis



(f) (M) tendons of flexor digitorum superficialis

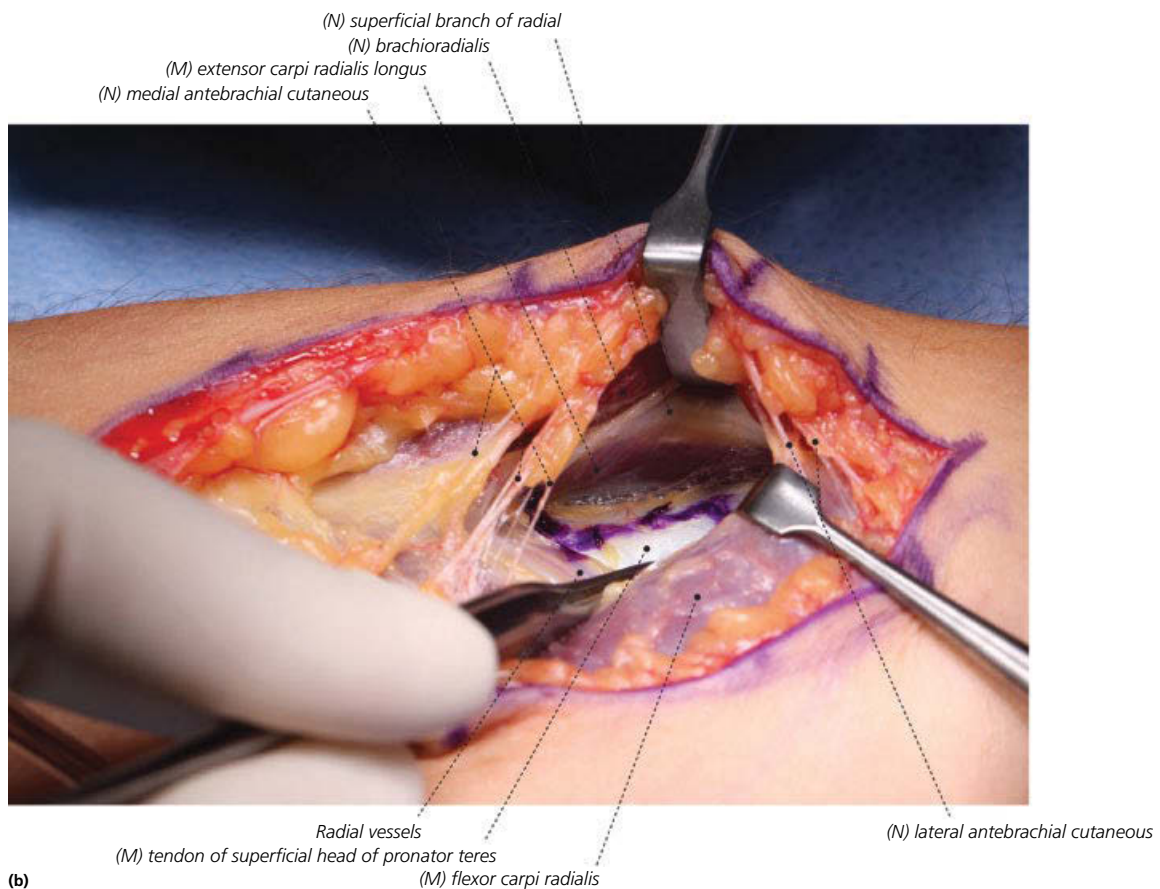
Figure 58.2 (Continued)

Medial epicondylectomy or ulnar nerve transposition should be considered if intraoperative subluxation of the ulnar nerve is seen following decompression. Complications of medial epicondylectomy include pain, weakness, and instability of the elbow joint.⁶⁵

Proponents of transposing the ulnar nerve anterior to the medial epicondyle cite several benefits, including reduced tension and compression forces on the ulnar nerve during elbow flexion, definitive treatment of nerve subluxation, and provision of a well-vascularized bed for the nerve.²⁰ In a subcutaneous



(a)



(b)

Figure 58.3 Author's preferred technique for decompression of the median nerve in the forearm. (a) Design of the skin incision in the proximal volar forearm. A carpal tunnel release (CTR) is simultaneously performed if there is evidence of distal median nerve compression on clinical evaluation. (b) The pronator teres (PT) tendon is identified between the radial vessels and radial sensory nerve in the mid-forearm. (c) Step-lengthening of the PT tendon. (d) With retraction of the superficial head of PT, the median nerve can be identified on the ulnar side of the radial vessels. The tendinous portion of the deep head of PT can be seen compressing the median nerve. (e) Resection of the tendinous portion of the deep head of PT. (f) Division of the tendinous leading edge of flexor digitorum superficialis (FDS) and any crossing vessels completes the decompression of the proximal median nerve. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

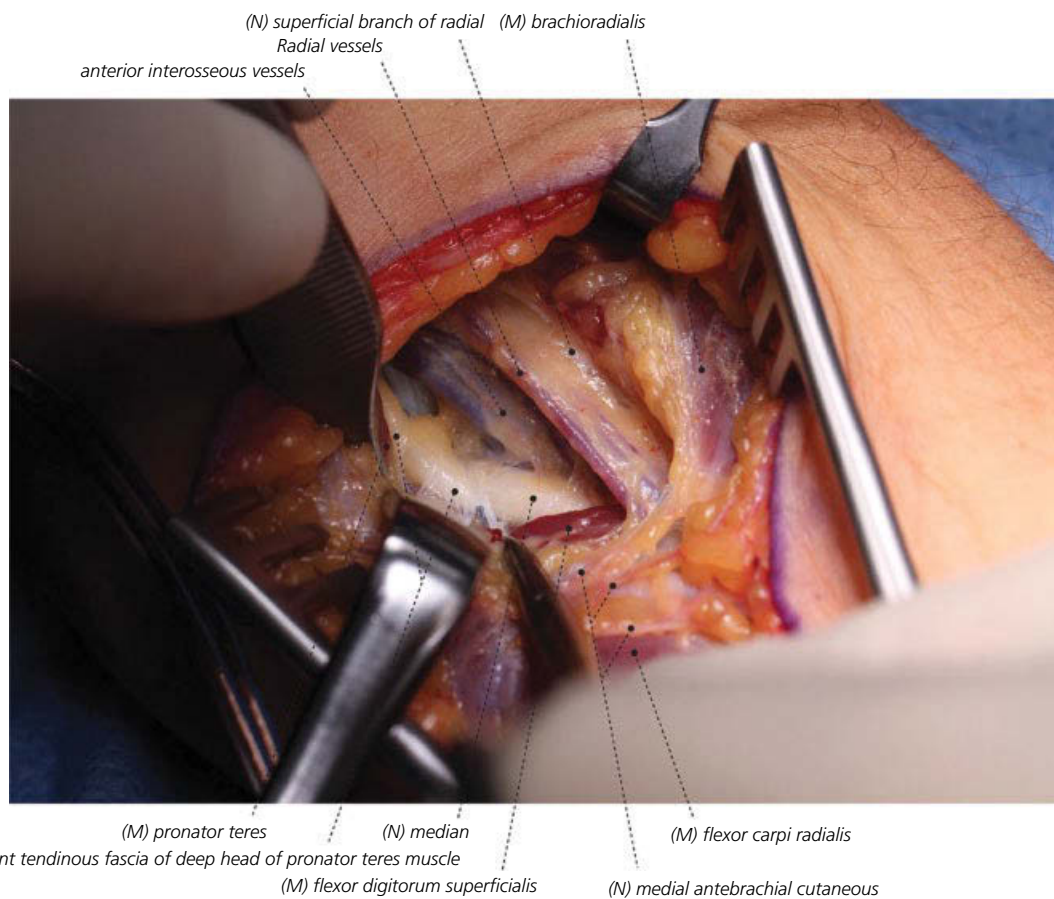
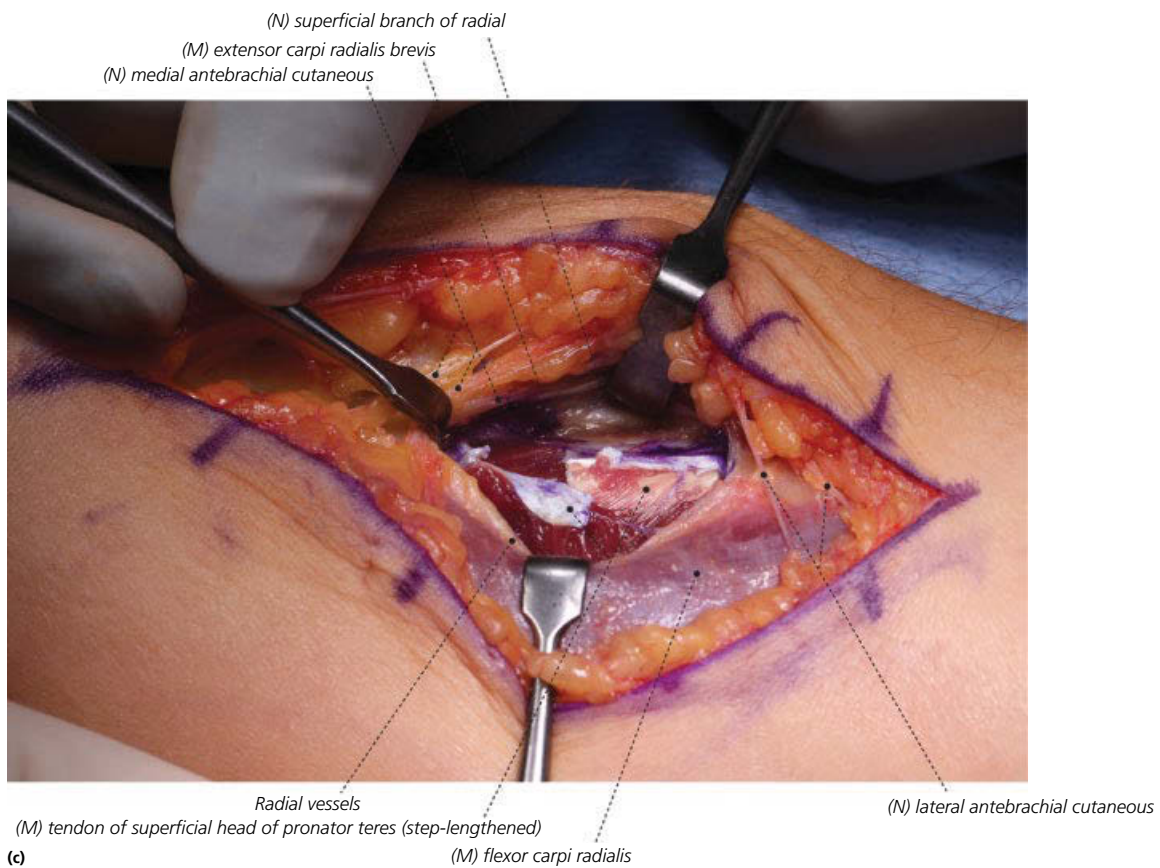
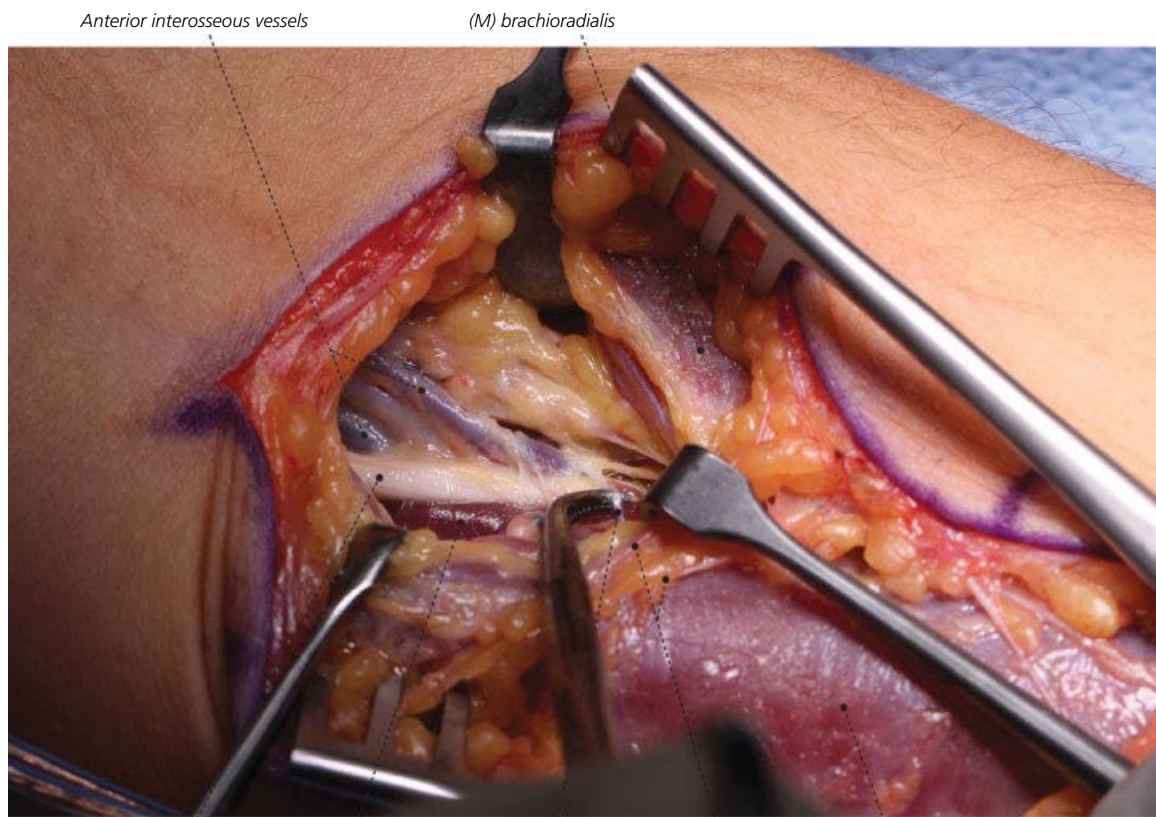
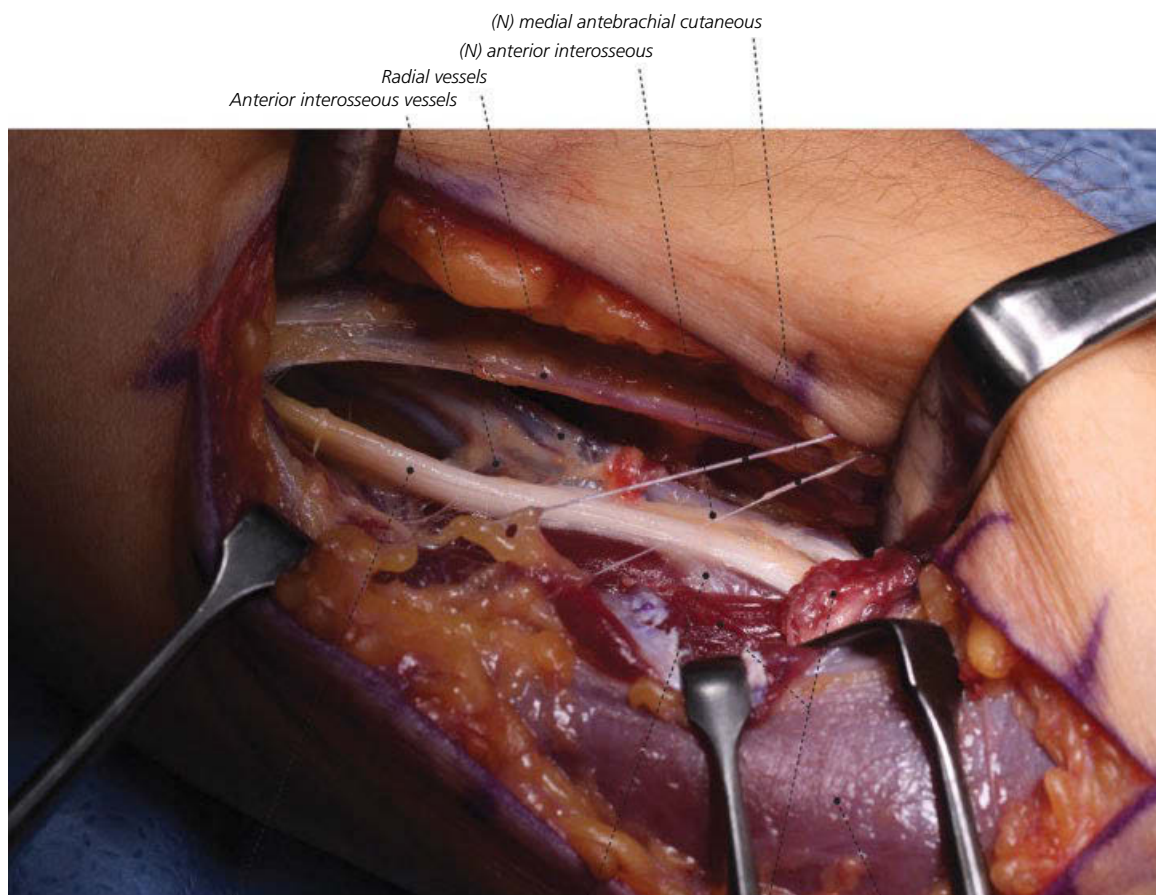


Figure 58.3 (Continued)



(e) Anterior interosseous vessels (M) brachioradialis (N) median (M) pronator teres (N) medial antebrachial cutaneous (M) flexor carpi radialis
 Remnant tendinous fascia of deep head of pronator teres muscle



(f) (N) medial antebrachial cutaneous (N) anterior interosseous Radial vessels Anterior interosseous vessels (N) median (N) flexor digitorum superficialis (M) flexor digitorum superficialis (M) flexor carpi radialis

Figure 58.3 (Continued)

transposition, the new anterior position of the ulnar nerve can be maintained by suturing the dermis to a fascial flap elevated off the flexor/pronator mass.⁶⁷ The subcutaneous technique involves less muscle dissection, but extensive mobilization of the ulnar nerve is required to avoid proximal or distal kinking.

To perform an intramuscular transposition, a groove is dissected in the flexor/pronator mass in line with the transposed position of the ulnar nerve, and a soft tissue or fascial sling is then created to maintain the nerve in its anterior position.⁶⁸ Relative to subcutaneous transposition, the intramuscular technique offers greater protection and places the ulnar nerve in a straighter axis. It also necessitates less dissection than submuscular transposition, but carries a risk of iatrogenic nerve compression.

Transposing the ulnar nerve under the flexor/pronator mass is advocated as the 'definitive' procedure for CuTS, especially in revision cases.^{69,70} The flexor/pronator mass is directly reattached to the medial epicondyle, with or without step-lengthening to minimize compression on the transposed nerve. Disadvantages of the submuscular technique include a longer incision, more extensive muscle dissection, heterotopic bone formation, and the potential for iatrogenic nerve compression.^{20,65}

Author's preferred technique (Figure 58.4)

Anterior transposition of the ulnar nerve in a transmuscular plane minimizes nerve tension during elbow motion, and requires less muscle dissection than submuscular techniques. The procedure is performed under regional or general anaesthesia using a sterile tourniquet. A 6–10 cm incision is made posterior to, and centred at, the medial epicondyle. During subcutaneous dissection, care is taken to identify and protect the medial antebrachial cutaneous nerve (MABC). The ulnar nerve is identified proximally below the medial intermuscular septum, which is then excised. The nerve is followed distally as it enters the retrocondylar groove, where the fascial roof of the cubital tunnel and Osborne's band are released. Distal to the elbow, another fascial septum exists between the ulnar-innervated FCU and the median-innervated flexor/pronator muscles; this is removed to avoid kinking of the nerve after transposition. Motor branches to the FCU and FDP are identified and preserved.

A step-cut incision in the flexor/pronator origin is made. The proximally based flap elevates easily with some muscle attached, whereas the distally based flap must be elevated with sharp dissection and close to the medial epicondyle to preserve innervation. Enough muscle should be released distally so that it does not kink the ulnar nerve when the nerve is transposed anteriorly. Intermuscular fascia must also be excised to avoid compression.

The ulnar nerve is then transposed anterior the medial epicondyle. Motor branches to the FCU or FDP that inhibit tension-free transposition should be neurolysed proximally. The entire course of the nerve is re-evaluated to ensure it lies in a straight line with no further proximal or distal sites of

compression. The fascial flaps are then closed loosely over the nerve with two non-absorbable sutures, leaving enough space to pass at least one finger.

The tourniquet is then removed, and meticulous haemostasis achieved. The ulnar nerve is palpated proximally to assess for an arcade of Struthers, which is released if present. The MABC branches are then inspected. If inadvertent avulsion or injury has occurred, the distal end of the injured branch should be crushed, cauterized and transposed proximally away from the incision. A layered closure is performed over a Jackson–Pratt drain. The arm is immobilized for 2–3 days with a well-padded splint to maintain the wrist in neutral, forearm pronated, and elbow flexed at 45–60°. ROM exercises are then initiated. Progressive strengthening starts at one month postoperatively, with return to full activities at two months.

Guyon's canal syndrome

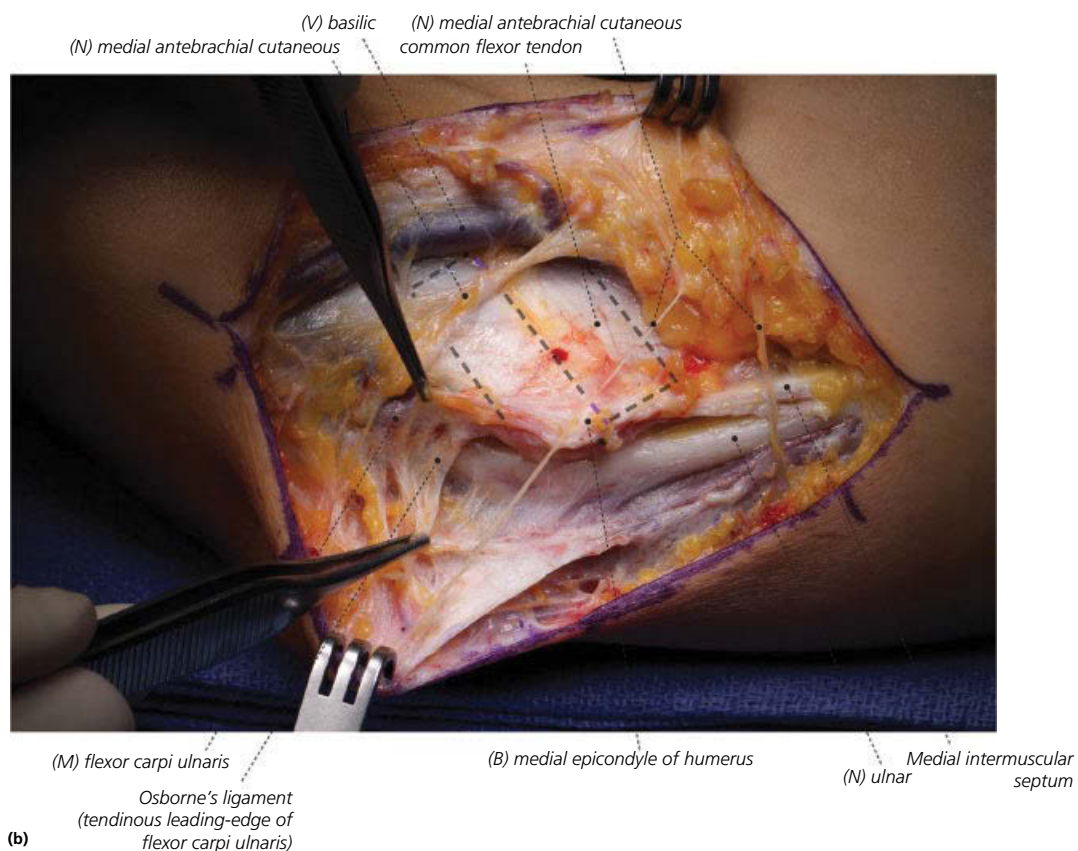
Management of ulnar nerve compression at the wrist is primarily surgical. The technique for surgical decompression of Guyon's canal entails six steps (Figure 58.5), and is performed under tourniquet control in the main operating room.²⁰ Step 1: a Taleisnik incision is made approximately 1 cm ulnar to the thenar crease, crossing the wrist in a zigzag fashion; dissection is carried through the subcutaneous tissue and palmaris brevis muscle, while preserving the cutaneous branch that is often present in the distal third of the palmar incision. Step 2: the ulnar neurovascular bundle is identified, exposed and retracted medially. Step 3: the tendinous leading edge of the hypothenar musculature is identified by palpating the hook of the hamate and looking just medial to it; the deep motor branch lies just deep to this tendinous leading edge. Step 4: the tendinous leading edge is carefully released with tenotomy scissors; often the deep motor branch is not visible until the initial 2–3 mm of the leading edge is divided. Step 5: decompression of the deep motor branch is completed by releasing the tendinous leading edge around the hook of the hamate to the small finger flexor tendons. Step 6: in order to adequately visualize and release the antebrachial fascia, the surgeon must incise and dissect proximal to the wrist crease. The ulnar nerve is then inspected proximally and distally to ensure there are no further sites of compression. The incision is closed with interrupted sutures, and a well-padded wrist splint is applied. Immediate ROM of the fingers are encouraged. The splint is removed in 2–3 days, and a gradual return to full activity is permitted over 6–8 weeks.

Radial tunnel/PIN syndrome

Conservative measures should always be attempted first, especially in radial tunnel syndrome. We have a lower threshold to operate in PIN syndrome owing to the motor deficits involved. Non-operative approaches include splinting and activity modification to help limit elbow extension, forearm pronation and wrist flexion. Anti-inflammatories and corticosteroid injections have also been utilized, especially if concomitant lateral epicondylitis is thought to be contributing to patient symptoms.²⁰



(a) Expected location of medial antebrachial cutaneous nerve Incision



(b) (M) flexor carpi ulnaris Osborne's ligament (tendinous leading-edge of flexor carpi ulnaris)

Figure 58.4 Author's preferred technique for decompression and anterior transposition of the ulnar nerve at the elbow. (a) Design of the skin incision just posterior to, and centred at, the medial epicondyle. (b) The medial antebrachial cutaneous nerve (MABC) should be preserved during subcutaneous dissection. The ulnar nerve is identified posterior to medial intermuscular septum above the elbow and is decompressed through the cubital tunnel. Design of the step-cut incision in the fascia of flexor/pronator mass. (c) Elevation of the fascial flaps at the flexor/pronator origin allows visualization of the intermuscular fascia that should be excised prior to transposition of the ulnar nerve. (d) Proximally, the medial intermuscular septum is exposed and resected prior to nerve transposition. (e) The ulnar nerve is followed as distally as possible to ensure it is completely decompressed, so that it will not be kinked when it is transposed anteriorly. (f) Anterior transposition of the ulnar nerve. The fascial flaps are very loosely closed over the ulnar nerve to maintain its new position. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

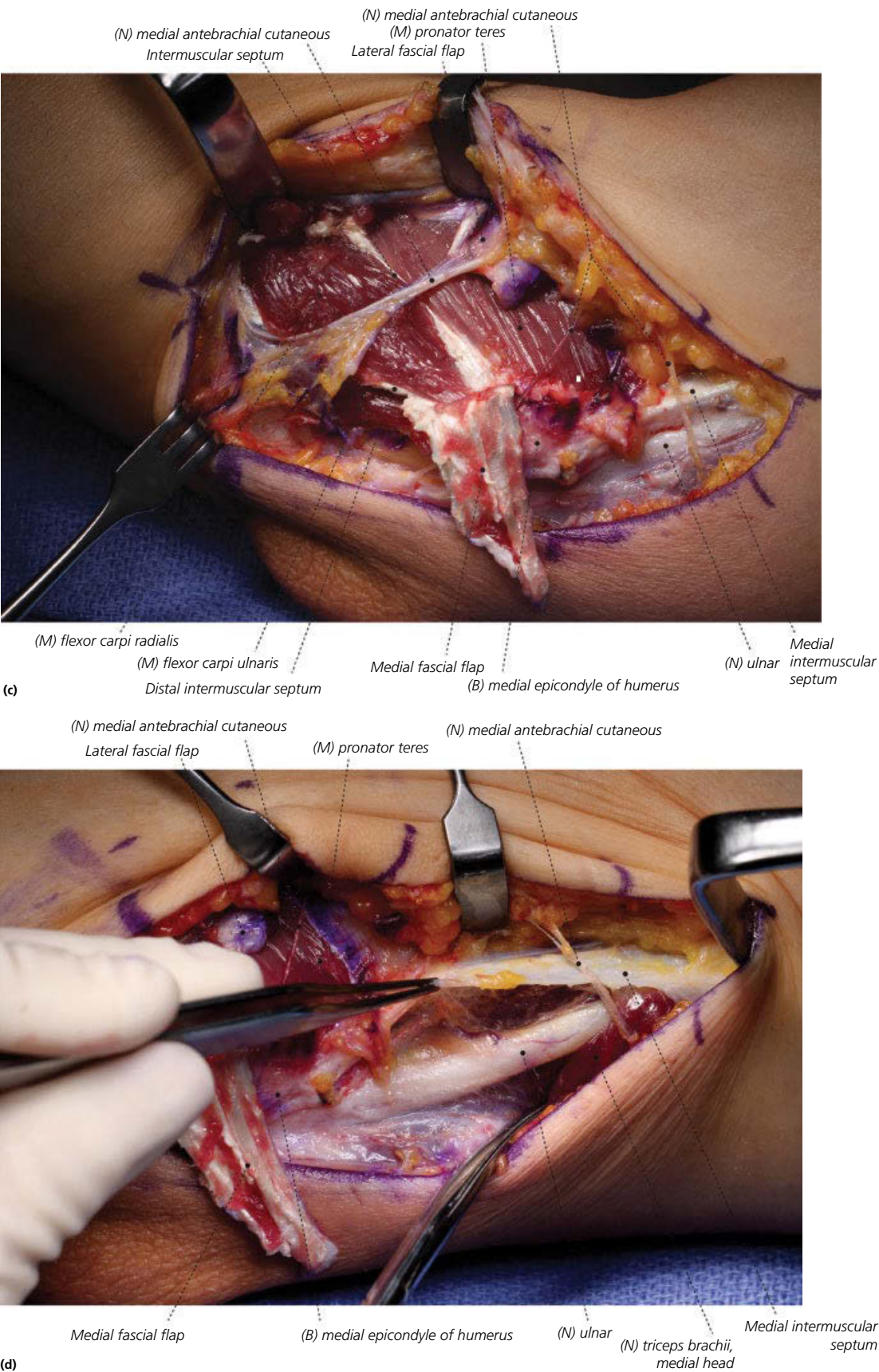
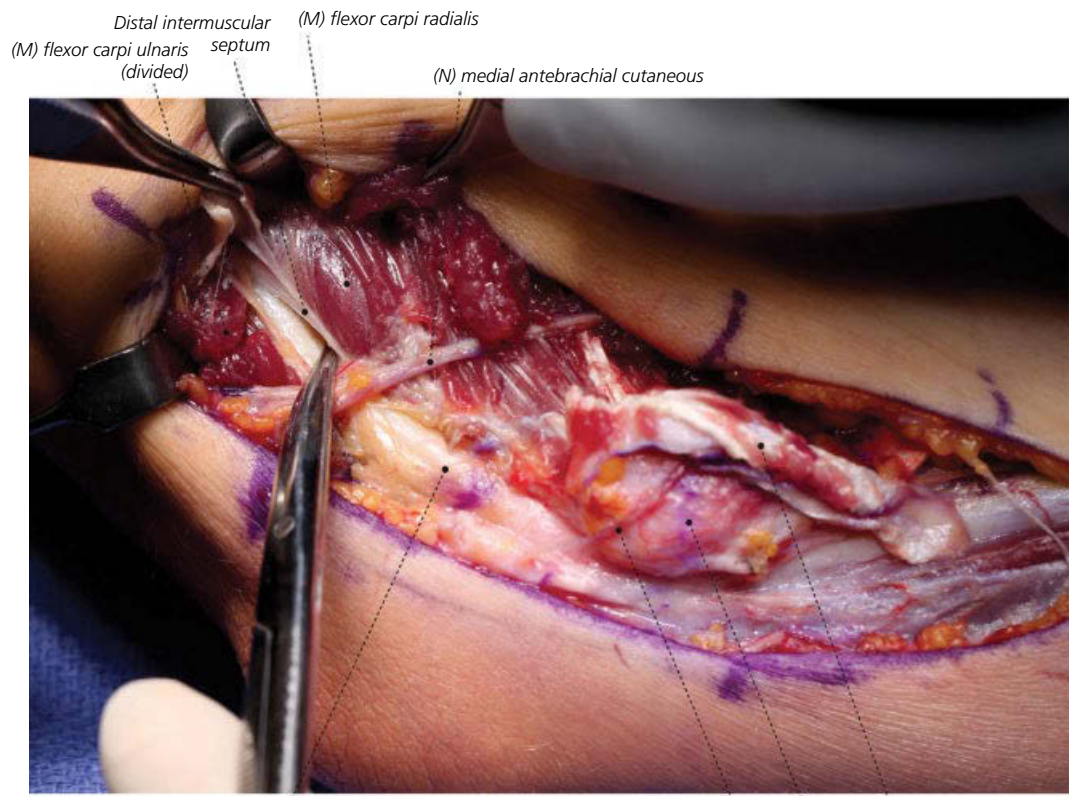
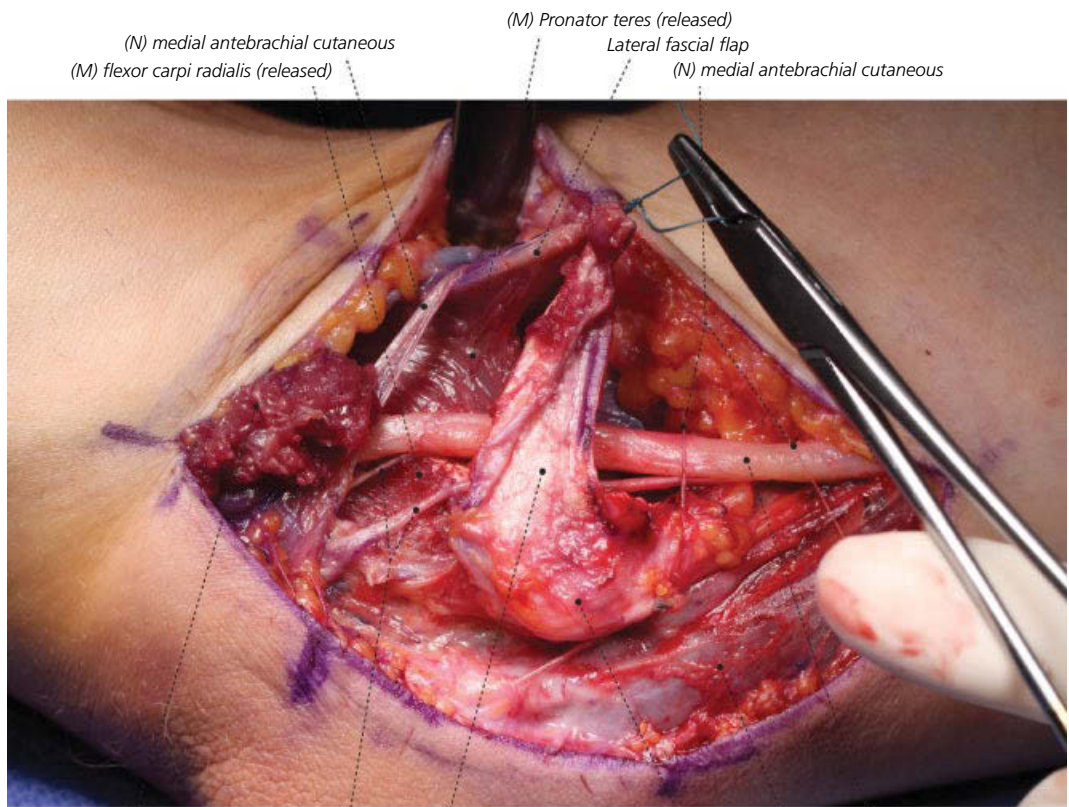


Figure 58.4 (Continued)



(e)

(N) ulnar
Medial fascial flap
(B) medial epicondyle of humerus
(N) medial antebrachial cutaneous



(f)

Figure 58.4 (Continued)

A wrist cock-up splint and ROM exercises for the wrist and hand are also useful in PIN syndrome.

Surgical decompression of the radial tunnel and PIN is approached in a standard manner that addresses all potential entrapment points on the radial nerve (Figure 58.6). Preoperatively, the patient is asked to flex the elbow against resistance with the forearm in neutral to identify the brachioradialis muscle. An inci-

sion is marked on the posterior aspect of the brachioradialis muscle in the proximal forearm. Dissection is carried through the subcutaneous tissues to fascia, where the interval between the brachioradialis and ECRL is identified. There are two landmarks to properly identify this interval: (a) the posterior cutaneous nerve of the forearm runs exactly longitudinally along this interval, superficial to muscle fascia; and (b) there is a colour

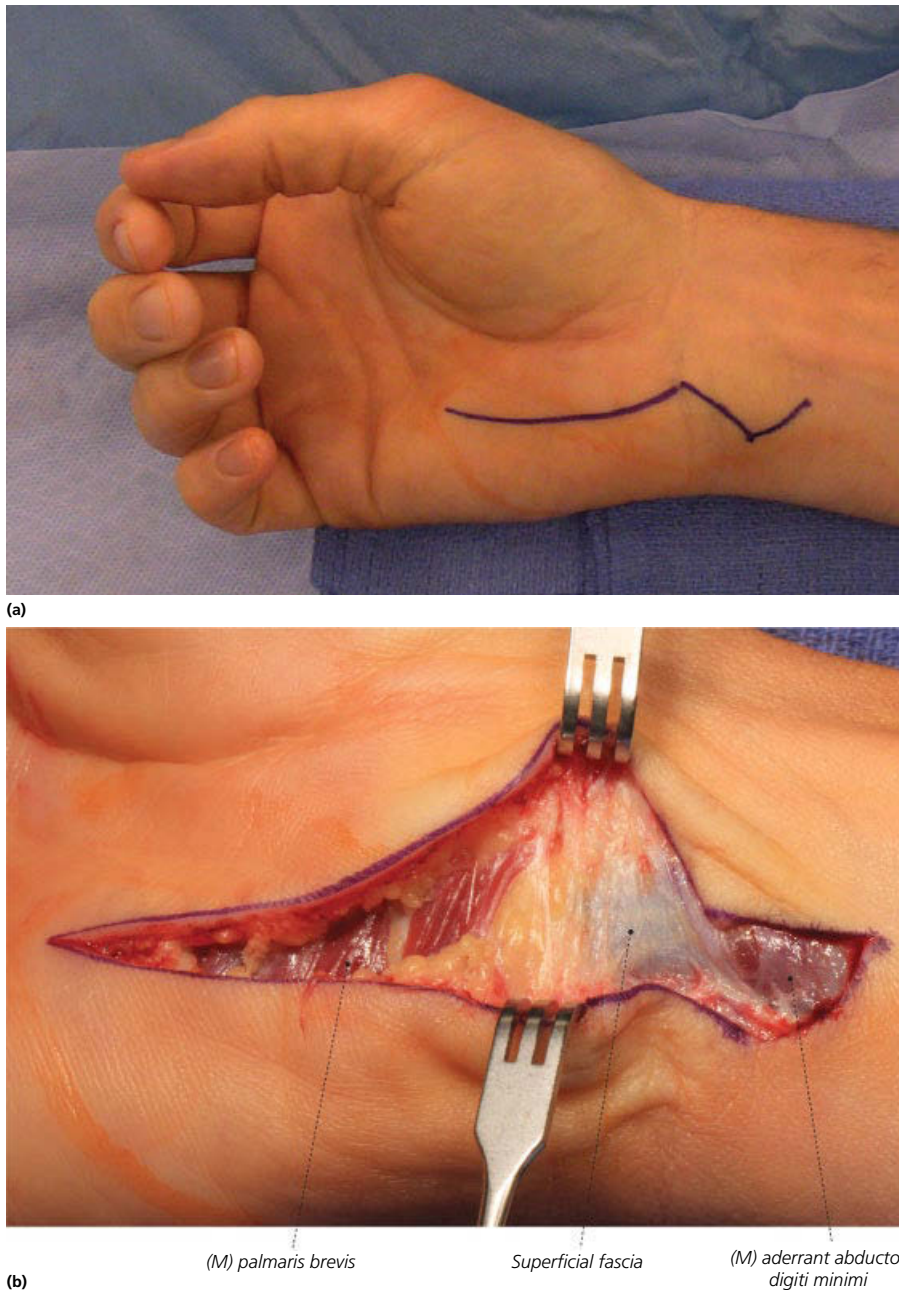


Figure 58.5 Author's preferred technique for decompression of the ulnar nerve at the wrist (Guyon's canal release). **(a)** Design of the skin incision. **(b)** Deep to the subcutaneous tissue, the palmaris brevis and fascia overlying the ulnar neurovascular bundle are identified and subsequently divided. **(c)** The ulnar nerve is retracted ulnarly, and the tendinous leading edge of the hypothenar musculature is identified just radial to the hook of the hamate. **(d)** The deep motor branch of the ulnar nerve is decompressed by carefully dividing the tendinous leading edge of the hypothenar muscles. The motor branch is often not visualized until the initial 2–3 mm of this leading edge is transected. Proximally, the antebrachial fascia overlying the ulnar nerve is divided to complete the decompression. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.

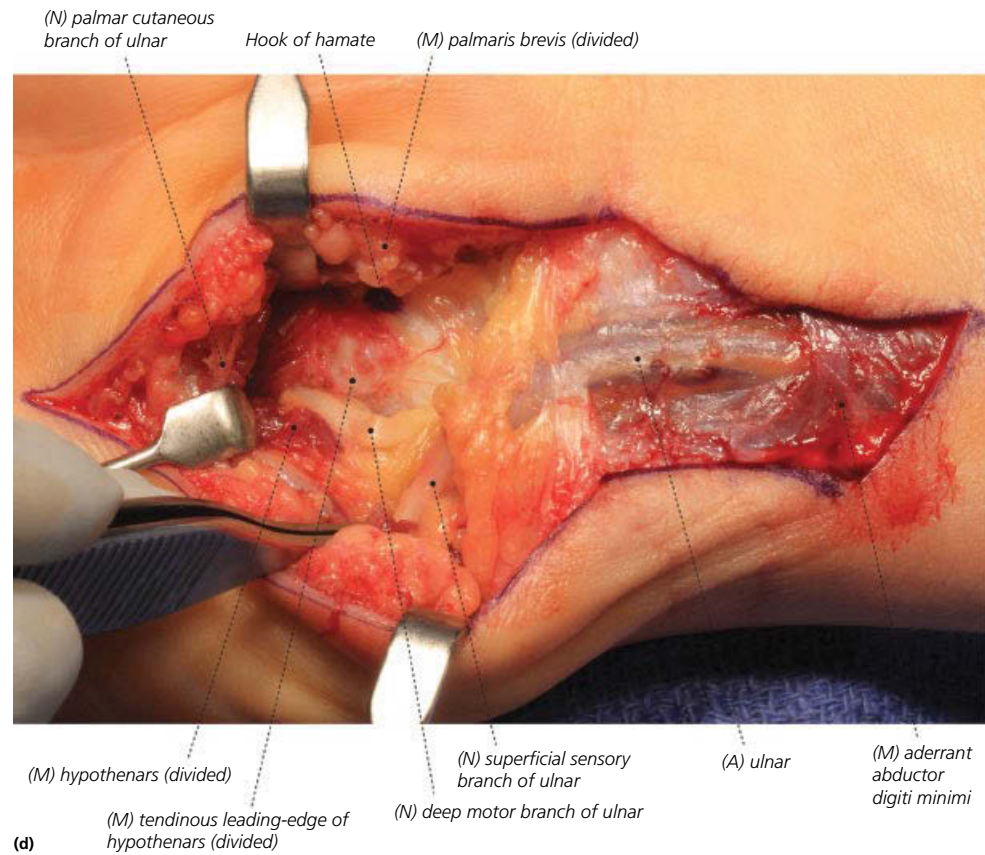
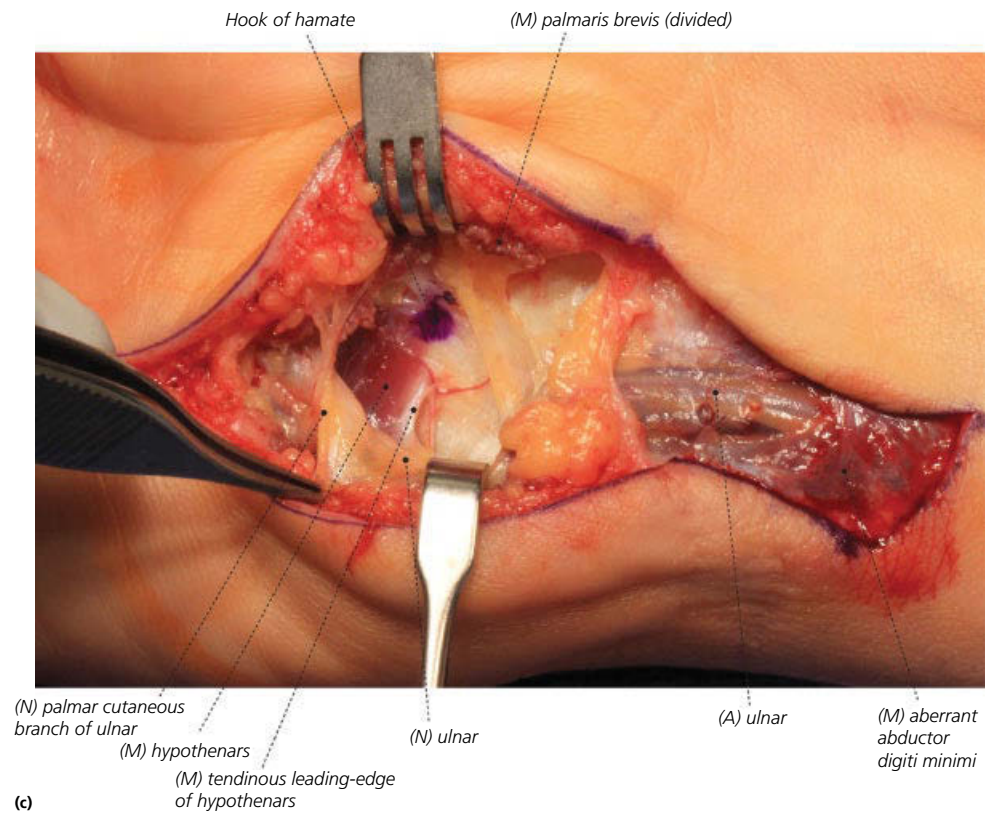


Figure 58.5 (Continued)



(a)

(M) extensor carpi radialis longus



(b)

(M) brachioradialis

Interval between brachioradialis and extensor carpi radialis muscles

Figure 58.6 Author's preferred technique for decompression of the radial tunnel and posterior interosseous nerve (PIN). (a) A straight incision is designed along the posterior margin of the brachioradialis muscle in the proximal forearm. (b) The interval between the brachioradialis and extensor carpi radialis longus (ECRL) muscles is identified, and the fascia is divided longitudinally at this location. (c) Finger dissection is performed in the interval between brachioradialis and ECRL down to the radial nerve. (d) The radial recurrent vessels (vascular leash of Henry) may be seen compressing the radial nerve proximally. These are ligated and divided. (e) The orientation of the branches of the radial nerve at this level: the radial sensory nerve (large) is most radial, the PIN (large) is most ulnar, and the ECRB nerve (small) lies between these. (f) Division of the tendinous edge of the ECRB muscle allows visualization of the arcade of Frohse (tendinous leading edge of the supinator muscle). (g) Complete transection of the arcade of Frohse decompresses the underlying PIN. The nerve branch to the supinator can be seen exiting from the PIN on its posterior aspect. Source: *Nerve surgery*, Susan Mackinnon (ed.), Thieme 2015. Reproduced with permission.



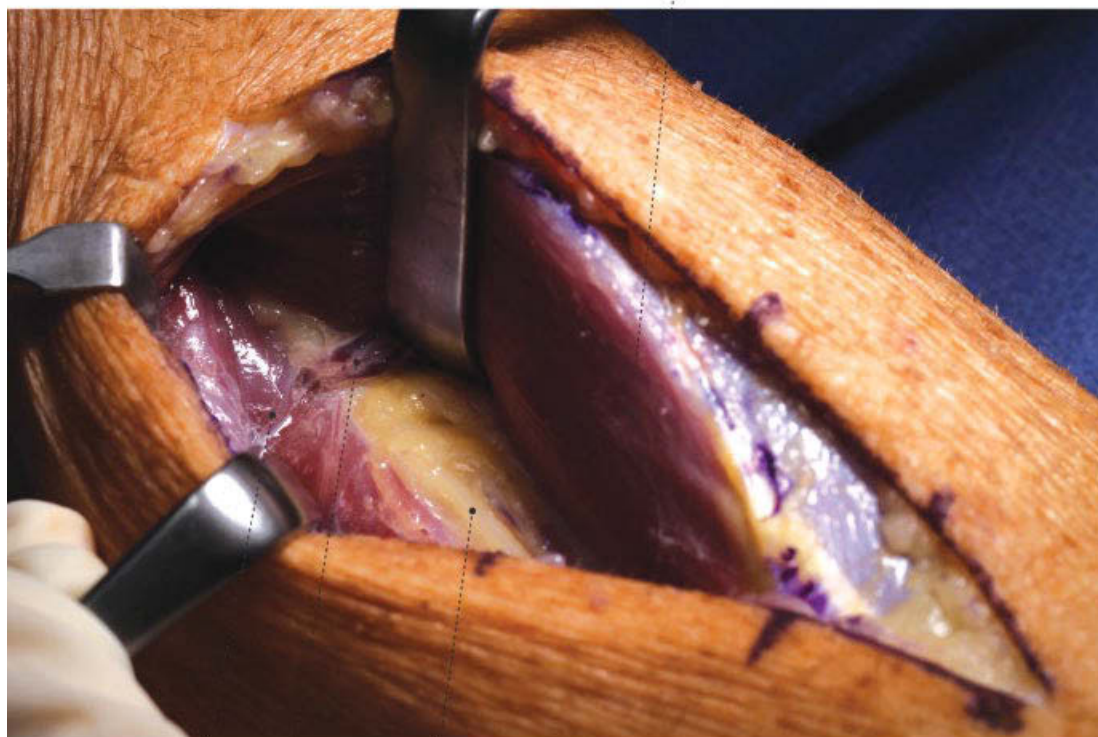
(c)

(M) brachioradialis

(M) extensor carpi radialis longus

(M) extensor carpi radialis brevis

(M) extensor carpi radialis longus



(d)

(M) brachioradialis

Radial recurrent vessels

(N) superficial branch of radial

Figure 58.6 (Continued)

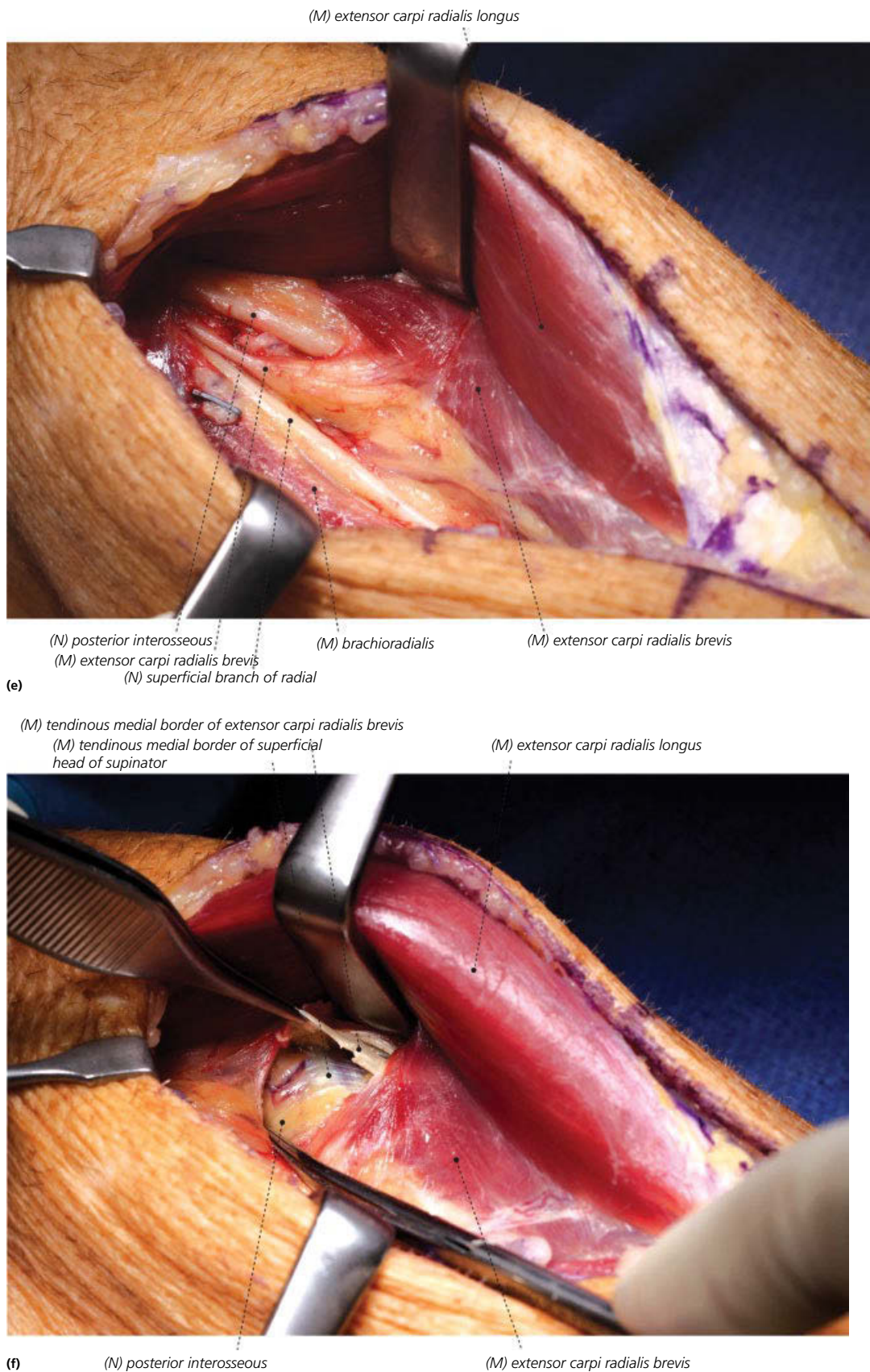


Figure 58.6 (Continued)

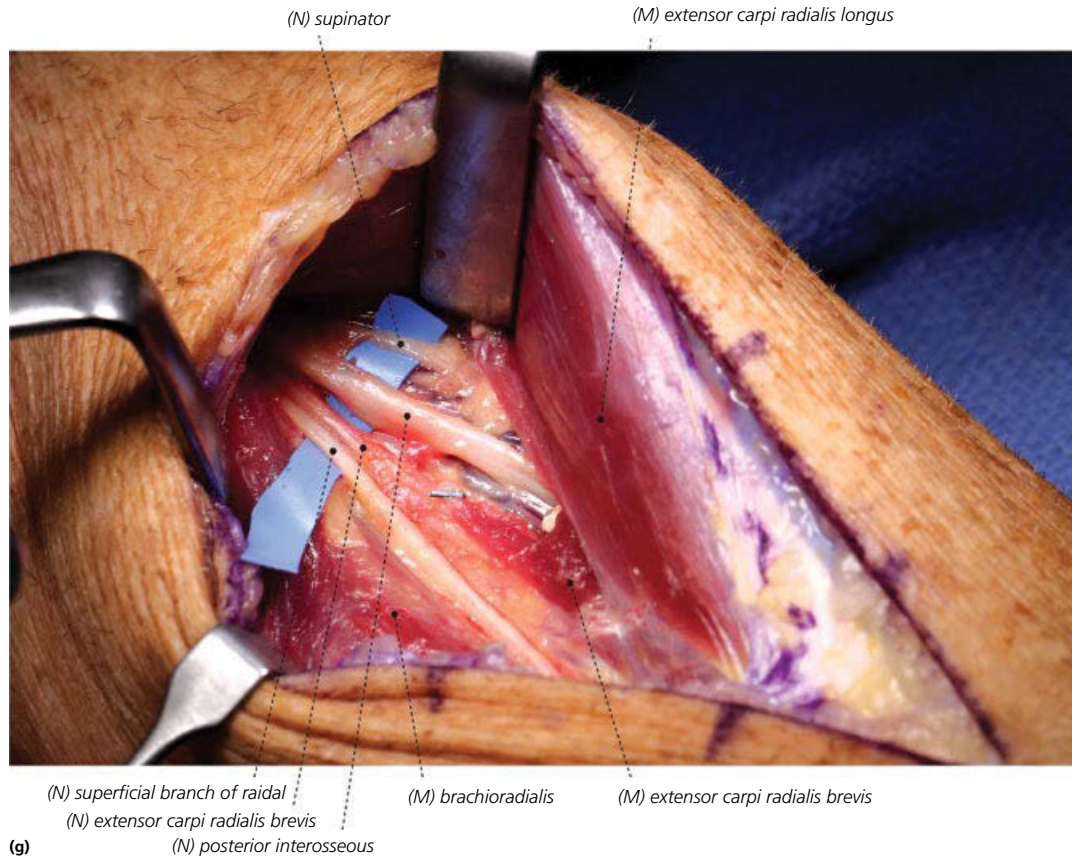


Figure 58.6 (Continued)

difference between the two muscles, with the brachioradialis appearing more red (thinner fascia) and the ECRB appearing lighter (thicker fascia). The posterior cutaneous nerve of the forearm is then elevated and protected, and the avascular plane between the two muscles is finger dissected down to the radial nerve. Deep retractors are placed to improve exposure.

The branches of the radial nerve can always be found in the same orientation: the SRN is most volar and large, the ECRB branch is thinner and lies between the SRN and PIN, and the PIN is large and most dorsal. The PIN is also obliquely oriented as it courses under the superficial head of the supinator. The fascial edge of the ECRB is released first, which allows the supinator muscle to be visualized. The fascial edge of the superficial head of the supinator (arcade of Frohse) is then completely released, which decompresses the underlying PIN. Any crossing vessels (vascular leash of Henry) compressing the PIN should be ligated and divided.

If the preoperative history and physical examination suggest radial nerve compression in the proximal radial tunnel (as indicated by aching pain over the radial nerve above the elbow), more proximal dissection and release may be required to release conjoined fibres of the brachioradialis and brachialis muscles. Occasionally, a second incision is required to achieve adequate visualization and release. If there is associated lateral epicondylitis, then a proximal tenotomy of the ECRB tendon is also performed.

The radial nerve and its three branches should then be once again inspected to ensure no residual compression points are seen. Neurolysis should be considered in patients who present with significant motor weakness or paralysis in the PIN-innervated muscles, especially if scarring of the nerve is identified intraoperatively. Neurolysis should progress until a good fascicular pattern and bands of Fontana are seen.

Postoperatively, the patient is placed in a well-padded dressing. This is removed in 2–3 days, at which point gentle, full ROM exercises are encouraged. Strengthening begins at one month, and return to full activities with no restrictions at two months postoperatively.

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CHAPTER 59

Dupuytren disease

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Definition

Dupuytren disease is a benign fibromatosis, which may lead to finger joint flexion and webspace contractures. The disease usually commences in the ring and little finger ray in the sixth or seventh decade of life and males are more affected than females. Systematic meta-analysis of all published work on prevalence has revealed that it ranges from 0.6 to 31.6.¹ The disease is most prevalent in white people from the north-western part of Europe and their offspring, but also affects other races.

Associated fibromatoses are *Ledderhose disease*, coinciding in 15% of cases with Dupuytren disease² and giving rise to nodules in the instep area of the foot; *Peyronie disease*, which encompasses the formation of plaques within the tunica albuginea of the penis, leading to penile curvature sometimes causing hinderance to sexual intercourse;³ and *knuckle or Garrod pads*, which are nodules at the dorsum of the proximal interphalangeal joints.⁴

Dupuytren disease generally does not disturb finger flexion, although it may be related to trigger finger.⁵ Awkwardness and functional impairment worsen with increasing contracture, especially when the total contracture of a ray becomes more than 90°, and contractures may interfere with job-specific tasks.⁶

There is a subset of patients in whom a more aggressive disease course (Dupuytren diathesis) develops. In these patients, the disease starts early, affects both hands and more family members, and associated fibromatoses are usually present.⁷ As a consequence, treatment is needed earlier and recurrences are more frequent. These patients also have more often radial involvement of the hand.⁸

Dupuytren disease should be regarded as a chronic disease that cannot be cured. In this chapter the current knowledge of this disease will be summarized.

Surgical anatomy

In the palm the fascial structures form a complex three-dimensional network.^{9,10} The longitudinal fibres are organized in a fan shape and form the palmar aponeurosis (Figure 59.1).

The fibres form the pretendinous and the prelumbrical bands, and all pass superficial to the transverse ligament of palmar aponeurosis (TLPA), which is situated at a line joining the proximal and distal palmar crease.¹² The ulnar border of the TLPA blends with the hypothenar fascia and the radial border is continuous with the proximal commissural band, which runs through the first webspace and ends in the thenar fascia at the level of the first metacarpophalangeal joint. Just beyond TLPA, the pretendinous bands divide into three layers (Figure 59.2): Layer 1 is the most superficial layer, which has its insertion in either the skin of the distal palm or that of the proximal phalanx. Layer 2 fibres spiral underneath the neurovascular bundle towards the lateral side of the fingers and form a spiral band, which is present in all fingers, except for the ulnar side of the little finger. Layer 3 fibres dive deep into the hand on both sides of the flexor tendon sheath to insert on either side of the corresponding metacarpophalangeal joint.

The natatory ligament lies just underneath the skin in the distal palm and superficial to the neurovascular bundles (see Figure 59.1). On its ulnar end it is continuous with the hypothenar fascia, and on the radial side with the distal commissural band. The natatory ligament has attachments to the flexor tendon sheath, lines the webs and has extensions along the lateral sides of the digits, where it blends with fibres from the spiral band and is continuous with the lateral digital sheet.

Important sagittal ligaments in the palm are the septa of Legueu and Juvara and are usually just as wide as TLPA and, except the thumb, connect the aponeurosis palmaris with the deep transverse palmar ligament that links the volar plates of all metacarpophalangeal (MCP) joints. Together these structures form nine boxes which on alternating basis hold either the lumbrical muscle and neurovascular bundles, or the flexor tendons (Figure 59.3). Much smaller vertical fibres are dispersed throughout the palmar aponeurosis and anchor it to the overlying skin.¹⁵

The most important digital fascial structures bear the names of Cleland and Grayson.^{16,17} Their detailed anatomical description has undergone significant changes over time and drawings

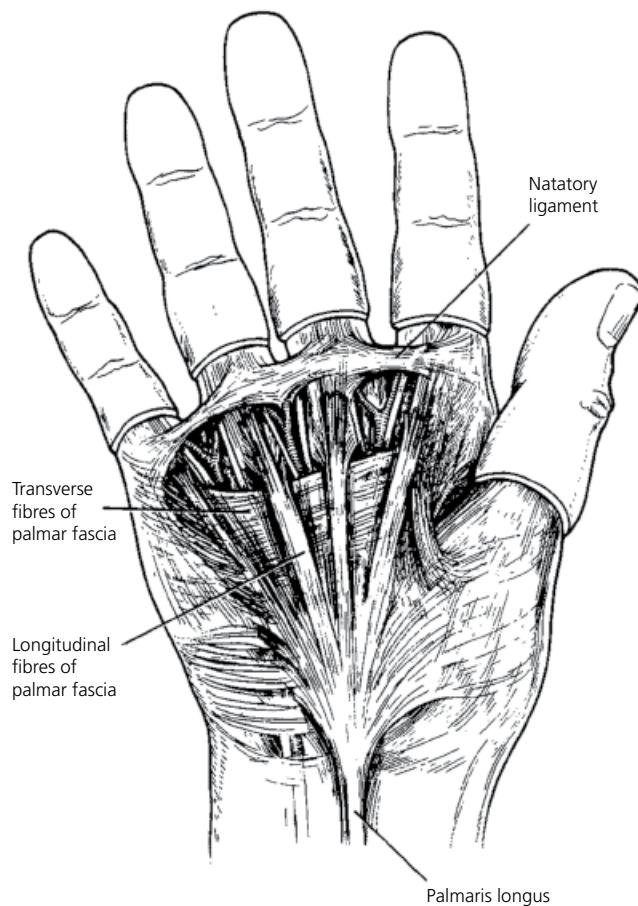


Figure 59.1 Anatomy of the superficial fascial structures of the palm of which the skin and subcutaneous fat has been removed. Source: Smith, 2002.¹¹ Reproduced with permission of Elsevier.

depicting the exact course of their fibres have been conflicting. Basically, there is a structure located palmar to the neurovascular bundle, which is known as Grayson ligament, and a ligamentous structure that lies dorsal to these structures, which bears the name of Cleland. Further detailed dissection studies are necessary to reveal their exact anatomy and that of the lateral digital sheet and retrovascular sheet.

Where the hypothenar muscles pass into the little finger there is a crossroads of fibres which all seem to play a role in Dupuytren disease.¹⁸ At the thenar and in the first webspace there is also a complex three-dimensional network of bands, which may also be involved.¹⁹

Already in the days of Baron Guillaume Dupuytren there was disagreement about the origin of the disease. Some authors, including Dupuytren himself, believed that the disease starts within the fascia. Others felt that it originates also in the tissues between the skin and the fascia.²⁰ There is mounting evidence that the latter theory may be true²¹ and there is consensus regarding the role in disease of some (but not all) of the mentioned fascial structures (Table 59.1). If a well-defined 'band' becomes affected by disease, it is then referred to as 'cord'. McFarlane tried to simplify the often complex phenotype of the disease in the finger by defining anatomic patterns of disease, such as central cord, lateral cord and spiral cord.¹⁰ Further work is required to be able to explain all these findings, including the double spiral around the digital nerve during surgery.²²

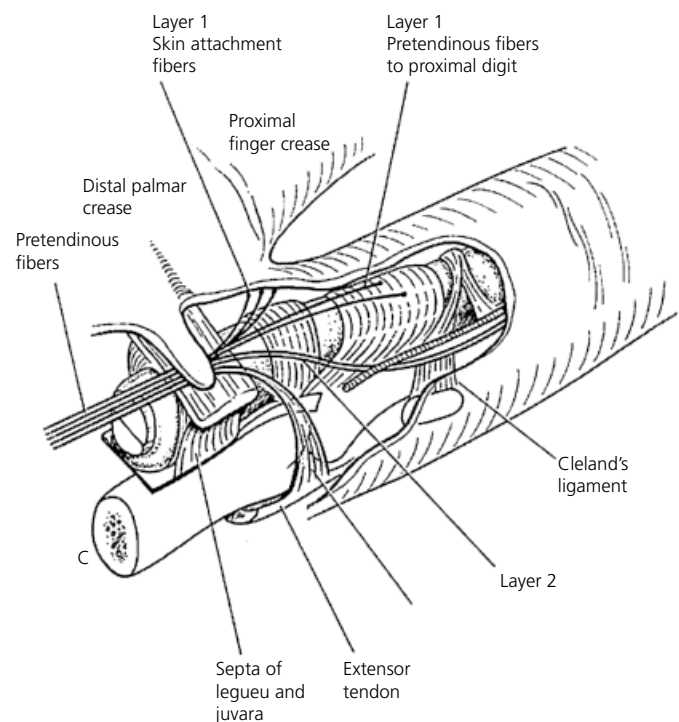


Figure 59.2 Division of pretendinous band in three layers. Source: McGrouther, 1990.¹³ Reproduced with permission of Elsevier.

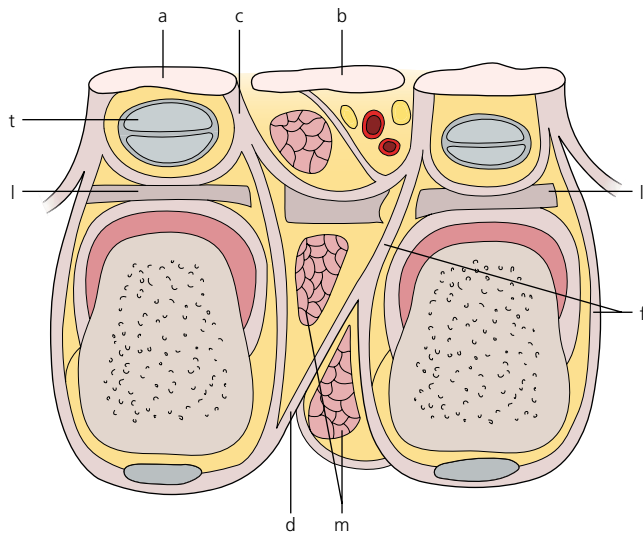


Figure 59.3 Cross-section of the hand at the level of the heads of the metacarpal bones. **(a)** Pretendinous band; **(b)** prelumbrical bands; **(c)** septa of Legueu and Juvara, penetrating the deep transverse ligament of the palm (**f** and **l**) and reaching the dorsal aspect of the hand (**d**); **(m)** interosseus muscles; **(t)** flexor tendons. Source: Legueu & Juvara, 1892.¹⁴

Table 59.1 Various fascial structures in palm and in fingers of interest in Dupuytren disease

Anatomical structure	Name if affected by DD	Clinical relevance
Fascial structures in the palm		
Pretendinous band (PTB)	Pretendinous cord (PTC)	Situated immediately beneath the skin in palm. Responsible for earliest signs of disease in most patients. Divides in three layers distal to transverse ligament of palmar aponeurosis
Prelumbrical band (PLB)	Prelumbrical cord (PTC)	
Layer 1 of PTB	Pretendinous cord in palm; central cord in finger	Situated immediately beneath the skin, distal to line joining proximal and distal palm crease. May cause MCP and PIP joint contracture. Does not displace neurovascular bundle
Layer 2 of PTB (spiral band)	Spiral cord	Contracture causes displacement of neurovascular bundle medially and palmarly. May cause MCP joint contracture. Warning sign: Short–Watson sign
Layer 3 of PTB	Vertical cord	Dives deep into the hand on both sides of MCP joint. May cause painful triggering
Transverse ligament of palmar aponeurosis (TLPA)	–	Runs along line joining proximal and distal palm crease, deep to PTB/PLB. Not affected by DD. Can be left behind during fasciectomy. Will facilitate subsequent surgery when left intact
Ligaments of Legueu and Juvara	–	Connect TLPA to deep transverse ligament
Natatory ligament (NTL)	Natatory cord	Situated immediately beneath the skin and superficial to neurovascular bundle. May cause web contracture
Proximal/distal commissural band	Proximal/distal commissural cord	Proximal band is extension of TLPA; distal band is extension of NTL. Situated immediately beneath the skin in the 1st web; may cause 1st web contracture
Abductor digiti minimi fascia	Abductor digiti minimi cord (ADMC)	Forms Y-shape together with pretendinous band and ulnar neurovascular bundle can always be found proximal to junction. ADCM may have multiple levels
Deep transverse ligament (DTL)	–	Connects volar plates of MCP joint. Together with ligaments of Legueu and Juvara and TLPA forms nine boxes through which flexor tendons and lumbrical muscles and neurovascular bundles pass
Fibres of Grapow	–/nodules in palm?	Dispersed over palmar aponeurosis. Anchor palmar skin to TLPA
Fascial structures in the digits		
Lateral digital sheet	Lateral digital cord	Situated immediately under the skin on each side of each finger; may displace neurovascular bundle towards midline. May cause MCP and PIP joint contracture when involved together with spiral cord and Grayson's ligament inserting to A4 pulley. May also cause DIP joint contracture as lateral cord
Grayson's ligament	Central cord (CC)	Harbours nodules and cord at proximal phalanx. May cause PIP contracture
Cleland's ligament	–/Retrovascular cord ??	Not easily visualized, since situated behind neurovascular bundle. May cause PIP and DIP joint contracture
Transverse retinacular ligament	–	Connects mid slip of the extensor apparatus at PIP joint to the lateral bands and the volar plate. Palmar portion may shorten in severe PIP joint contractures, preventing the lateral bands from sliding back after contracture release
Oblique retinacular ligament (Landsmeer)	–	Connects volar plate of PIP joint to dorsal capsule of DIP joint. May shorten and cause Boutonnière deformity

DD, Dupuytren disease; MCP, metacarpophalangeal; PIP, proximal interphalangeal.

Aetiology

Interplay of both genetic and environmental factors (smoking and alcohol use) sets the stage for the emergence of the disease. Additionally, the disease occurs more frequently when diseases such as diabetes mellitus are present or following long-term barbiturate use.^{23,24} In addition, a recent meta-analysis underscores the association between manual labour and vibration and the development of Dupuytren disease.²⁵

A number of older population studies have suggested that the disease has an autosomal dominant trait with variable penetrance. Using a Genome Wide Association Study it was recently shown that Dupuytren disease is a polygenetic disease. Nine regions in the genome were identified that are strongly associated with Dupuytren disease.²⁶ In six of these regions, genes were located that play a part in the WNT-signalling pathway. In a subsequent study a clear correlation between the genetic load and the presence of certain diathesis factors came to light.²⁷ It has been calculated that the sibling recurrence risk for Dupuytren disease is 4.5 and that the currently known loci that predispose to Dupuytren disease account for 12.1% of the total heritability of the disease.²⁸

Pathophysiology and histology

The cells responsible for the deposition of superfluous extracellular matrix (ECM) proteins in Dupuytren disease are specialized fibroblasts with contractile properties named myofibroblasts.²⁹ Myofibroblasts derived from Dupuytren disease nodules exhibit an abnormal response cycle to mechanical tension with anchorage to the ECM and shortening, which is thereafter locked by the secretion of collagen type I and III and other ECM molecules.^{30,31} Stiffness of the ECM has been found to be crucial in myofibroblast behaviour and research is underway to identify modulating factors of this process.³²

In this respect the work of Luck in 1959 and Lam *et al.* in 2010 is relevant.^{33,34} The former proposed a subdivision in three phases and the latter analysed the respective collagen content:

- The *proliferating phase* is where there is an abundance of disorganized cells present in nodules. Over 35% of the collagen that is deposited is of type III.
- The *involutional phase* is where the number of cells declines but alignment along lines of stress becomes obvious. The amount of collagen type III diminishes (20–35%), whereas the amount of type I collagen increases.
- The *residual phase* of scar-like hypocellular tissue consists of less than 20% of collagen type III.

Clinical assessment

The first manifestations of Dupuytren disease are subtle subcutaneous irregularities in the palm, which may be difficult to discern. Although nodules and skin pits suggest the diagnosis,

the differential diagnosis in that stage should include ganglia and inclusion cysts, occupational hyperkeratosis, callous formation, tenosynovitis, giant cell tumours and epitheloid sarcoma or even metastatic disease.^{35,36}

The natural progression is towards cords that may cause contracture. Once contractures emerge, the diagnosis is usually evident (Figure 59.4), although differential diagnosis of contracture includes dermal scars, congenital conditions (i.e. camptodactyly), stuck trigger finger, tendon bowstringing from pulley incompetence, tendon adhesions or imbalance (as in sagittal band rupture), intrinsic joint contractures and spasticity. Cords can cause extension deficit of any finger joint, although distal interphalangeal (DIP) joint contracture is rare. More often, long-standing proximal interphalangeal (PIP) joint flexion position leads to an attenuation of the central slip, forcing the DIP joint into hyperextension and thereby causing a Boutonnière deformity of the affected finger (Figure 59.5).

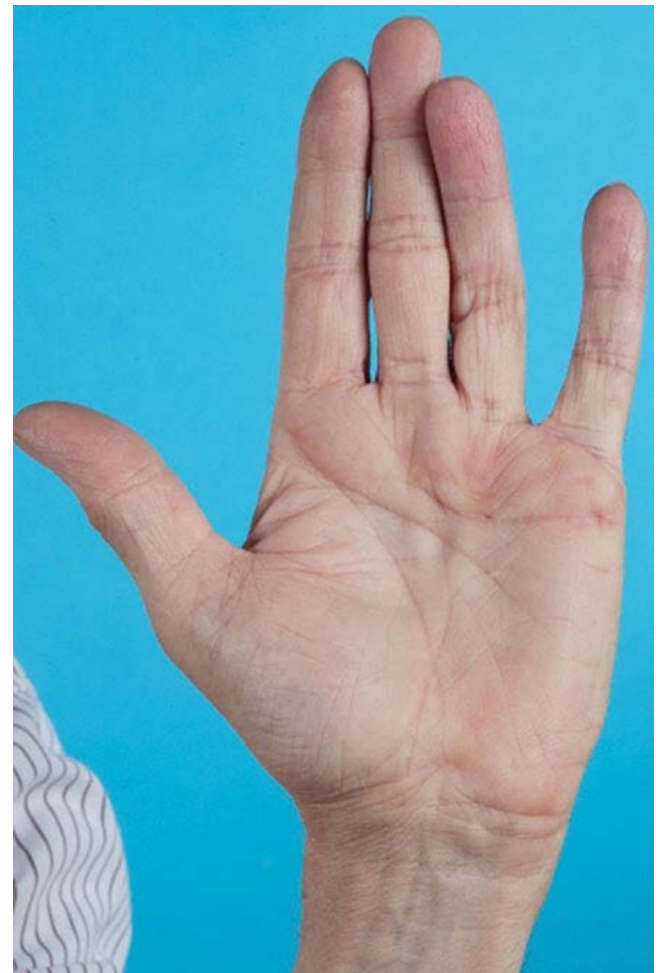


Figure 59.4 Left hand of 63-year-old patient with pretendinous cord in the ring and middle finger ray, and a lateral digital sheet cord on the radial side of the ring finger with skin pitting in the palm and just proximal of the proximal interphalangeal joint crease and radial deviation of the affected finger. Photograph courtesy of Bert Tebbes. Reproduced with permission of University Medical Center Groningen (UMCG).



Figure 59.5 Patient with Dupuytren disease of right little finger with Boutonnière deformity.

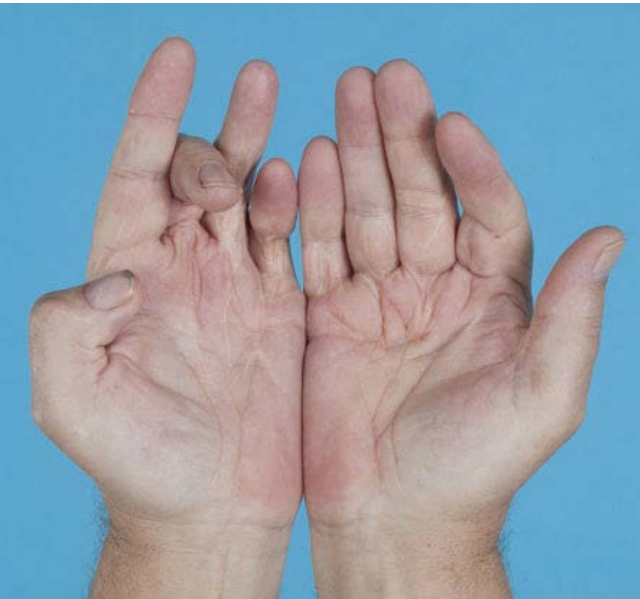


Figure 59.6 Sixty-year-old male with severe Dupuytren disease of the left thumb, middle ring and little finger and of the right little, ring, middle and index finger. Photograph courtesy of Bert Tebbes. Reproduced with permission of University Medical Center Groningen (UMCG).

Dupuytren disease often affects both hands, without synchronicity and may cause pathology in any ray (Figure 59.6). Nodules may be painful when compressed but this sign usually disappears with time. Cords are almost always insensate. The most practical grading systems are those of Iselin, and Tubiana and Michon (Table 59.2).^{37,38} Iselin's staging system is very practical for quick scoring, such as during prevalence studies. Tubiana's system is more sophisticated and its 1986 modification makes the system relatively complex, hampering its widespread application.³⁹ The down side of this system is that progression into a higher stage may take 5–40° and is therefore

Table 59.2 Grading systems for Dupuytren disease: every ray is given a stage number

Described by	Stage	Meaning
Iselin	1	Dupuytren disease nodules and cords without contracture
	2	MCP joint contractures
	3	MCP and PIP joint contracture
	4	Boutonnière deformity with MCP and PIP joint contracture and DIP joint hyperextension
Tubiana	0	No signs of Dupuytren disease
	N	Nodules only. No contracture
	I	Total passive extension deficit (TPED) smaller than 45°
	II	TPED in between 46 and 90°
	III	TPED in between 91 and 135°
	IV	TPED in between 136 and 180°

MCP, metacarpophalangeal; PIP, proximal interphalangeal.

not linear, but stepped. Therefore, for follow-up studies it is better to register and report the passive extension deficit (PED) by joint and its sum by ray (TPED). Apart from these objective data, patient-rated outcome measures (PROMs) have become increasingly important to evaluate outcome and there is an increasing demand to harmonize their use among researchers.^{40,41}

Management principles

In the discussion of the treatment strategies for Dupuytren disease, the following aspects need to be addressed (Table 59.3): how the method manages skin, fascia and joints; indication for and best timing of treatment; specific treatment benefits, risks and complications; immediate outcome and time of disability; late outcome including definition and rate of recurrence; additional treatment options.

Description of treatments, short-term outcome and complications

Non-operative treatments

To relieve pain in early-stage (stage N) Dupuytren disease, patients may be advised to wear padded (biking) gloves. Steroid injection is also advocated by some.⁴²

The newest injection treatment modality employs collagenase, which weakens pathological Dupuytren disease tissue. The drug (Xiaflex®/Xiapex®) is injected into the palpable cord that is responsible for a joint contracture of at least 20°, and at a minimum of 24 h later the treated joint is extended using light force to rupture it. If the obtained result is not satisfactory, the treatment may be repeated after 30 days. Skin ruptures occur frequently and the risk of skin rupture may be reduced by delaying the extension manoeuvre for as long as two weeks.⁴³

In double-blind studies, collagenase injection was found to be significantly more effective than placebo in releasing

Table 59.3 Overview of the treatments for Dupuytren disease, including results and recurrence risks

Treatment	Indication	Mean disability time	Results	Complications	Recurrence	Disadvantages	Level of evidence
Limited fasciectomy ^{53,71,79}	>30° contracture; painful nodules ^{53,71,79}	6 weeks	79% reduction of contracture ⁷¹	19% Haematoma, skin necrosis, neurovascular damage ⁷³	In 21% within 5 years increase of TPED of >30° compared to 6 weeks result ⁷⁹	Invasive treatment. Complications in 19% of patients. 6 weeks of rehabilitation ^{71,73}	Level 2
Dermofasciectomy ^{5,4,85,86}	Recurrences. Aggressive type in patients younger than 40	8 weeks	Comparable to limited fasciectomy	Haematoma, loss of skin graft, infection, neurovascular damage	8.4% after 6 years; ⁵⁴ 12.2 after 3 years ⁸⁶	Most invasive treatment, further disadvantages same as with limited fasciectomy + those of skin grafting	Level 3
Percutaneous needle fasciotomy ^{57,76,79,103}	Isolated cord patients; wish for minimal invasive treatment	<1 week	63–99% reduction of contracture ^{76,103}	Nerve damage, tendon damage skin fissures especially in the treatment of recurrences	In 85% within 5 years increase of TPED of >30° compared to 6 weeks result ⁷⁹	‘Blind technique’ high risk of recurrence	Level 2–4
Clostridial collagenase ^{44,104,105}	Contracture associated with a palpable cord	<2 weeks	Mean clinical success 65%. MCP joint 77%; PIP joint 40% (correction within 0–5° of full extension)	Local, temporary reaction to injection	In 37% ⁷⁷ increase of PED of >20° after 3 years ⁸⁰	Treatment of more than one cord is still off label; expensive drug	Level 1–3
Radiotherapy ^{47,82}	Disease in nodular phase	2× 1 week: 2 treatment sessions of 5 irradiations on consecutive days	Stable in 59%; progression in 31%; regression in 10%	32% erythema and other skin conditions ⁴⁷	NA	Only effective when applied early in early-stage disease	Level 3

PED, passive extension deficit; TPED, total passive extension deficit; MCP, metacarpophalangeal; PIP, proximal interphalangeal.

contractures.⁴⁴ In 77% of the MCP joints and in 40% of the PIP joints, collagenase injection released the contracture to 0–5°. Adverse events, such as swelling and pain, occurred in 97% of cases. Because Xiaflex/Xiapex action is limited to type I and III collagen, but digital nerves and arteries are primarily type II and IV collagen, they are not at risk from enzymatic damage.

Halfway through the twentieth century, the first report on radiotherapy emerged as a possible treatment modality for Dupuytren disease.⁴⁵ Keilholz reported results of a protocol of irradiating the affected area and surrounding margin five times a week in two sessions, 6–8 weeks apart with a total of 30 Gy. After three months, the authors found that disease was stable in 92% of Tubiana stage N, N/I and I cases, had regressed in 7% and had progressed in 1%. In 75% a non-significant reduction in size and consistency was found.⁴⁶ Complications related to the treatment are only minor late toxicity (skin atrophy, dry desquamation) in 32% of the patients. Radiotherapy is most effective when applied early after disease onset.⁴⁷

Operative treatments

Management of the diseased fascia

Limited or selective fasciectomy (LF) is still the most commonly performed surgical procedure for Dupuytren disease.⁴⁸ Only the diseased tissue is removed. The generally accepted indication for treatment is a painful nodule that does not respond to conservative measures or a progressive flexion contracture of MCP joints of 20–30° or any PIP joint contracture.^{49,50}

Segmental fasciectomy (SF) is a less invasive variant of LF in which only 1-cm-long pieces of diseased fascia at strategic places are removed.^{51,52} As with all surgical techniques for Dupuytren disease, MCP joint contractures can be very well corrected, but the technique is less successful for PIP joint contractures and the complication rate is similar to that for LF.⁵³

Dermofasciectomy (DF) is the removal of both skin and underlying fibromatosis. It is advocated by some for primary Dupuytren disease cases, since they have found that recurrences are more delayed.⁵⁴ Most only apply it in cases with aggressive disease and early recurrence.

Open fasciotomy (OF) was first proposed by Henri Cline in 1787 for Dupuytren disease,⁵⁵ although there is no record that he ever performed it. Through transverse incisions, cords responsible for contractures are identified, isolated from the neurovascular bundles, and divided. In the short term it is just as effective as LF.⁵⁶

All above-mentioned procedures are usually performed under regional or general anaesthesia. This is one of the major differences with percutaneous needle fasciotomy (PNF). With PNF, cords are percutaneously divided at multiple levels with a needle under local anaesthesia during the same session.⁵⁷ By using intradermal local anaesthesia only, nerve conduction can be maintained and monitored during the procedure, enabling the patient to warn the surgeon if the needle stimulates a nerve. Some authors advocate combining PNF with steroid injection, but the evidence that supports this is debated.^{58,59}

PIP joint management

One approach for persistent PIP joint contractures after fasciectomy is sequential transverse division of the flexor tendon sheath, the check rein ligaments and the release of the accessory collateral ligaments.^{60,61} Another just as effective approach is intraoperative gentle manipulation.⁶² Attenuation of the central slip of the extensor may also be a significant factor in the inability to sustain correction of PIP joint contractures greater than 60°.⁶³

Messina and Messina advocated continuous traction via an external fixator to reduce higher Tubiana contractures to such an extent that fasciectomy became easier,^{64,65} and various progressive distraction mechanisms have since been developed and successfully applied.⁶⁶

Skin management

When there is only limited shortage of skin, common fasciectomy incision options include a longitudinal incision from palm to finger, a zigzag-type incision, or a transverse incision at the level of TLPA and at the joint creases, or a combination of these. For SF, small C-shaped incisions are used. McCash has shown that transverse incisions can be left open as long as bare tendons are not exposed.⁶⁷ For the fingers, one or more Z-plasties in the longitudinal incisions or YV-plasties in the zigzag incisions can be employed to gain extra length for closure.

If skin transposition is not sufficient to close a defect, or if the skin is removed, it can be augmented or replaced using skin grafts. Skin grafts have also been advocated as 'firebreaks' and in some hands proved very successful in the prevention of recurrence.⁶⁸ Full-thickness skin grafts are recommended because they are more durable and undergo less secondary contraction than split-thickness skin grafts. Full-thickness skin grafts are usually taken from the ipsilateral extremity or the groin. Donor sites are closed primarily but leave scars, one of the disadvantages of this treatment. Other drawbacks are the increased duration of the procedure, a prolonged rehabilitation period, and increased risk for complications: a graft may not take, thereby extending the recovery period to a mean of eight weeks.

Apart from grafts, a great variety of local (homo- or heterodigital), regional⁶⁹ or even free flaps can be used to manage skin shortage with very good long-term results in one case report.⁷⁰

Postoperative management

Following LF, SF and DF, the treated hand is usually put in a soft bulky dressing for a week, sometimes reinforced by a splint. After PNF, a very small dressing for just one day suffices. As soon as the wound permits, patients begin flexion and extension exercises and this process is often supervised by hand therapists.

Early outcome

With LF an average reduction of total passive extension deficit (TPED) of 79% at six weeks postoperatively has been reported.⁷¹ Results were best at the MCP joint (87% reduction of passive

extension deficit (PED)) and worst at the PIP joint (49% reduction of PED). Most authors agree that surgical prognosis of Dupuytren disease of the PIP joints is worse, especially in the little finger, regardless of treatment technique.⁷²

Treatment-specific complications of surgery are common.⁷³ The cumulative complication rate for LF has been found to be 19% and even higher in DF. Skin slough can usually be treated conservatively. Haematoma formation should be evacuated before settling, thereby preventing fibrosis and early recurrence. Joint stiffness is most difficult to treat and functionally is sometimes worse than the original flexion deformity. Nerve injury occurs in approximately 1% of primary and, if feasible, should be handled by direct repair. Surgery may also cause injury to the digital arteries, the incidence of which is most likely underreported. In a worst-case scenario, especially after repeated surgery, vascular injury may result in gangrene of the finger, necessitating partial or complete amputation. Mean recovery period to normal hand use is for LF on average six weeks and for DF two weeks longer.⁷⁴

PNF is also very effective for MCP joints, but less so for PIP joint contractures. Early outcome of PNF is similar to that for LF when the initial TPED is less than 90°, but significantly less successful above this.⁷¹ Complications are limited to skin tears and the risk to damage nerves or tendons is less than 1%. The cumulative risk of serious complications of PNF is much less than that for the more invasive treatments.^{75,76} Skin fissures may occur with adherent skin, and in recurrent cases are almost the rule, although one can try to prevent this by releasing the skin from the cord.⁵⁷ Since they heal without leaving a trace they usually do not bother the patient.

Long-term outcome: recurrence and extension

One problem with recurrence is the wide disparity in definitions, with few randomized studies that compare treatment modalities and few studies with sufficient follow-up.⁷⁷ Another issue is differences in treatment modalities: diseased fascia may be incised (OF, PNF), partly removed (SF), fully removed (LF, DF; with or without skin grafting), injected with proteolytic enzymes, or subjected to ionizing radiation. In some studies, recurrence is defined qualitatively as any sign of new tissue in a previously operated spot.⁷⁸ In others, recurrence is defined quantitatively by measuring the PED of each joint and the TPED for each ray, and recurrence is defined as an increase of TPED by a certain number of degrees (20° in the collagenase studies, 30° in most other studies).^{44,79}

Long-term outcome of non-operative treatments

Ketchum investigated the efficacy of triamcinolone injections. Fifty per cent of his patients experienced reactivation of disease in the nodules 1–3 years after the last injection, necessitating one or more injections.⁴²

The recurrence rate in collagenase studies is defined as increase of more than 20° of PED in joints, which had an initial complete correction. Because this definition excludes recurrence in joints that were not initially fully corrected (and have higher

recurrence rates,⁵⁰ it skews results favourably and cannot be compared to other definitions of recurrence which include all treated digits. Using this definition of recurrence, recurrence following collagenase was 19% after 2 years and 37% after 3 years.⁸⁰ At present, there is only one very small study with long-term follow-up (8 years) data from only eight patients. Recurrence was found in 67% of the MCP joints and 100% of the PIP joints.⁸¹

The progression rates of radiotherapy is as follows: After a mean of 10 years, 87% of Tubiana stage N patients and 70% of Tubiana stage N/I patients remained stable or regressed. In more advanced stages, the rate of disease progression increased to 62% (stage I) or 86% (stage II). Sixty-six per cent of the patients showed a long-term relief of initially reported side effects (i.e. burning sensations, itching and scratching, pressure and tension).⁴⁷ A recent non-randomized trial with long-term follow-up showed a benefit of radiotherapy over observation alone.⁸²

The basic issue that needs further study, however, is the time versus progression relation in Dupuytren disease which, in the author's experience, is more likely exponential than linear. Since radiotherapy has only been found to be effective in early stages of the disease, it is unclear what it is actually being accomplished.

Long-term outcome of operative treatments

Using reappearance of deformity necessitating additional surgery as the definition of recurrence, Dias reported 15% recurrence at a mean of 27 months after LF.⁸³ Others, using the reappearance definition, have published recurrence figures of up to 73% 7 years after LF.⁸⁴ A randomized controlled trial (RCT) comparing LF and PNF with a follow-up duration of 5 years showed a recurrence rate following LF of 21%.⁷⁹

For the long-term outcome of skin grafting in Dupuytren disease, the work of Ketchum is instructive.⁸⁵ He found no recurrent disease in 36 hands of 24 patients an average of 4 years after fasciectomy and 'firebreak' skin grafting. The incidence of extension outside the grafts was 8%. Other authors, however, have not been able to achieve similar results with this technique. In a very well-executed RCT, no difference in recurrence rate was found at 3 years comparing LF with or without firebreak skin grafting, with a recurrence rate at the PIP joint of 12.2% in both groups.⁸⁶ It is, however, critical to distinguish the above technique of simple intercalated 'firebreak' skin grafting and dermofasciectomy and skin grafting. Armstrong *et al.* reported a recurrence rate with the latter technique of 8.4% of 143 treated rays.⁵⁴

Recurrence rates of SF are similar to those of LF.⁵² Recurrence rates at PIP joint are always higher than those at MCP joints and irrespective of the technique used.⁸⁷ PNF recurrence rates are relatively high compared to more aggressive treatment modalities. After 3 years, Foucher found, just as we did, recurrence rates in approximately 60% of cases and our 5-year recurrence rates were 85%.^{79,88} Hovius *et al.* are in the process of

testing if this high recurrence rate can be reduced by adding grafted fat by means of lipofilling to the cord that has been extensively divided. Long-term results are pending.⁸⁹

Some patients experience recurrence earlier than others. Van Rijssen *et al.* and Pess *et al.* showed that there is a strong correlation between age at the moment of treatment and chance for recurrence.^{76,79} This is probably the most important diathesis characteristic that influences recurrence. In an effort to prevent or postpone recurrence, especially in such cases, researchers have found that drugs such as tamoxifen and 5-fluorouracil *in vitro* were able to delay fibroblast proliferation.^{90,91} Subsequently, some added these measures to their treatment regimen. Systemic treatment with 5-fluorouracil in an RCT unfortunately proved ineffective.⁹² In a similar design, Degreef *et al.* found that highly dosed neo-adjuvant tamoxifen improved the surgical outcome of segmental fasciectomy.⁹³ The same group has successfully attempted to delay recurrence by placing cellulose implants in the gap created by segmental fasciectomy.⁹⁴

Treatment of recurrence

Evidence for the best treatment for recurrence is scarce and there is a need for comparative studies in this respect. What is known is that there is a greater risk of complications during LE.⁹⁵ PNF can also be used safely for the treatment of recurrences and is just as effective as during primary PNF.⁷⁹ With skin involvement and skin scarring, the likelihood for a skin fissure or rupture becomes greater and more effort needs to be directed to the release of the pathology from the skin.⁵⁷ Collagenase can also be used safely in recurrences.⁹⁶ Surgery following PNF is in my personal experience not more difficult than virgin surgery. No unequivocal opinion exists with regard to surgery after treatment with collagenase.⁹⁷ The role of radiotherapy for recurrent disease is yet unclear.

Rehabilitation

Splinting and hand therapy are commonly advised in the treatment of Dupuytren disease after surgery and a variety of protocols are used.⁹⁸ Evidence for the use of either of them is lacking, however.^{99–102} And some are of the opinion that only patients with rapidly reoccurring extension deficits may benefit from night splints.

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CHAPTER 60

Fractures and dislocations in the hand

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Introduction

Hand surgery always involves teamwork between the surgeon, the patient and the therapist. One cannot get a successful outcome without the cooperation of the other two. The following chapter deals with the diagnosis and management of fractures of the phalanges and metacarpal bones in adults and children.

Paediatric fractures of the digits

Assessment, classification, management and complications

The differences between paediatric and adult hand fractures lie in the elasticity of children's bones and the potential disruption of their growth. Children's bones have increased water content and plasticity; and tend to fracture incompletely with trauma. Incomplete fractures are seen as bows (bending forces), buckle fractures (one cortex buckles with compression) or greenstick fractures (one cortex ruptures under tension, the other is intact or buckles). The plastic deformation may prevent complete reduction of the fracture when manipulated, but most fractures still do not require open operations, but can be managed with closed or percutaneous techniques, due to the remodelling potential.

The assessment of the fracture is clinical and radiological. The child will have point tenderness in the non-displaced fracture over the physis, which should increase suspicion of a fracture. If plain X-rays in two, if not three planes, cannot explain pain or deformation, MRI or CT will. The tenodesis test adequately demonstrates any rotational deformity, as the fingers all point to the scaphoid tubercle. If in doubt; compare with the other hand. Angular deformities in children may remodel; rotational deformities will not and need to be corrected.

Any fracture interfering with the growth plate has a potential for disrupting normal growth. Paediatric fractures involving growth plates are classified according to Salter and Harris,¹ Ogden² expanded this in 1984 (Table 60.1).

The most common fracture pattern encountered in the fingers is a Salter–Harris II, which is satisfyingly corrected using

a simple manoeuvre with a pen, over which the finger is manipulated (Figure 60.1).

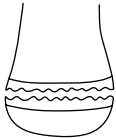
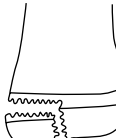
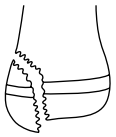
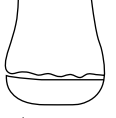
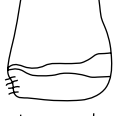
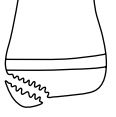
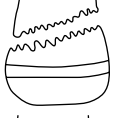
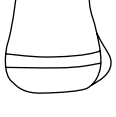
Crush distal phalanx fractures and nailbed injuries are the most common paediatric fracture. These occasionally need a K-wire or an orange needle to pin the distal fragment in place if large enough. A special type of nailbed injury called Seymour fractures are displaced Salter I or II fractures of the distal phalanx, with avulsion of the proximal edge of the nail from the eponychial fold and the germinal matrix often interposed in the fracture itself (Figure 60.2). Accurate and timely debridement and repositioning of all tissues is paramount to adequate growth of both digit and nail. The clinical presentation can be confused with a mallet finger (because the extensor tendon inserts itself in the epiphysis whereas the flexor tendon inserts itself in the metaphysis), but a true lateral X-ray and a thorough examination under anaesthetic should reveal the injury.

Fractures through the neck of the phalanges are usually only found in children and mostly involve some unfortunate interface between fingers and doors. Al-Qattan divided them into three categories, which determined prognosis and treatment: type I fractures are undisplaced, type II fractures are displaced but maintain some bone-to-bone contact, and type III fractures have no bone-to-bone contact.³ Type III is furthermore subdivided according to the location of the distal fragment, type IIID especially being associated with an avascular fingertip. Since the smallest displacement is poorly tolerated, all types apart from type I require open reduction and K-wiring, but have progressively worse prognoses (Figure 60.3).

Hand therapy intervention for these patients is minimum as they rarely become stiff at the distal interphalangeal (DIP) joint and start functional use once the bulky bandage or splint has been removed. Older children may require a small volar splint of the affected digit until pain has settled and oedema control work is completed as necessary.

It is worth remembering that there is no gain in changing the dressings in nailbed injuries before 10 days, unless you are worried about infections, which of course is a different matter.

Table 60.1 The paediatric fracture: the management and potential complications according to the Salter–Harris classification, including later additions

Classification	Clinical	Configuration	Management	Complication
I: Through physis, showing increased width of physis	Point tenderness only		Conservative	Possible shortening
II: Through physis and metaphysis	Occurs >10 years old		Manipulation under anaesthetic if displaced	Minimal shortening
III: Through physis and epiphysis	>10 years old		Accurate surgical reposition	Can result in chronic disability as extending into joint
IV: Metaphysis, physis and epiphysis	Any age, rare in hand		If displaced consider open reduction internal fixation	Intra-articular so can result in chronic disability and can cause premature focal fusion of the joint
V: Compression of the epiphyseal plate	Usually from axial loading		Conservative	Growth disturbances
VI: Open fracture in which part of the physis is missing	Open fracture		Initial fat grafting to prevent physeal bar; later reconstruction with bone graft	Growth disturbances
VII: Epiphyseal plate only			Requires accurate repositioning as intra-articular fracture	No impact on growth
VIII: Isolated injury to the metaphysis			Usually can be manipulated under anaesthetic or, if necessary, fixed with K-wire	Potential injury related to endochondral ossification
XI: Injury to the periosteum				May interfere with membranous growth

Adult fractures

I always consent my patients for bone grafts, just in case I need it and I have never regretted speaking to the patient about this option before the operation.

Metacarpal fractures

Metacarpal fractures can be divided into their anatomical location or configuration but most commonly occur in the little or ring fingers and the thumb.

Neck

Neck fractures of the fourth and fifth metacarpal are usually treated conservatively. They can be manipulated and the digit neighbour-strapped, accepting that the patient will lose the knuckle and may have some extensor lag, unless there is a rotational element. If there is rotation, the rotation must be corrected surgically.

Neck fractures in which the head is completely off the metacarpal or that involve the index or little fingers may be treated with mini-condylar plates, or for all fingers with H-plates (the

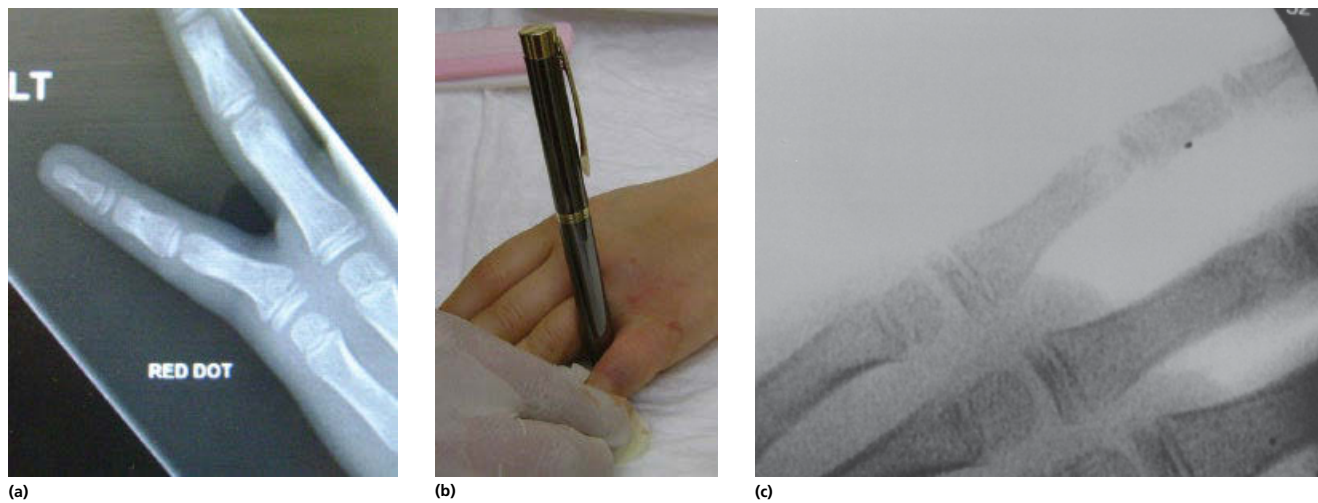


Figure 60.1 Example of Salter–Harris II. (a) Prereduction radiograph. (b) Mode of reduction. (c) Postreduction radiograph.



Figure 60.2 Seymour fracture.

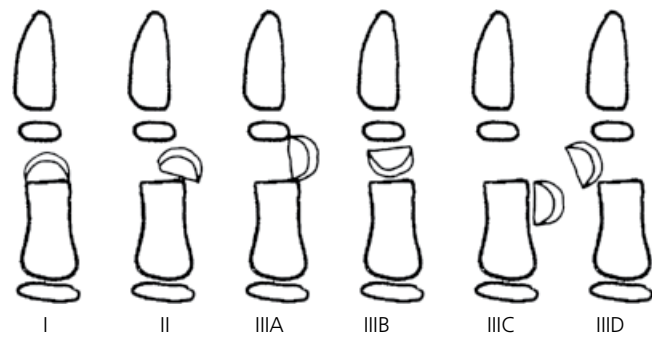


Figure 60.3 Classification of fractures of neck of proximal phalanx in children. Source: Al-Qattan, 2001.³ Reproduced with permission of Elsevier.

two-hole fixation on each side of the fracture can be transverse (remember) or intramedullary K-wiring.

Intra-articular

Intra-articular metacarpal head fractures are best seen on Brewerton's views and can be fixed with countersunk screws, if the fragments are large enough. The fragment must be three times the size of the screw head to hold it. If this is not possible a primary joint replacement or a free joint from the second toe must be considered. Very rarely does this joint get considered for fusion in the fingers.

Shaft

Metacarpal shaft fractures can be divided according to their configuration into transverse, spiral, short and long oblique, butterfly fragments and comminuted.

If the fracture can be held in position with one or two bone-holding forceps, you can fix it with screws (which must be placed perpendicular to the fracture) (Figure 60.4).

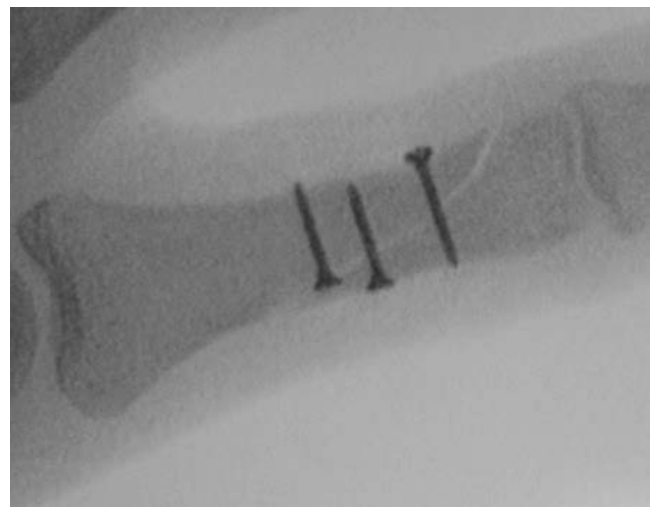


Figure 60.4 Screw fixation of spiral fracture. Note the spiral placement of screws.

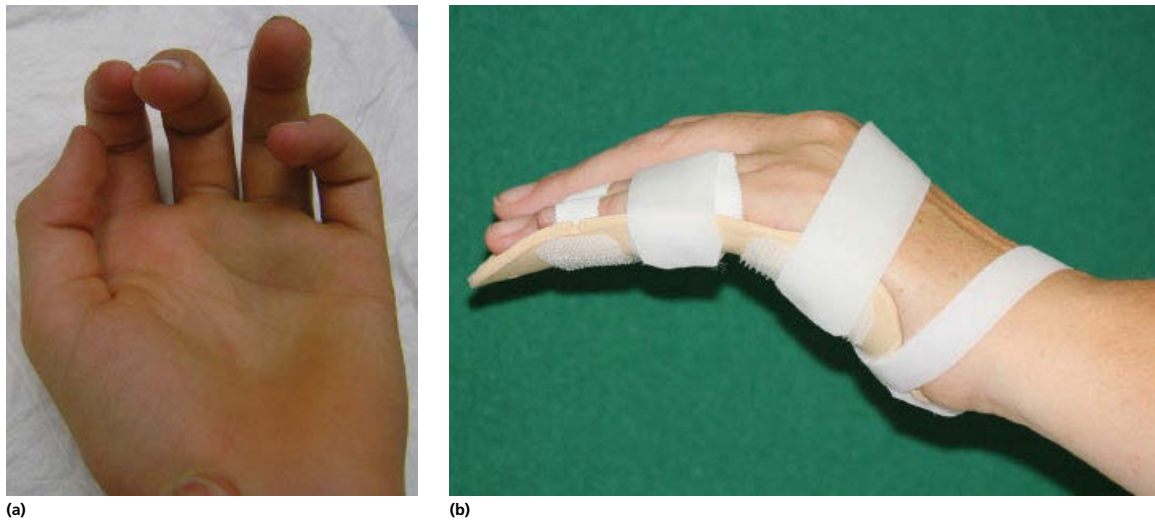


Figure 60.5 (a) Rotation: spot the odd one out. (b) Volar hand splint.

If you cannot maintain position with a bone-holding forceps, you must consider a fracture fixation which involves a plate (or even an external fixator for the first or fifth metacarpal). For a simple transverse fracture intramedullary K-wiring is also a possibility. Crossed K-wires often distract the fragments rather than compressing the fracture and should be discouraged.

It is important to keep checking the position of the fingers, even when the fracture looks to be perfectly aligned, before drilling and inserting screws, to avoid rotation (Figure 60.5).

When applying plates to fractures it is important to understand the difference between locking and non-locking plates. Conventional plates require precise adaptation of the plate to the aligned bone. The bone is pulled towards the plate when the screws are tightened and screw insertion can therefore alter the fracture position if everything is not precisely positioned. In locking plates the threaded screw head fits into the threaded plate hole and locks when tightened, thus providing a stable constellation of plate and screws. The bone is therefore not pulled towards the plate when the screws are tightened: the underlying cortical bone perfusion is not disrupted and the screws are unlikely to loosen from the plate, even if they have been inserted into a bone graft or fracture gap.

There are two plates to consider in metacarpal shaft fractures: a normal plate and a bridging plate. If the fracture is simple (i.e. transverse or short oblique), a normal plate can be used, bent appropriately to produce compression of the far cortex. A bridging plate (which is also a locking plate) is best viewed as an extramedullary splint, which 'bridges' the part of the bone which is unsuitable for direct fixation, as in very comminuted or bone-grafted fractures. With a dorsally placed locking plate, it is not necessary for the screws to reach the far cortex.

Plates may be placed on the lateral cortex of the metacarpals, to avoid the adherence of the extensor mechanism, though this

flies in the face of fracture fixation principles, which are based on the forces exerted on the fracture when clenching the fist.

If unsure whether a fracture needs operative intervention, splint the fracture and re-X-ray after one week; if the position is not maintained, it needs to be surgically corrected, but remember to act on the X-ray no later than a week to 10 days maximum.

Base

A reversed oblique view is a useful additional view to a true lateral and three quarters radiograph in demonstrating injury at the base of the fourth and fifth metacarpals (in addition to the adjacent carpal bones and carpometacarpal (CMC) joints).

Metacarpal base fractures, which cannot maintain position, are almost always fixed with percutaneous K-wires, rarely plates, as there is not enough proximal bone for a plate.

Stable simple metacarpal fractures can be treated with buddy strapping, compression bandage and reduced function for 3–4 weeks until they are pain free and stable. Patients must limit their movement, otherwise they will develop large amounts of callus as the fracture heals.

Many patients experience swelling and pain. Resting in a custom-made thermoplastic volar/dorsal splint in the position of safe immobilization (Figure 60.5b) can help ease this and allow the patient some light hand-function using the unaffected digits while fracture healing occurs. Feehan and Bassett discuss how custom-made splints reduce complications compared with off-the-shelf splints or plaster of Paris.⁴ Hand-based splints are made for head and neck fractures, and longer splints for shaft and base fractures. Unfortunately the research on rehabilitation is limited. Cochrane reports by Poolman *et al.* showed there was no evidence of which method to splint and protect a fifth metacarpal neck fracture was the most effective.⁵

Feehan discussed how experienced hand therapists can mobilize the adjacent joints around a potentially unstable fracture, as an alternative to immobilization, in the first three weeks to achieve good long-term function.⁶

Early active mobilization of the affected digits by removing the splint just for exercises is often used to prevent stiffness and allow good tendon glide. Passive exercises will be introduced once healing has occurred, normally around six weeks and patients will have movement controlled until this time.

After surgical repair, scar management is essential to avoid adhesions of the extensor mechanism. Depending on the nature of the fracture, an extensor lag at the metacarpophalangeal (MCP) joint may be visible, and the therapist can work with the patient to resolve this.

After basal fractures of the little and ring finger metacarpal bones some patients complain of discomfort when shaking hands again. This is due to intermetacarpal stiffness and the inability to arch the hand and oppose the little finger to the thumb utilizing the hypothenar muscles. This motion must always be restored with targeted exercises.

Thumb metacarpal fractures

Two base-of-thumb fracture patterns must be mentioned separately: Bennett's and Rolando's:

- A *Bennett's fracture* is an intra-articular fracture dislocation of the thumb metacarpal base that has a fragment attached to the palmar beak ligament and is usually fixed with K-wires.
- A *Rolando's fracture* is also an intra-articular fracture of the base of the thumb metacarpal, but consists of three fragments, with a T or Y configuration and is usually fixed with a T or Y plate.

Thumb metacarpal fractures, which are treated conservatively, are usually put in a forearm-based thumb splint, immobilizing the fracture for 3–4 weeks and protecting it for 6 weeks. This splint must maintain the first web space so patients do not develop adduction contracture. All finger joints are mobilized fully.

Phalanges

Distal phalanx

Distal phalanx fractures are classified into tuft, shaft or intra-articular fractures. They account for about half of all digital fractures seen.

Tuft fractures rarely need treatment themselves, but are often associated with nailbed injuries which must be treated as open fractures and the nailbed matrix meticulously apposed.

Shaft fractures can usually be treated conservatively, but a longitudinal K-wire for a transverse fracture or the occasional cerclage wire or even screw may be necessary for the longitudinal fracture.

The treatment of *intra-articular fractures* depends on the size of the fragment. Small fragments can be treated conservatively

in a mallet splint, but when the dorsal or the volar fragment in a fracture/dislocation of the distal phalanx is large enough, the collateral ligaments are attached to the fragment, and the rest of the distal phalanx becomes unstable on the middle phalanx.

In volar fractures the flexor digitorum profundus (FDP) pulls the attached fragment away from the distal phalanx, and in dorsal fractures the fragment is held in place by the extensor tendon while the FDP pulls the distal phalanx volarly, the latter resulting in a bony mallet or a mallet fracture. When the fragment is dorsal, Ishiguro's method of fixation is recommended.⁷ The dorsal wire supports the dorsal fragment and is placed first with the DIP joint in flexion; the fracture/dislocation is then reduced and the longitudinal wire is placed under image intensifier guidance (Figure 60.6a). In volar fractures the fragment, if large enough, can be secured with a screw. If, however, it is too small to withstand instrumentation, then the FDP can be fixed to the distal phalanx by bony anchors, pull-out sutures, or by sutures passed through the distal phalanx transversely using a white needle drilled through the bone. If the joint is unsalvageable a primary fusion is best.

Therapy for tuft fractures often includes a small volar splint if this helps settle the pain and rest the DIP joint (Figure 60.6b). Patients should not perform strong gripping with this digit until the fracture has healed. Active motion at the DIP/PIP joints and oedema control is commenced as soon as possible. Desensitization techniques and scar management are also often needed before the patients feel comfortable to use the digit normally.⁸

Mallet fractures must be treated with DIP joint immobilization for six weeks, followed by a weaning period of two weeks. The splint used varies from centre to centre but a small dorsal splint seems to be the most effective at holding full DIP joint extension and preventing complications such as skin maceration (Figure 60.7). Off-the-shelf stack splints often provide sub-optimal correction of DIP joint extension and are often so long that they limit PIP joint flexion. This can lead to unnecessary stiffness of this joint.

Proximal and middle phalanx

Proximal phalanx fractures are more common, can be the most challenging to treat and can lead to deformities due to soft tissue imbalance and alteration of bone length. Shaft fractures of the proximal and middle phalanges are managed according to their configuration, though splinting and percutaneous screw fixation or wiring is preferred. Plates can be used, but complication rates are high, probably due to the extensor mechanism getting irretrievably stuck to the scar tissue.⁹

Stable proximal and middle phalanx fractures are often placed in a custom-made hand splint for protection, pain relief and to ensure that the interphalangeal joints do not contract while oedema resolves. The splint keeps the interphalangeal joints in neutral and Coban wrap is often used as the first line of intervention for oedema control and prevention of joint contractures. Early motion and tendon glide of both the flexor and extensor mechanism is essential. Protection splints are worn until fracture



Figure 60.6 (a) Ishiguro frame. (b) Volar gutter distal interphalangeal joint splint.



Figure 60.7 Dorsal mallet splint.

union occurs clinically. Dorsal blocking splints are very useful if there is also soft tissue damage or instability at the PIP joint as the patient can easily mobilize the joint within this splint.

It makes no sense to fix these fractures and then keep them immobile because tendon adherence occurs quickly.¹⁰

The most common complication found is PIP joint extensor lag or fixed flexion deformity and once these occur it is very difficult, if not impossible to correct fully with therapy if over 30°. This most commonly occurs after proximal phalanx fractures of the little and ring fingers when the patient can easily hyperextend their MCP joint and after surgical repair when the tendons become adhered. The hyperextension of the MCP joint must be controlled by dorsal blocking splintage (Figure 60.8) or strapping to allow the extensor mechanism to improve the lag. Longer term night splintage may be required for PIP joint fixed flexion deformities. If the PIP joint has a very tight end-feel many therapists use plaster of Paris serial casting to improve range of motion.

These fractures take at least six weeks to heal, so protection is required for longer than some metacarpal fractures. Many patients experience intrinsic tightness and will require flexion strapping to improve flexion once healing has occurred.

Intra-articular proximal interphalangeal joint fractures

PIP joint fractures are difficult and can potentially lead to severe disability and multiple operations and therefore have many solutions.

External frames rely on the principle that the periosteal sleeve of the fractured bone, when pulled, will push the bony fragments together rather like a Chinese finger-trap. There are



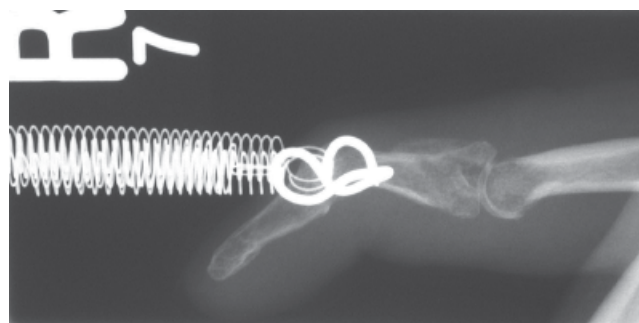
Figure 60.8 Example of a dorsal splint for proximal phalanx injuries.

many methods. Some rely on an elastic band pulling on the frame (Suzuki frame¹¹ and the Banjo frame¹²), some rely on the bend in the K-wires to put the periosteum under tension,¹³ and yet again others rely on springs or hinges to achieve the same effect (S-Quattro¹⁴) (Figure 60.9a).

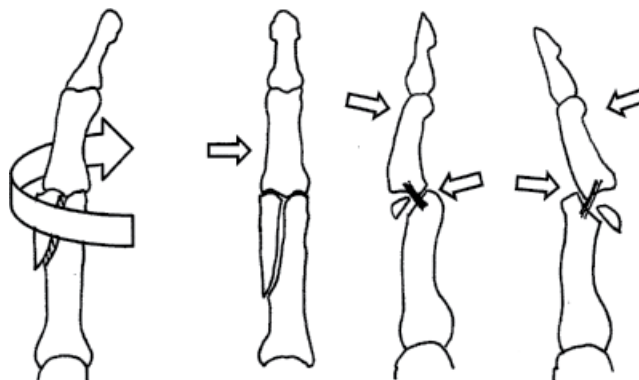
The success of these frames relies on the joints involved being mobilized while maintaining the fracture in a stable position. However, for the majority of these fixators the degree of traction exerted depends on how much the finger is flexed (the joint surfaces are not round), which is, to some degree, compensated for by the elastic element of the frame. The Banjo addresses this point because the wire frame is made so that the forces pulling on the digit are constant through the whole range of motion.

If the fracture/dislocation of the PIP joint still tends to dislocate after the two main K-wires have been inserted in a rubber band or spring system, a third transverse wire (reduction wire) can be placed through the base of the middle phalanx. When the finger is bent, the long proximal wire of the frame pushes against the reduction pin and the dislocation is reduced.

Unicondylar fractures have been classified according to their radiological appearance and on the four different types of forces hypothesized to produce the fractures.¹⁵ All fractures were treated with screws, single or multiple wires: the statistical analysis revealed no difference in outcome according to the fracture types or how long after the operation they were mobilized, though screw fixation seemed to give better results (Figure 60.9b).



(a)



(b)

Figure 60.9 (a) Spring-based traction frame for intra-articular fracture. (b) Weiss-Hastings classification of unicondylar fractures of the proximal phalanx. Arrows indicate direction of force. Source: Weiss & Hastings, 1993.¹⁵ Reproduced with permission of Elsevier.

For unstable dorsal fracture/dislocations of the PIP joint (dorsal coronal, class III, Weiss-Hastings) a method similar to the Ishiguro method described for the DIP joint has been described recently.¹⁶ The primary K-wire is drilled through the fragment from the volar surface of the middle phalanx after the fracture has been reduced percutaneously with a bone-holding forceps; the K-wire is then retracted dorsally until it is flush with the volar cortex, and then finally a dorsal blocking wire is inserted through the head of the proximal phalanx.

For the PIP joint that has an unsalvageable fracture, there are a few other options: fusion, volar arthroplasty, hemi-hamate, a joint replacement or a free joint transfer.

A volar arthroplasty¹⁷ is used when the volar lip is beyond salvage: through a volar incision all but the most volar collateral ligaments are excised, the joint is hyperextended, an incision between the volar plate and collaterals is made and the volar plate is advanced into the joint to cover the defect (usually up to 5mm). The volar plate is secured through pull-out sutures, taking care to avoid pinning down the lateral bands.

A hemi-hamate graft¹⁸ is employed because the dorsal portion of the hamate looks like the volar lip of the PIP joint: the joint is approached from the volar side, the volar plate is detached distally and the ruined volar lip is excised (with a saw) to make room for the dorsal portion of the hamate (taken with

an osteotome). The graft is secured with a couple of screws and the position checked with a lateral X-ray (as a hamate graft must provide adequate volar support (lip)). The volar plate is then reattached.

The choices for PIP joint replacements are many: proximal interphalangeal replacement (PIPrTM),¹⁹ total metacarpophalangeal replacement (TMPrTM), the Saffar, the Digitos,²⁰ DJ03A, the Mathy, Avanta PIP SRA, RM finger joint replacement system²¹ and Swanson's.²² Some joint replacements do well; some have failed repeatedly and been taken off the market, confirming the PIP joint as one of the hardest joints to replace well. Free joints taken from the second toe are a life-long solution and give useful movement, though not a full range.²³

Early hand therapy after any PIP joint injury is essential for a good outcome. Small fractures within the PIP joint can be treated with a dorsal blocking splint, which prevents hyperextension, Coban wrap for early oedema control and early mobilization. Controlling oedema is very important as if it persists especially in this joint it can limit joint motion for many months. As well as PIP joint motion it is essential for the patient to perform good DIP joint motion as often intrinsic tightness occurs and remains a concern for months. Flexion straps are often used to try to resolve this tightness once the fracture has healed (Figure 60.10).

If external fixation has been utilized to stabilize the fracture again oedema control around the fixation is important and Coban works well for this. Early motion and tendon gliding of both the PIP joint and DIP joint is important. Patients are generally supplied with a custom-made splint to protect the fracture and fixation and prevent flexion deformities.

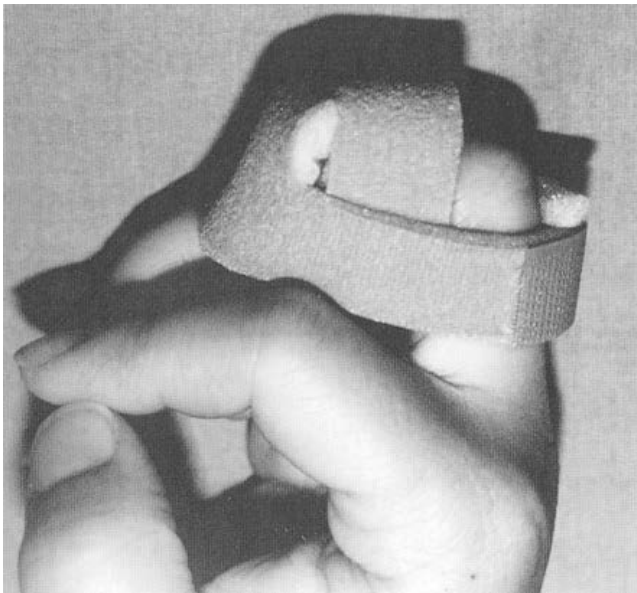


Figure 60.10 Example of a flexion strap to increase interphalangeal joint flexion.

Postoperative management and hand therapy

Volar plate injuries

Volar plate injuries can occur at any of the above joints but are usually diagnosed at the PIP joint with point tenderness, if not a dorsally unstable joint. Ninety per cent of these injuries occur dorsally and they are common in ball sports. Severe volar plate avulsion can occur after dislocation of the PIP joint and will require surgical repair to restore stability of the joint. This type of injury commonly occurs together with injury to other soft tissue structures around the PIP joint, such as the collateral ligaments, and if untreated can lead to flexion contractures of the affected joint.

Most injuries are treated conservatively and the healing time is usually 4–6 weeks.

In adults with stable PIP joints, buddy strapping may be enough to protect the volar plate and collateral ligaments while healing. Oedema control around the affected joint is essential, as is early motion of all the digit joints.

In children and hyperextending adults a dorsal blocking splint prevents hyperextension and tension on the volar plate while it heals. This blocking splint may start in slight flexion at the injured joint but should be straightened as pain eases so the patient does not end up with a flexion deformity. Early active motion of the digit is essential to prevent adherence of the flexor tendons, especially in more severe injuries (Figure 60.11).

A volar PIP joint injury will need treating in an extension splint if the central slip has been damaged.

Ulnar and radial collateral ligament injuries to the MCP joint

All ligamentous injuries can be classified into three grades:²⁴

- Grade I: Pain, but no laxity
- Grade II: Laxity, but a firm endpoint, stable arc of motion
- Grade III: Grossly unstable, no firm endpoint.

The most common ligament to be injured in the hand is the ulnar collateral ligament (UCL) of the thumb. Thumb radial collateral ligament (RCL) damage can occur, but it is uncommon, however RCL tears of the MCP joints can occur if lateral force is applied to the digits when in flexion at MCP joints.

When the thumb UCL is injured, X-rays and high-resolution ultrasound will show whether there is an associated fracture and Stener lesion, indicating a complete tear of the ligament. The clinical diagnosis is made by stressing the ligament with the thumb in flexion and comparing with the other side. Incomplete injuries are treated by splinting, complete injuries by bony anchor fixation of the ruptured ligament, with screw fixation of the bone if the fragment is large enough. It is important to fix the concomitant tear in the capsule as well to obtain stability. Complete tears will never heal by splinting alone as interposition of the adductor aponeurosis prevents apposition of the torn ends.

The thumb acquires a Z-deformity in chronic UCL tears, which may be repaired surgically by a bony anchor, free tendon

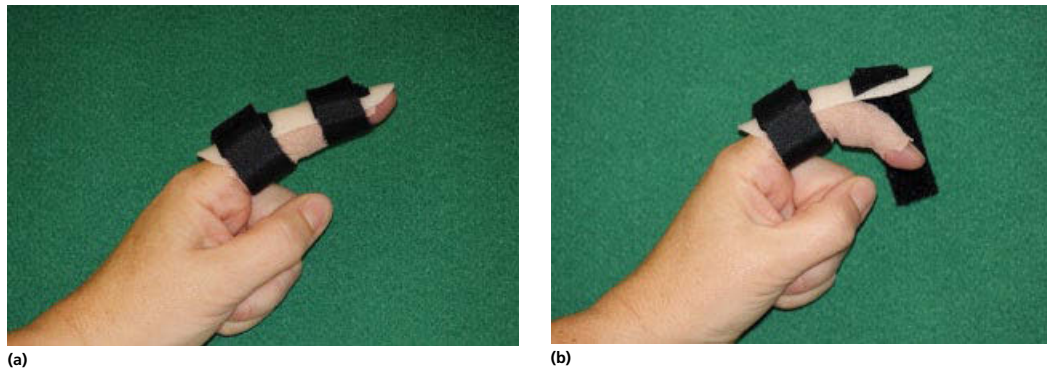


Figure 60.11 Dorsal blocking splint and exercises.

graft or adductor advancement (adductor pollicis in detached and reattached to the ulnar side of the proximal phalanx at the MCP joint), or as a last resort the joint can be fused.

Damage to the RCL of the digits is diagnosed by passive flexion of the MCP joint and ulnar pressure causing pain, and X-rays (Brewerton's views) may show avulsed bone. Grade I and II can usually be treated conservatively with buddy strapping and splinting, while the ligament in grade III is fixed with a bone anchor with the MCP joint at 45° when tensioning the repair or splinted in radial deviation.

Chronic injuries can be repaired with tendon grafts or the joint fused.

Collateral injuries of the digits are effectively managed by buddy strapping of the affected digit with its neighbour until stability and pain improves, usually after 4–6 weeks.

Sometimes a small thermoplastic splint is made to support the affected joint if extra protection is required while allowing good tendon glide at the other joints.

Protection of the thumb MCP joint UCL is essential, as stability is required at this joint. A custom-made thumb Spica splint which only immobilizes the MCP joint is the splint of choice for most hand therapists. This is worn full time whether the ligament has been surgically repaired or is being conservatively treated for 4–6 weeks. Commonly after surgical repair the splint is removed after two weeks for scar massage as this often becomes sensitive. Gentle exercises may commence after four weeks.

Management principles and operative options

As a rule, any major injury starts with the bony fixation to provide stability for the finer neurovascular repairs and for the forces exerted by the repaired tendons. This stabilization is ideally so stable that it allows immediate mobilization. Therapy aims must include early controlled motion, pain and oedema control to prevent stiffness, joint contracture and tendon adherence. The therapy programme must be tailored to the individual

needs of the patient and their injury. Surgeons must be confident that they can stabilize a fracture to allow early motion before intervening, otherwise a longer period of immobilization can lead to a poor result. Immobilization of the hand/digit after soft tissue injuries or digital fractures can lead to very poor functional outcomes for the patient. Although the literature on early mobilization of fractures is limited and not always robust,⁴ hand therapists would agree that early referral and mobilization has a direct effect on final outcome after these injuries.

If immobilization of a hand fracture is requested by the hand surgeon for a high-risk unstable fracture, it should be limited to a period of less than four weeks and preferably three weeks.^{4,25}

All fractures that can be stabilized with a bone-holding forceps can be stabilized by screws, but the bony fragment must be three times the size of the screw head to hold it. Otherwise it will shatter when the screw is inserted and tightened. Periosteal stripping is always held at a minimum, and if at all possible soft tissue interposed between metal and tendons. Positioning of the reduced fracture is checked on I-I and rotation is checked before the first screw hole is drilled. Deviation usually remodels in children, but not rotation, which must be corrected.

Closed versus open

All open fractures should be washed out immediately and given antibiotics. They should, of course, be fixed on the next list.

The main open fracture involving the metacarpal bone is a fight bite, which is when the tooth of the person your patient has punched breaches cartilage of your patient's metacarpal head. If this is not recognized and appropriately washed and treated with antibiotics it will result in septic arthritis and possibly osteomyelitis.

Complications

Complications from fractures can be in the bones itself, such as osteomyelitis, in the fracture itself, such as non-union and mal-union, including rotation and more generalized problems, such as cold intolerance, complex regional pain syndrome (CRPS) and overall persistent swelling and pain which results in a reduced range of movement and ultimately use.

Importantly, a hand fracture can mean the end of an occupation and the beginning of chronic disability. Luckily, most fractures, however, heal well and the patient returns to near pre-injury functional levels.

Osteomyelitis is diagnosed by persistent pain, fever and sometimes swelling. X-Rays only become helpful after osteopenia sets in and therefore MRI is the investigation of choice. If the infection is connected with metal, it must be removed, even if this means the fracture becomes unstable. Temporary external fixators can be the solution in this situation. Any dead bone, such as a sequestrum, must be removed surgically and long-term antibiotics administered. The choice of antibiotics will vary from place to place but usually includes fucidic acid, which penetrates bone especially well.

Blood cultures can sometimes give the actual bacterium causing the infection, but bone biopsies are the most secure way of obtaining appropriate specimens for the microbiologist if the infection is not resolving.

Non-unions needs to be opened again and bone grafted, to bring growth factors and scaffolding material to the site. Mal-unions can either be corrected at the fracture site, but more often a mal-union, say with a rotational element, at the level of the digits, is corrected with a derotational osteotomy of the metacarpal.

The overall chance of getting CRPS after hand trauma is unknown, and the only known factor to reduce the likelihood of getting CRPS postoperatively, is ingestion of large doses of vitamin C for a minimum of six weeks preoperatively. It is, of course, rare that one can predict injury with this accuracy, but it is a useful tool in elective surgery. The earlier CRPS is diagnosed and treated the better the outcome. When suspecting the diagnosis, the patient is issued with an increasing dose of gabapentin or pregabalin, possibly amitriptyline for night time and enough simple analgesia to tidy them over, before they are seen by the expert pain consultant, who can design an individualized treatment plan.

Cold intolerance affects up to 38% of digital fractures²⁶ and can be worse if metalwork is left in. This usually improves after a couple of winters, but can be permanent and is best treated by local heat and warm gloves in winter.

Therapy complications include the following:

- Persistent oedema which influences joint deformities and reduces range of motion of these small joints.
- Joint stiffness which can mostly be prevented by early motion but may occur in some intra-articular fractures despite extensive therapy.
- Intrinsic muscle tightness which often occurs in digit injuries and may take many months to be completely resolved by function and intrinsic stretch exercises.
- Weakness of grip which needs to be assessed and treated with strengthening work once the fracture has healed.
- Reduced function and delayed return to work which can be improved with rehabilitation for most patients.
- Scarring and tendon adhesion which can often be alleviated

by scar management and exercises but sometimes requires tenolysis or removal of any internal fixation.

CRPS is generally prevented by early referral to hand therapy, good early pain relief and extensive advice and education of the patient.

Outcomes

Outcomes can be objective, such as total range of motion or DASH score, or subjective, such as pain analogue score and discussion about functional ability and return to work.

The aim for therapy is to prevent chronic oedema, restore normal range of motion by the time the fracture has healed at 8–10 weeks post these injuries. The patient can then concentrate on increasing strength with the aim of returning to work with full capacity. Many patients will have been able to return to work in some reduced capacity much earlier due to the fact the therapists can make much smaller bespoke splints for them.

Last comments

Close liaison with the hand therapy team is essential as early therapy intervention is crucial to ensure the best outcomes and prevent stiffness.

Surgeons should only intervene if they are certain they can improve stability and fracture configuration or the injury is open. Immobilizing hand fractures post surgery as the surgeon is anxious about stability will lead to poorer outcomes, more stiffness and potential tendon tethering. Gentle early controlled motion 24–36 h post injury/surgery is ideal for the best functional outcomes.

Although hand fractures may be forgiving, soft tissue injuries are not.²⁶ Providing hand splints which protect and rest only the affected digits instead of full plaster casts should limit complications and improve outcomes.

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CHAPTER 61

Osteoarthritis and prosthetic joints in the hand

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Osteoarthritis

Aetiologies

Osteoarthritis of the hand is a multifactorial condition with an interplay between biomechanical and biochemical entities. It is the end stage of a number of processes within the joint. It can be secondary to mechanical derangement of the joint (fracture, dislocation, instability), infection or metabolic processes (secondary osteoarthritis) or occur as a primary idiopathic condition (primary osteoarthritis). The causes of osteoarthritis in the interphalangeal joints, metacarpophalangeal joints and first carpometacarpal joints may be different. Anatomical factors may play a part in the predisposition of women to the development of osteoarthritis in the latter of these.

Primary osteoarthritis is typically a polyarthropathy in the hand, often affecting multiple proximal and distal interphalangeal (PIP and DIP) joints as well as the first carpometacarpal (1st CMC) joint. This appearance in conjunction with the presence of Heberden's or Bouchard's nodes is recognized as nodal generalized osteoarthritis (NGOA) and is predictive of osteoarthritis in other joints. Women are more frequently affected and Heberden's nodes are a particular feature of this condition. Epidemiological studies have indicated that lifestyle factors can contribute to the development of osteoarthritis in the hand. There appears to be a relationship between obesity and hand osteoarthritis.¹⁻³ An association has been demonstrated between osteoarthritis in the hand and shorter time spent in education, low income, marital status and having a child.⁴ The relationship to offspring was stronger in men than women, indicating that it is an effect of environment rather than biology that increases this risk. Genetics also appear to play a role in the development of osteoarthritis in the hand, and may interact with environmental factors. An association has been demonstrated between *COL2A1* gene, vitamin D receptor gene and IL-1 gene cluster polymorphisms, repetitive tasks and hand osteoarthritis (Figure 61.1).⁵⁻⁷

A less common form of osteoarthritis, erosive osteoarthritis, displays a more florid inflammatory component, subchondral

erosive changes and a tendency toward instability and ankylosis. Endocrine causes of erosive osteoarthritis include hypothyroidism and renal dialysis. Hyperparathyroidism may be associated with periarticular erosion, especially of the metacarpophalangeal (MCP) joint, but there is debate as to whether this is associated with the development of osteoarthritis. Haemochromatosis should be considered in patients presenting with polyarticular disease in the hand, especially in those with younger age of onset, males and those with involvement of the MCP joint or wrist.⁸

Joints

Primary osteoarthritis predominantly affects the DIP joints, the CMC joint of the thumb and the PIP joints in descending order of frequency. The MCP joints are rarely affected in primary disease but may be involved in secondary osteoarthritis. The incidence of MCP joint osteoarthritis is equal in men and women, whereas women are more frequently affected by osteoarthritis in the other joints. The presence of osteoarthritis within a joint of a row (DIP, PIP or MCP joints) increases the risk of developing osteoarthritis in another joint within that row. Similarly there is also a risk of developing arthritis within another joint within the same ray.⁹ The association of within-row osteoarthritis is stronger than the within-ray association in all populations studied. Symmetry of symptomatic osteoarthritis in the hands has been reported in a number of studies.

Clinical history and assessment

Pain and stiffness are the typical presenting symptoms of osteoarthritis in the hand. DIP joint involvement is often first noticed as hard swellings either side of the joint known as Heberden's nodes. The appearance of these nodes often precedes the onset of pain, if indeed these joints ever become symptomatic in this way. Heberden himself is quoted as stating 'What are those little hard knobs, about the size of a pea, which are frequently seen upon the fingers, a little below the top, near the joint? . . . they continue for life; and being hardly ever attended with pain, or disposed to become sores, are rather unsightly than inconvenient, though



Figure 61.1 Clinical appearance of osteoarthritis of the hand.

they must be some little hindrance to the free use of the fingers.¹⁰ In a randomly selected population of white women aged 45–65, 26% were found to have clinical signs of osteoarthritis of the DIP joint, namely bony swelling, tenderness or pain on movement, of whom 68% reported the joints to be symptomatic.¹¹ In comparison, only 17% and 16% had signs at the PIP and 1st CMC joints, respectively. In this cohort, bony swelling was found to be the most sensitive finding for the presence of radiographic changes of osteoarthritis at the DIP and PIP joints. Tenderness and pain on movement are the most specific signs and all signs have high negative predictive value at the PIP joint.

In the 1st CMC joint the clinical findings are different. Here deformity and tenderness are the most common and the most sensitive findings. ‘Shouldering’ of the joint, due to subluxation of the metacarpal on the trapezium is often seen. This subluxation may lead to a compensatory hyperextension at the MCP joint. Angular coronal plane deformity of the joints is less common but may be seen. It should be noted that for all these joints the clinical signs have quite low sensitivity in the general population but their sensitivity is increased in patients who report that they are symptomatic.

Patients may present with other sequelae of osteoarthritis, such as mucous cysts. These may result in pain or swelling and may rupture, discharging a clear straw-coloured jelly. Occasionally the presenting complaint may be nail deformity as a result of pressure symptoms on the germinal matrix from an occult cyst.

The American College of Rheumatology (ACR) has defined clinical criteria by which a diagnosis of osteoarthritis can be made in the hand.¹⁰ These are given in Table 61.1. The presence of all of criteria 1, 2 and 3 plus one of 4a or 4b yields a sensitivity of 92% and specificity of 98% for the diagnosis of osteoarthritis in a patient presenting with hand pain.

Investigation

The diagnosis of osteoarthritis can be made on clinical grounds in most cases. The primary role of investigation is to guide treatment and more rarely determine cause. Plain postero-

Table 61.1 American College of Rheumatology Criteria for the diagnosis of osteoarthritis in the hand

Level	Criteria
1	Hand pain, aching or stiffness
2	Hard tissue enlargement of 2 or more of the ring or middle DIP joints, ring or middle PIP joints or 1st CMC joints
3	Fewer than 3 swollen MCP joints
4a	Hard tissue enlargement of 2 or more DIP joints
4b	Deformity of 2 or more of the ring or middle DIP joints, ring or middle PIP joints or 1st CMC joints

DIP, distal interphalangeal; PIP, proximal interphalangeal; CMC, carpometacarpal; MCP, metacarpophalangeal.



Figure 61.2 Radiographic appearance of osteoarthritis of the hand.

anterior (PA) radiographs can be used to confirm the diagnosis (Figure 61.2) and assess severity of the disease using radiographic grading systems. A lateral radiograph of a particular joint may be helpful where treatment is planned. A widely used classification is that of Kellgren and Lawrence, which defines five categories; no radiographic features of osteoarthritis, doubtful changes, minimal, moderate and severe (Table 61.2).¹² They defined the radiological features of osteoarthritis: formation of osteophytes on the joint margins,

Table 61.2 Kellgren and Lawrence radiographic grading of osteoarthritis

Grade 0	None	No features of osteoarthritis
Grade 1	Doubtful	Minute osteophyte, doubtful significance
Grade 2	Minimal	Definite osteophyte, no joint space narrowing
Grade 3	Moderate	Moderate joint space narrowing
Grade 4	Severe	Joint space narrowing with subchondral sclerosis

periarticular ossicles, narrowing of joint cartilage associated with sclerosis of subchondral bone, small pseudocystic areas with sclerotic walls situated in the subchondral bone, and altered shape of the bone ends. However many different interpretations have been placed on the definition of the changes present in each category. This has led to substantial variation in categorization between studies and makes interpretation difficult. Even Kellgren and Lawrence in their original paper recognized that 'personal bias may result in a very different assessment of severity and prevalence'.¹²

The Kellgren and Lawrence classification has been criticized for depending on the presence of osteophytes to make the diagnosis of osteoarthritis. Kallman *et al.* observed that in many cases of osteoarthritis of the hand joint space narrowing and cyst formation can occur without their presence. They proposed a classification that provided a 4-point Likert scale of severity for features such as joint space narrowing and osteophytes, and dichotomous scores for the presence or absence of subchondral sclerosis, subchondral cysts, lateral deformity of at least 15° and collapse of the central joint cortical bone.¹³ This results in a scale from 0 to 10 for from no to severe arthritic changes. They compared their new scoring system with the Kellgren/Lawrence scale and found that both had good reliability and reproducibility for most variables with the exception of the presence of cysts.

For basal thumb arthritis the Eaton Littler classification, refined in the paper by Eaton and Glickel to include a treatment algorithm, is still widely used.^{14,15} This is based on radiographic features of joint space, subluxation of the metacarpal on the trapezium, cysts and sclerosis. This approach has been shown to yield only moderate interobserver agreement, and the treatment indicated fair agreement (Table 61.3).¹⁶

Other imaging modalities such as ultrasound, bone scintigraphy, MRI and CT have little utility in clinical practice due to the relatively low cost and ease of access to plain radiographs. Ultrasound has the advantage that it does not involve the use of radiation, however only superficial pathology is visible. Osteophytes, synovitis and periarticular erosions may be assessed. Ultrasound may more easily determine the presence of active inflammation or erosion than plain radiographs, with the use of power Doppler. MRI is used largely for research purposes. It is possible to visualize all osteoarthritis joint pathology, but overdiagnosis of synovitis may be a pitfall.

Table 61.3 Eaton classification of thumb carpometacarpal joint osteoarthritis

Stage	Radiographic appearance	Possible treatment options
I	Normal or slight joint space widening due to effusion. Possible mild subluxation	Ligament reconstruction or Metacarpal extension osteotomy
II	Joint space narrowing and osteophytes less than 2 mm present	Trapeziectomy ± ligament reconstruction and tendon interposition
III	Joint space narrowing, subchondral sclerosis and osteophytes greater than 2 mm	or Arthrodesis or Arthroplasty
IV	Advanced degenerative changes involving the trapeziometacarpal and scaphotrapezial joint	Trapeziectomy ± ligament reconstruction and tendon interposition

Management principles and operative options

Non-operative

Consideration of the management of any condition must start with an understanding of the natural history, and for musculoskeletal disease the deficits that need to be addressed. For patients with osteoarthritis pain is a predominant feature of the condition. Radiographic progression is minimal over a 10-year period with most progression seen radiographically at the DIP joints.¹⁷

Functional deficits in NGOA may be minimal. The patient may have more difficulty, may employ trick movements and have a reduced grip strength but are able to complete daily activities.¹⁸ Those with erosive osteoarthritis have been reported to have more functional difficulties and more severe pain.

Non-operative treatment modalities include activity modification, splintage and pharmacological intervention with oral or topical anti-inflammatory analgesia, or corticosteroid injection. The use of splints can effectively control symptoms for some patients but their utility is limited by functional restriction. Intra-articular corticosteroid injection has been shown to be an effective treatment, either using X-ray guidance or blind injection. For basal thumb arthritis it has been reported that only one quarter of patients will go on to further intervention within 2 years of injection.¹⁹ The effectiveness of corticosteroid injection and splinting decreases with higher Eaton stages of disease, with most of those that are Eaton stage I and 35% of those Eaton stage II and III achieving sustained relief.²⁰

Specific joints

First carpometacarpal joint

First CMC osteoarthritis is a very common condition, particularly among elderly females. A number of prostheses have been developed to address this problem but comparative studies have not demonstrated an advantage of prosthetic replacement over

simple trapeziectomy with or without tendon interposition. As a result, many surgeons have been reluctant to expose patients to the risks of 1st CMC joint arthroplasty.

Imaeda *et al.* studied the surgical anatomy of the 1st CMC joint and described five major ligaments: the anterior oblique, ulnar collateral, first intermetacarpal, the posterior oblique and dorsal radial ligaments.²¹ Of these, the anterior oblique, also known as the beak ligament, was thought to be the most important for stability of the joint and should be retained for successful joint reconstruction.

More recently, there has been greater interest in the role of the dorsal ligaments to joint stability. Anatomical studies have suggested that in fact there are three robust dorsal ligaments: the dorsal radial, dorsal central and posterior oblique ligaments.²² The double saddle-shaped articular surface is asymmetrical, the convex surface is an ovoid arc (similar in cross-section to an aircraft wing where the radius of curvature decreases from back to front). The opposing concave surface has a small radius of curvature centrally that is maintained on one side of the central ridge but has a gentler slope on the other. Xu *et al.* demonstrated that the joint is incongruent, allowing flexion, extension, adduction and abduction. The contact area is small, particularly in females, which may account for the increased female susceptibility to osteoarthritis of the 1st CMC joint.²³ The forces that the 1st CMC joint must withstand are considerable. Cooney and Chao calculated that a tip pinch of 1 kg will generate 12 kg of joint compression. For power grip the load may be as high as 120 kg.²⁴

The surgical approach to the 1st CMC joint may be dorsal radial or volar. The dorsal radial approach is centred over the 1st CMC joint in line with the first extensor compartment. Care must be taken to identify and protect the branches of the superficial radial nerve as injury may result in painful neuromata. The radial artery should be mobilized and protected, any fine branches being cauterized with diathermy. A distally based T-shaped capsulotomy is performed to expose the joint. The volar approach is through a curvilinear incision on the radial border of the thenar eminence centred over the 1st CMC joint. Cutaneous branches of the median nerve should be protected. The thenar muscles are elevated from the underlying capsule by sharp volarward dissection and the abductor pollicis brevis tendon is mobilized dorsally before a longitudinal capsulotomy to expose the joint is performed. For successful arthroplasty, capsular stability is essential. If there is instability as a result of the previous pathology then ligament reconstruction may also be performed using tendon grafts such as a split flexor carpi radialis tendon graft. At Wrightington the joint is immobilized in a static splint for six weeks with the thumb abducted and the nail plate aligned at 90° to the palm.

Signs and symptoms

Pain at the base of the thumb is the chief presenting complaint of 1st CMC joint osteoarthritis. Swelling and tenderness occur early in the disease and are sensitive indicators of the

presence of osteoarthritis within the joint. Metacarpal subluxation leads to squaring or 'shouldering' of the joint. Osteophytes may be palpable. Use of the grip and grind test should be avoided. Instead a more subtle, and possibly kinder, manoeuvre of reduction and shift may reproduce the symptoms. Hyperextension at the metacarpophalangeal joint may be present and will often worsen with key pinch. Pain on radial and ulnar deviation of the wrist is suggestive of coexistent scaphotrapezial osteoarthritis. Provocative tests for carpal tunnel syndrome should also be performed as the two will coexist in up to 43% of cases.²⁵ Release of the carpal tunnel can be undertaken simultaneous to trapeziectomy either through the same or separate incisions.²⁶ Other causes of radial-sided wrist pain, such as de Quervain's tenosynovitis, should be excluded.

Operative treatment

Eaton stage I

Where the joint surfaces are preserved, non-operative measures described above may be employed but will not prevent progression of the condition. In their original description of stages of CMC joint osteoarthritis, Eaton and Littler proposed a technique of ligament reconstruction that they argued could arrest the progression of symptoms. Their technique was intended to reconstruct the anterior oblique or 'beak' ligament that they felt was the primary restraint to dorsal subluxation of the metacarpal and was deficient in CMC joint osteoarthritis. This used a distally based graft from the flexor carpi radialis (FCR) tendon passed through a drill hole in the base of the thumb metacarpal that runs from the radial surface in the plane of the nail plate, to the volar proximal ulnar corner of the metacarpal. The graft is then brought back beneath the remaining 50% of FCR tendon and sutured to the base of the metacarpal, thereby supporting the joint in orthogonal planes. Numerous other techniques have been described using other local tendon grafts.

Over the past 20 years anatomical and histological dissections have challenged the pre-eminence of the anterior oblique ligament in thumb CMC joint stability. Work by Ladd *et al.* has defined seven consistent ligaments: three dorsal, two volar and two ulnar ligaments.²² They found that the anterior oblique ligament was thin and hypocellular with disorganized collagen fibres. In contrast, the three dorsal ligaments – dorsal radial, dorsal central and posterior oblique ligaments – were thick, highly organized and cellular with profuse neural innervation. Furthermore, it was their impression that these ligaments restrained subluxation of the metacarpal. A procedure to reef the dorsal ligaments has recently been described but with only five cases with follow-up from 8 to 50 months. More data are required before this can be recommended.²⁷

Closing wedge metacarpal extension osteotomy is an alternative to ligament reconstruction for early arthritic changes with good results reported for Eaton stages I, II and III. Wilson described the technique in 1973 of excising a dorsal and radially based 30° wedge from near the base of the metacarpal. This is

then held with a transosseous cerclage wire and percutaneous K-wires. The concept, supported by biomechanical studies, is that by extending the metacarpal the joint mechanics are altered such that the joint is more stable and loads are moved away from areas of damaged cartilage on the volar articular facet to prevent disease progression.²⁸ Hobby *et al.* from Cambridge reported 80% good or excellent results with this technique at a minimum of 2 years for all Eaton stages other than those with stage IV arthritis. The longest follow-up in this series of 41 thumbs was 12 years with satisfactory outcome maintained.²⁹ Their series contained only two patients with stage I osteoarthritis, but Tomaino has reported on a series of 12 patients with stage I osteoarthritis in whom an osteotomy successfully relieved pain in all but one case. He reported an average time to union of 7 weeks.³⁰ Trapezial opening wedge osteotomy of 15° has been shown mimic the effects of metacarpal osteotomy in cadaveric experiments.³¹

Eaton stages II and III

Simple trapeziectomy remains the gold standard treatment against which alternative interventions are compared. CMC fusion can reliably relieve pain, and has the advantage of maintaining pinch strength but at the cost of higher risk of complications, stiffness of the thumb and the potential for degenerative changes in adjacent joints. Synthetic arthroplasty, which may be interpositional, hemiarthroplasty or total joint replacement, can produce excellent short-term outcomes but the complication rate remains high. Where these techniques fail they can be salvaged by excision of the trapezium with equivalent outcomes to primary trapeziectomy,³² however to date there is no evidence that these procedures produce a superior outcome to trapeziectomy with or without interposition or suspension, so the added cost and risk is questionable.^{33,34} Arthroscopic debridement with interposition or distraction arthroplasty have been reported to produce good short-term outcomes.^{35,36}

Isolated trapeziectomy was described in 1949 by Gervis, who reported promising results.³⁷ Concerns emerged, however, regarding reduced pinch strength, proximal migration of the metacarpal and the potential for hyperextension of the MCP joint. A range of soft tissue interposition or suspension procedures were devised to try to counter these issues. These techniques variably use whole or hemi-FCR grafts, abductor pollicis longus or palmaris longus interposition. More recently, suture button techniques have been reported to produce equivalent results, although index metacarpal fracture has been reported with this technique.^{38,39} The ligament reconstruction and tendon interposition (LRTI) procedure gained a deal of popularity and involved passage of 100% of the FCR divided proximally, maintaining the distal attachment, that is then passed through a drill hole in the base of the metacarpal and the remaining tendon interposed and an anchovy within the joint. This construct is augmented with K-wires. Distraction arthroplasty after simple trapeziectomy has been reported to achieve similar outcome to more complicated procedures.⁴⁰

Despite the proliferation of procedures and their apparent widespread acceptance, there remains little evidence of their benefit over simple trapeziectomy. Indeed repeated randomized trials have shown equivalent outcomes in terms of pain relief, grip strength and recurrence.^{41–49} This may be because these studies are not designed to identify problems within subgroups of patients, such as the young or manual workers, in whom reduced pinch strength may pose more of an issue. Alternatively, the problem of ray collapse and degenerative change at the metacarpal scaphoid pseudoarthrosis may be overstated. Although undoubtedly there are some patients who will develop symptomatic degeneration following trapeziectomy, the majority will remain asymptomatic.⁵⁰

Fusion of the trapeziometacarpal joint is favoured by some for patients with high functional demands, such as younger manual workers. These patients are unlikely to tolerate a weak pinch. Achieving union can be challenging, however, and failure to do so is associated with low rates of patient satisfaction and poor pinch strength. Various techniques have been employed, including K-wire fixation, tension band wires, plates and screws, compression staples and headless compression screws. Non-union rates vary widely in cases performed for osteoarthritis and complication rates and the risk of further surgery is higher following arthrodesis than other surgical techniques for management of 1st CMC osteoarthritis.⁵¹ Long-term studies of the outcome of CMC joint fusion in osteoarthritis have demonstrated significant improvement in pinch strength, low pain scores and excellent patient satisfaction.

In the patient with coexistent compensatory hyperextension of the MCP joint some have considered the value of surgical remedy with K-wire fixation, capsulodesis, sesamoid bone fixation or MCP joint fusion if the joint can be passively hyperextended 35° or more.⁵² Although correction can be achieved, this does not appear to improve hand function, and gradual loss of reduction is seen following soft tissue procedures over time.

Eaton stage IV

Patients with pan-trapezial osteoarthritis are not likely to be helped by procedures addressing only the CMC pathology. CMC joint fusion, hemiarthroplasty and total joint arthroplasty will not address the scapho-trapezium-trapezoid (STT) pathology. In these cases, total trapeziectomy with or without interposition, or suspension is the only operative procedure likely to be of benefit. Evaluation of the scapho-trapezoid joint must be undertaken, and if this articulation is also arthritic then hemitrapezoidectomy should be undertaken simultaneously to eliminate this secondary source of radial-sided wrist pain. Concerns have been raised surrounding carpal collapse as a result of trapezial excision and consequent volar STT ligament disruption.⁵³ In one series the incidence of dorsal intercalated segment instability (DISI) deformity was raised from 39% preoperatively to 62% post-trapeziectomy in those with Eaton stage IV osteoarthritis.⁵⁴ These authors reported a correlation between the presence of DISI deformity and lower patient satisfaction.

MCP joints

The MCP joint is a condylar joint with a convex, cam-shaped surface on the metacarpal head and an incongruent (larger radius of curvature) concave surface on the proximal phalanx. This allows 150° of flexion/extension and up to 57° of radio-ulnar deviation in extension, although most activities of daily living are achieved in an arc of 10–70° of flexion.^{55,56} The primary stabilizers of the joint form a sling, whose sides are formed by the collateral ligaments, which originate from the tubercle on the metacarpal head and insert onto the base of the proximal phalanx, the accessory collateral ligaments, which insert into the floor of the sling, and the volar plate, which is mobile at the MCP joint.

Work from the Mayo Clinic in 1984 showed that the collateral ligaments have differing actions at various positions of the joint. The ligaments are, however, at full stretch in MCP joint flexion, thereby preventing abduction and rotation but are relatively slack in full extension in order to allow these movements.⁵⁷ The secondary stabilizers are the musculotendinous units that cross the joint, the long extensors (primary extensors of the MCP joint), the long flexors (primarily act on the PIP and DIP joints) and the intrinsic interosseous and lumbrical muscles, which are the primary flexors of the MCP joints.

The MCP joint is subjected to significant forces, up to 190 N during a pinch manoeuvre and probably more with power grip.⁵⁸ The centre of rotation is unlikely to be through a fixed point on the metacarpal head because of the latter's cam shape. Despite this, most prostheses are designed to incorporate a fixed point of rotation.

The MCP joint is best approached through a dorsal incision, orientated either longitudinally over the MCP joint or transversely if multiple joints are to undergo surgery. It is important to preserve the vascular bundles that run in the gutters between the metacarpals in order to retain venous drainage and prevent swelling. We would advocate a radial paratendinous approach, as the radial sagittal band often requires imbrication to centralize the tendon. The tendon can be elevated from the underlying capsule and a median capsulotomy performed. With anatomical joint replacement, any soft tissue release should be limited and, in particular, care must be taken to retain the attachments of the collateral ligaments.

For the less frequent cases of MCP joint osteoarthritis, motion-preserving surgery is preferred to maintain grip and function of the hand. Silicone and anatomical arthroplasty techniques can result in satisfactory outcomes. Arthrodesis, in the main, is a salvage procedure for painful failed arthroplasty. The position of fusion depends on the digit involved and increases by 5° for each digit from 25° for the index finger through to 40° for the little finger. For all small joint arthrodesis it is our preference to use a 'cup and cone' technique, whereby the articular cartilage is removed with rongeurs to create matching concave and convex surfaces, rather than using an oscillating saw to produce flat surfaces. The most common indication for arthroplasty of the MCP joint is rheumatoid arthritis; in these cases silicone arthroplasty with soft tissue rebalancing is most usually

indicated and does well.^{59,60} In patients with primary and post-traumatic osteoarthritis, it is reported that the silicone implant may not fare as well. Early failure may occur as a result of the high demands placed through the implant in an otherwise normal hand.⁶¹

Proximal interphalangeal joints

The PIP joint is a bicondylar joint with an intercondylar concavity on the proximal phalanx.^{62,63} On either side of the head of the proximal phalanx are pits from which the collateral ligaments originate. On the middle phalanx are two concavities, incongruent with the middle phalanx possessing a larger radius of curvature, separated by a saddle-shaped ridge that dorsally extends into a tubercle for the attachment of the central slip. On the volar margin of the articular surface is a rough, flat area for insertion of the volar plate; this takes origin proximally from the checkrein ligaments attached to the neck of the proximal phalanx. The accessory collateral ligament takes origin close to the true collateral near the pit on the proximal phalanx and inserts into the volar plate.

Movement of the PIP joint varies from 0° to 30° of hyperextension to 100° of flexion. The PIP joint is not a true hinge joint as there is a small amount of rotation and angulation of the coronal plane during flexion; the axis of rotation is centred on a fixed point on the head of the proximal phalanx. Minamikawa *et al.* demonstrated that with a lateral stress, 5° of adduction and 9° of supination is seen at the PIP joint.⁶⁴ In full extension and flexion the joint is more stable.

Enthusiasm for PIP joint arthroplasty has increased as a result of the success of MCP joint replacement. PIP joint fusion can result in acceptable hand function and excellent pain relief. However, where there is concomitant disease of the MCP or DIP joints, fusion of the PIP joint will result in a stiff finger and impaired function, and a motion-retaining arthroplasty may be preferred. Equally, an adequately counselled patient may choose to accept the risk of arthroplasty in order to maintain some movement in the joint rather than opt for a fusion. The only contraindications to arthroplasty in our practice are young active patients, manual workers and patients with significant bone loss, gross instability or previous infection.

Arthroplasty of the PIP joint can be performed under local anaesthetic (if only one joint), regional block or general anaesthesia. Wide-awake surgery gives the advantage that soft tissue balance can be assessed on the operating table through active movement. The joint may be approached through dorsal or volar approaches, or through the ulnar lateral border. The dorsal approach allows for excellent exposure and is of particular benefit where large osteophytes are present. Our preferred technique is to split the extensor hood in the midline, elevating the central slip insertion off the middle phalanx as a continuous sleeve for reattachment with a transosseous suture. Alternatively, a dorsal, distally based chevron Chamay approach may be used.⁶⁵ The central slip is divided in a chevron shape with the base on the middle phalanx. The lateral bands are preserved and

allowed to sublux volarward to allow the joint to be hinged open. Here too, retention of soft tissue attachments is critical to the stability of the prosthesis (Figure 61.3).

In the operation note the surgeon should document the stability of the PIP joint, and whether the joint is suitable for early active mobilization. The finger should be splinted immediately with a volar slab, with the wrist in neutral, the MCP and PIP joints in slight flexion and the hand elevated to reduce swelling. Rehabilitation should begin on day 2, preferably under the supervision of an experienced hand therapist. If suitable, early active mobilization can be commenced with a resting splint to block extension at 20°. If a lateral approach has been used, the collateral repair should be protected with a radial outrigger or by buddy strapping. At six weeks protective splints can be discontinued but a resting night splint should be worn for a minimum of three months.

Osteoarthritis of the PIP joint may present with swelling, stiffness and pain. Isolated stiffness in the absence of pain is not an indication for surgical intervention. Specifically, arthroplasty of the PIP joint is not indicated as the current body of literature indicates that preoperative range of movement governs the postoperative range, with minimal, if any, functional gains. Function is often well preserved but where pain affects daily life intervention is required.

Denervation of the PIP joint under local anaesthesia has been described as a joint-preserving alternative to fusion or replacement with successful relief of pain in 80% of patients.⁶⁶ This technique is not widely used currently. Joint replacement has been available for several decades but has failed to gain widespread acceptance due to high rates of complication and re-operation (approximately 40%). The need to preserve motion is greatest in the little and ring fingers to allow finger flexion for power grip. In the middle, and most especially the index finger, stability to lateral forces such as in key pinch are essential. This may be achieved with resurfacing arthroplasty as long as the radial collateral ligament is not disrupted. In the author's view silicone arthroplasty should not be performed in the index finger

PIP joint as the collaterals will be defunctioned and the digit will not be able to resist valgus forces.

PIP joint arthrodesis can be performed as a primary treatment or for salvage of failed arthroplasty. The benefits of fusion are a stable, painless PIP joint but at the cost of joint motion. The optimum position of fusion depends on the digit, with progressive flexion from 40° at the index finger through to 55° at the little finger PIP joint, however the kinematics of precision pinch will be altered with sacrifice of pinch precision.⁶⁷ Various methods have been described for arthrodesis. Dorsal compression plate fixation and tension band arthrodesis have been reported to achieve 100% union at the PIP joint.^{68,69} Biomechanical analysis supports the use of a tension band wire technique over crossed K-wires or intraosseous wiring because of superior anteroposterior bending and torsional strength.⁷⁰ Headless compression screws have been shown to achieve similar biomechanical strength on three-point bending to tension band wires with 98% successful fusion reported in a series of 51 joints (Figure 61.4).⁷¹

Arthrodesis performed as salvage for arthroplasty is a challenge. Bone loss may be considerable due to implant subsidence or due to difficulty removing the implant. Restoration of digit length is essential to maintain functional length of the flexor and extensor apparatus, otherwise the DIP joint may become floppy. This can be achieved with an intercalary autograft but the risk of non-union is increased. In one series union was achieved in 8 of 13 digits at an average of six months.⁷² Stern was able to demonstrate a 100% fusion rate using an intramedullary Steinmann pin and cerclage wire but with PIP joints fused in full extension functional limitations were reported.⁷³

Distal interphalangeal joint

The painful osteoarthritic DIP joint is most commonly managed with fusion although success with silicone arthroplasty has been reported in small series. The joints are commonly fused in full extension or 10° flexion to allow for precision pinch and grip. Fusion can be achieved with crossed K-wires, bioabsorbable



Figure 61.3 Proximal interphalangeal arthroplasty through a dorsal approach.



Figure 61.4 Fusion of proximal interphalangeal joint with dorsal plates.

poly-l-lactide pins, tension band wires, cerclage wires, headless compression screws, lag screws, intramedullary devices or compression staples with comparable rates of union.^{74,75} Biomechanical studies have indicated superior strength with use of a headless compression screw compared with a tension band wire technique.⁷⁶ However, the complication rate with headless compression screws is high when compared to alternative techniques, with infection rates as high as 15% and soft tissue complications of over 10%.^{77,78} Arthroscopic DIP joint fusion has been described but there are clearly technical and cost considerations for this technique, and the benefit over open surgery has not been proven.⁷⁹

Postoperative management

As with all complex surgery of the hand, the involvement of a hand therapist is strongly recommended. Traditionally, patients have been advised to take oral analgesia and use high elevation to reduce swelling. Where fusion is required non-steroidal anti-inflammatory analgesia should not be taken due to the deleterious effect on bone healing. Recent evidence suggests that high elevation is not effective in limiting swelling in the postoperative phase although it may prevent throbbing pain from the limb.

There is substantial variation in periods of immobilization and rehabilitation regimen between units and depending on the joint involved, the surgery performed and the technique used. The ethos within the Wrightington upper limb unit is to allow protected mobilization as early as possible with splintage where this aids rehabilitation. Dorsal splints are applied to the PIP or DIP joints to protect fusions with K-wire fixation for a minimum of four weeks (Figure 61.5). Where memory staples or headless screws are used, this period can be shortened. Tendon glide is assisted by early active mobilization of the adjacent joints. For small joint arthroplasty mobilization is begun at day 2 as long as

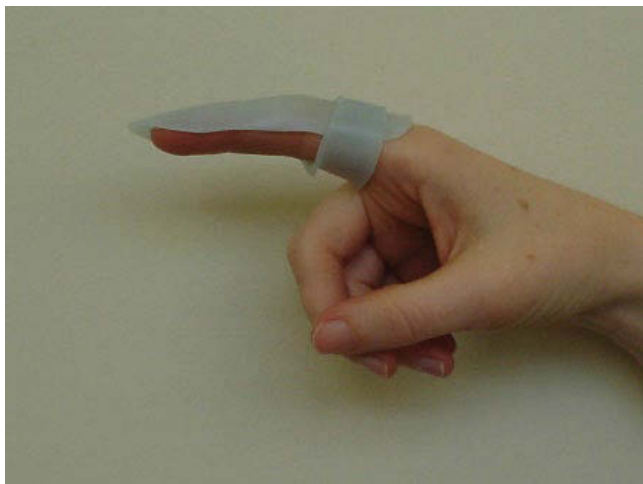


Figure 61.5 Postoperative splint for proximal interphalangeal joint replacement.

the joint is stable and a resting splint is worn between exercises and at night for six weeks. The position of immobilization depends on the operated joint. For the PIP joint the MCP and wrist are at neutral and PIP at 30° flexion. After trapeziectomy with or without ligament reconstruction the period of immobilization varies from six weeks to immediate mobility. Current evidence would suggest that prolonged immobilization does not confer advantage over immediate mobilization.⁸⁰

Complications

Complications of the surgical management of osteoarthritis in the hand depend on the reconstructive technique selected. It is important to consider the effect that surgery to one joint can have on adjacent joints within the ray and also to consider the exquisite balance of the flexor and extensor apparatus, where even minor alterations can lead to dramatic consequences. Iatrogenic swan-neck or boutonnière deformity is common after small joint arthroplasty. Painful neuromata are frequently reported after basilar thumb surgery and can be difficult to treat. They are best avoided by appropriate care being taken at the initial surgery. For refractory cases some success has been seen with excision of the tender skin and a full-thickness skin graft.

Arthrodesis is associated with the unique problem of non-union. The risk of this is greatest at the trapeziometacarpal joint, but wide variation has been reported of between 8 and 20%. In our experience at Wrightington Hospital the rates of union following arthrodesis are equivalent using headless screws or memory staples, but the use of K-wires is associated with higher rates of non-union. The risk of non-union must be balanced against the complications arising from hardware. At the DIP joint fusion is associated with a rate of non-union of just over 10% in osteoarthritis, regardless of technique used. Headless compression screws are associated with more minor complications than crossed K-wires because the metalwork is retained. Implant fracture, prominent metalwork requiring removal, nail growth disturbance and screw cut-out have all been reported.^{81,82} In one series the rate of dorsal skin necrosis was 14% with the use of a Herbert screw.⁷⁸ Salvage of non-union at the DIP joint can be challenging because of retained hardware and loss of bone stock. Owusu and Isaacs have described a technique using a corticocancellous iliac crest bone plug inset to the middle and distal phalanges, having removed the hardware through a dorsal wedge corticotomy of the middle phalanx, but more data is required before this technique can be recommended.⁸³

Arthroplasty of the small joints of the hand is most commonly complicated by joint stiffness. In few cases a specific cause is discernible, such as heterotopic ossification, and re-operation in these cases rarely improves the arc of movement. Recognition that, on current evidence, preoperative arc of movement will dictate the postoperative outcome may reduce the rate of re-operation in a vain attempt to improve movement. Where tendon imbalance results in swan-neck or boutonnière deformity



Figure 61.6 Dislocated proximal interphalangeal joint replacement.

there may be value in attempting rebalancing surgery in some cases, as long as mobility has been retained. If a fixed deformity exists, re-operation is unlikely to be successful. Dislocation and implant loosening (Figure 61.6), squeaking due to friction and both implant fracture and peri-prosthetic fracture have been reported, resulting in overall complication rates of around 30% for small joint arthroplasty.⁸⁴ For silicone implants there appears to be a linear relationship between time since implantation and implant fracture, although this does not appear to translate to surgical revision.⁵⁹

Outcomes

For early stage 1st CMC joint arthritis (stage I or II) surgery can produce good outcomes in over 80% of patients with either a metacarpal osteotomy or ligament reconstruction. Longitudinal

studies have indicated that successful early intervention can prevent progression of radiographic changes.^{85,86} Where osteoarthritic changes are more advanced, satisfactory long-term pain relief can be achieved with trapeziectomy with or without suspension, prosthetic replacement or fusion with no clear superiority in outcome with any procedure, although complication rates will vary.

For the PIP joint arthroplasty with either a silicone spacer or metalloplastic or pyrocarbon anatomical prosthesis can maintain joint motion and provide satisfactory pain relief. The long-term outcome of anatomical replacement is not known but medium-term results indicate favourable outcomes but with high rates of re-operation. Arthrodesis may produce a more durable outcome at the expense of joint motion and the ideal indication for each is yet to be determined. Joint fusion remains the favoured treatment at the DIP joint although the best method of achieving union is debated.

Arthroplasty

Classification of implants

Small joint arthroplasty is performed in the hand to reconstruct MCP joints, PIP joints, the CMC joint of the thumb and less frequently the DIP joint. A number of implants are available and these can be classified variously depending on location, materials, design, constraint or fixation. We prefer to categorize the implants into three main groups: interposition implants, ball and socket joints and two-part anatomical implants. Metallic hinge designs have been trialled but their use has been discontinued due to early implant loosening.⁸⁷

Interposition implants are the earliest and arguably the most successful group of arthroplasty designs in the hand. Silicone implants were popularized by Al Swanson in the late 1960s, primarily for use in inflammatory arthropathy.⁸⁸ These and all subsequent designs for the MCP and PIP joints have been of a flexible hinge design. Although they are designed as a hinge, many authors consider them as flexible internal splints or spacers that augment a soft tissue balancing procedure. For arthritis of the base of the thumb, a trapezium spacer was conceived by Swanson with a silicone stem into the thumb metacarpal (Figure 61.7).⁸⁹ Latterly, other materials have been employed to circumvent issues of silicone fragmentation, especially at the base of the thumb, including a number of prostheses made from pyrolytic carbon.

Ball and socket joints have been employed most successfully at the CMC joint, with a number of implants of this type in use. These designs do not attempt to replicate normal joint anatomy. The implants can be designed to be implanted uncemented or with polymethylmethacrylate (PMMA) cement. This ball and socket concept has been trialled in the MCP joint but with high rates of early failure.⁹⁰

Two-part anatomical implants are designed to restore as closely as possible the normal anatomy of the joint with the aim



Figure 61.7 Silicone arthroplasty first carpometacarpal joint.

of returning normal kinematics. This implant group necessitates retention of soft tissue stabilizing elements and restoration of soft tissue balance. These implants have bearing surfaces of metal on ultra-high molecular weight polyethylene (UHMWPE) or pyrocarbon contoured to mimic the anatomical joint surface. The implants have stems that are fixed by a variety of methods, including osseointegration, cementation and press-fit.

Evolution

Early attempts to address painful arthritic joints in the hand involved excision arthroplasty that was later augmented with interposed soft tissues. At the base of the thumb excision arthroplasty remains the 'gold standard' against which the success of alternative interventions must be judged.³³ On the back of the success of hip arthroplasty there was a wave of enthusiasm for

metalloplastic articulations and a number of ball and socket designs were introduced for MCP joint and even the PIP joint. Early failure occurred due to implant loosening and these were abandoned.

Then came silicone interposition arthroplasty. The concept was to provide a flexible internal splint with interposition and encapsulation. In patients with inflammatory arthropathy, poly-articular involvement and low functional demands, silicone arthroplasty was very successful, especially in reconstruction of the MCP joints, and remains a useful tool in the hand surgeon's armament. Of the silicone implants, those of Swanson became the most widely used. Expansion of the indication for arthroplasty to those with post-traumatic or primary osteoarthritis resulted in a perception of less favourable outcomes and alternative designs were sought.

Two-part anatomical prostheses were thought to have advantages, especially in high-demand individuals. It was proposed that anatomical designs would restore joint kinematics and improve outcomes. The lack of constraint can minimize the risk of implant loosening as seen in early hinge designs, or of implant fracture observed in silicone arthroplasty, however, new risks present, with joint instability, angulation and dislocation all commonplace.

Outcomes

Reported outcomes: first carpometacarpal joint

The unique anatomy of the 1st CMC joint has resulted in significant challenges for arthroplasty design. Ball and socket designs have shown mixed results with high rates of loosening of the 'cup' within the trapezium, although satisfactory long-term pain relief and function have been reported.^{91–94} When combined with an STT arthrodesis for treatment of pan-trapezial osteoarthritis, loosening was observed in most cases.⁹⁵ Rates of failure have been reported to be higher in men of working age and this has been proposed to be a relative contraindication for this type of arthroplasty.⁹⁴ Cemented implant designs appear more successful for basal thumb arthritis than uncemented equivalents, with high rates of early loosening observed with the Ledoux, Moje and Elektra uncemented ball and socket designs although, as with the cemented arthroplasties, this does not always lead to revision or have a detrimental effect on the outcome.^{96–102}

As with other joints in the hand, the initial experience of 1st CMC joint arthroplasty was with silicone implants. Silicone arthroplasty, despite early promising reports, has been shown to be associated with high rates of joint subluxation, implant fragmentation and silicone synovitis in osteoarthritic patients at 15 years follow-up.^{103–109} Comparative studies have not shown significant benefits of silicone replacement over trapeziectomy with or without interposition, although pinch strength at 1 year may be superior with the silicone implant.^{106,110,111} A systematic Cochrane review identified no benefits of arthroplasty over simple trapeziectomy, with the latter producing fewer complications.¹¹²

The SR trapezometacarpal prosthesis (Avanta Orthopaedics, CA, USA) was the first anatomical design, with biconcave cobalt–chrome on UHMWPE-bearing stemmed implants. Initial results reported by the originators were encouraging and were supported by other authors and impressive kinematic studies.^{113–116} However, an independent study by Perez-Ubeda *et al.* reported 70% failure in 20 implants followed for between 24 and 45 months, with loosening in 55% and ankylosis in 15%.¹¹⁷ In 1982 Braun reported the initially favourable results of a titanium metacarpal component and a polyethylene trapezium component, both cemented with PMMA cement.¹¹⁸ Good outcomes were also reported at a mean of 59 months with only one patient complaining of minimal pain, and one revision for post-traumatic loosening.¹¹⁹

These problems with implant loosening within the trapezium have led to the development of a number of hemiarthroplasty designs. A pyrocarbon resurfacing hemiarthroplasty prosthesis for the base of the first metacarpal has been developed (Ascension Orthopedics, Austin, TX, USA) (Figure 61.8). At Wrightington, a review of the first 19 patients to undergo pyrocarbon hemiarthroplasty with a minimum follow-up of 1 year presented at the British Hand Society¹²⁰ indicated an improvement in pain scores but no change in function or grip strength. However, 4 of 19 joints subluxated, a finding observed

in other series.¹²¹ The authors conclude that the implant may have a role in the young patient who wishes to retain strength. More recently, a modular metal uncemented hemiarthroplasty with titanium plasma spray stem has been introduced. It is proposed that modularity will allow better soft tissue balancing and use of head sizes that better fit the anatomy. Furthermore, the authors suggest that the varus position of the head of the implant more closely replicates normal anatomy than other designs. Initial results from the originator indicate 94% implant survival at 72 months and high patient satisfaction.¹²²

Interposition arthroplasty at the base of the thumb with pyrocarbon implants has been studied using a number of forms including spheres, ellipsoids and tori or ‘doughnuts’ that are inserted by reaming the trapezium to create a cup. Despite high rates of patient-reported satisfaction, spherical prostheses have been reported to subside into the bone in almost all cases, causing pain, weakness and stiffness.^{123,124} Ellipsoid-shaped implants have also struggled to demonstrate benefits over trapeziectomy, with high rates of dislocation or subsidence.³⁴ The use of other materials has been no more successful. Artelon TMC spacer (Artimplant AB, Sweden) is a biocompatible degradable polyurethaneurea ‘T’-shaped device designed to stabilize the joint and interpose between the joint surfaces.¹²⁵ Exuberant granulomatous foreign body giant cell reactions have been demonstrated in relation to the use of this implant; these have resolved with implant removal.^{126–128}

Reported outcomes: metacarpophalangeal joint arthroplasty

The predominant cause of MCP joint destruction is rheumatoid arthritis. Consequently much of the literature concerning arthroplasty at this joint concerns this patient group. Silicone arthroplasty has been shown to produce reliable outcomes in the MCP joint in inflammatory conditions. In osteoarthritis there is a scarcity of evidence. A series of 15 silicone MCP joint replacements in 14 osteoarthritic patients at an average of 40 months reported good or excellent outcomes in 12 patients. One implant had been revised for fracture but of the remaining implants none were reported to show signs of fracture or joint subluxation or malalignment.¹²⁹

Osseointegrated titanium implants followed from the use of osseointegration in dental surgery. Titanium-stemmed implants have been shown to osseointegrate when implanted at the MCP and PIP joints. At the MCP joint early reports using this concept with a silicone spacer indicate a high rate of failure of the joint mechanism, although pain relief and high patient satisfaction have been reported.^{130,131}

The first unconstrained ‘anatomical’ implant was made from alumina ceramic with favourable outcomes, but the authors felt that the design could be improved by moving the centre of rotation more volarward.^{90,132–134} Other designs now use pyrocarbons and other composite materials. Pyrocarbon is a synthetic material with a high-strength graphite core coated with a pyrolytic carbon layer formed by heating a hydrocarbon



Figure 61.8 Pyrocarbon first carpometacarpal joint. Hemiarthroplasty.

gas to approximately 1300°C. Pyrocarbon was first used in mechanical heart valves, is inert and compatible with normal bone. It was initially thought that osseointegration would occur, but this has been shown not to be the case.¹³⁵ In 1999, Cook *et al.* reported their experience in 53 predominantly rheumatoid patients with 151 anatomical unconstrained MCP joint implants made from pyrocarbon.¹³⁶ The authors reported a revision rate of 12% with a 10-year survival rate of 81.4%. The short-term outcome of a pyrolytic carbon implant in patients with osteoarthritis has also been shown to be good.^{137,138} Medium-term follow-up at 2–4 years indicates that promising early results are maintained with high rates of patient satisfaction and low pain scores.¹³⁹ No problems of implant migration, fracture or dislocation were observed.

Harris and Dias have reported their results using an anatomical implant with cobalt–chrome on an UHMWPE bearing.¹⁴⁰ The proximal cobalt–chrome component is inserted into an UHMWPE press-fit sleeve that is itself inserted into the metacarpal with an interference fit. The authors followed up 13 joints for a mean of 5 years; there was one revision for infection, and evidence of loosening of two phalangeal and one metacarpal component.

MCP joint arthroplasty with either silicone or resurfacing implants appears to reliably relieve pain and maintain function. Subluxation or dislocations of the unconstrained components, loosening of the implants, implant fracture and joint stiffness have all been described.^{136,141}

Reported outcomes: proximal interphalangeal joint arthroplasty

As in other joints, constrained or semi-constrained implants have not performed well in the PIP joint. The LPM prosthesis, a semi-constrained ceramic-coated cobalt–chrome implant, was reported to undergo high rates of early failure.¹⁴² Silicone arthroplasty can reliably relieve pain in the short term, but complications of angular deviation have been observed in a series of patients with osteoarthritis in whom the implant was inserted through a lateral approach.¹⁴³ The risk is greatest in the radial digits due to lateral forces during pinch.

Anatomical replacement is less constrained. The first anatomical resurfacing PIP joint was developed by Linscheid using stemmed unconstrained implants with a bearing of UHMWPE on cobalt–chrome.¹⁴⁴ The stems were fixed with PMMA cement. Linscheid *et al.* reported the results of 66 of their implants in 47 patients, 56% of with primary osteoarthritis, 24% with post-traumatic arthritis and the remainder with inflammatory arthropathy, with a minimum follow-up of 1 year.¹¹³ Using their own criteria they reported 32 good, 19 fair and 15 poor outcomes. The outcomes were worst in digits with pre-existing deformity or extreme bone and/or soft tissue loss. A more recent cohort reported excellent long-term results in 18 of 20 patients with predominantly primary or post-traumatic osteoarthritis.¹⁴⁵ Importantly, Linscheid's group has shown that in the long term, pain scores remain low and there is no substantial change in

range of motion. They observed a higher failure rate for those implanted via a volar approach.¹⁴⁶

Moller *et al.* reported a series of 22 osseointegrated titanium PIP joint replacements with a silicone hinge between the stems.¹⁴⁷ They reported initial good outcomes with 41 of 44 stems showing osseointegration but high rates of silicone failure with deformity or fracture in 45% at a minimum follow-up of 12 months. They concluded that the silicone component would need further development.

The outcomes of uncemented anatomical pyrocarbon PIP joint arthroplasty have been reported in a number of studies.^{148–154} At Wrightington, we have undertaken the largest study to date of 97 joints in 72 patients, 60% of whom had primary osteoarthritis, with a minimum follow-up of 2 years. The results indicate good implant survival rates of 85% at 5 years. Although arthroplasty resulted in good relief of pain, there was no significant increase in the arc of motion of the digit from preoperatively. The main concern with the pyrocarbon implants is at the bone–implant interface. Osseointegration does not occur and, in many cases, a radiolucent line may be seen around the implant.¹⁴⁸ To date this worrisome lucency has not translated into large numbers of implant failures but it does give cause for concern. A comparative study of pyrocarbon arthroplasty against silicone arthroplasty showed equivalent outcome in all parameters except stability in the coronal plane, which was better in the pyrocarbon group.¹⁵¹ A prospective multicentre randomized trial of titanium on polyethylene, pyrocarbon and silicone implants reported equivalent significant reductions in pain scores but revision rates as high as 39% in the resurfacing implants, compared to only 11% in the silicone group at 3 years post implantation.

The PIPr (MatOrtho Ltd) has a mobile UHMWPE-bearing distal component articulating with a cobalt–chrome proximal component; both components have a hydroxyapatite-coated cobalt–chrome uncemented stem (Figure 61.9). The 1-year results with this prosthesis in 43 digits have been reported from Wrightington with favourable results; pain scores and range of



Figure 61.9 PIPr arthroplasty design.

movement improved although two revisions occurred at six months and 2 years and three patients developed a swan-neck deformity.¹⁵⁵ Ongoing analysis continues.

Taken together, the literature suggests that PIP joint arthroplasty using silicone or anatomical arthroplasty can result in high patient satisfaction and pain relief but that gains in arc of motion have been disappointing. Most series have used a dorsal approach and the disruption caused to the extensor mechanism may influence the outcome. Results of small series using a volar approach to the PIP joint have reported substantial improvements in the arc of motion that warrants further investigation, however reports of higher rates of failure using this approach cause concern.^{146,156}

As with all implants, infection is a major concern after PIP joint arthroplasty. From our own data we estimate the risk at approximately 1%. This is best treated by excision and secondary reconstruction with a silastic implant, or volar plate arthroplasty. With unconstrained implants, instability is a problem that is best avoided with careful soft tissue balance and accurate implant alignment at the primary procedure. Early dislocations may be addressed with closed reduction and splintage. If this management fails then revision to a hinged silicone implant is likely to provide a stable, pain-free joint. Implant loosening, a common problem with highly constrained arthroplasty, is also seen with unconstrained components. Revision with larger components and bone grafting may be successful, otherwise a silicone hinged implant can be used.

Reported outcomes: distal interphalangeal joint arthroplasty

At the DIP joint arthrodesis forms the mainstay of treatment of painful osteoarthritis. Small series of flexible silicone implant arthroplasty have been reported. These indicate durable results at an average of 5 years with good pain relief and mean motion of 33°.^{157–159}

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CHAPTER 62

Wrist pathology

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Physical exam of the wrist

For many wrist pathologies, a good physical exam is more helpful in determining the diagnosis than expensive imaging studies. However, it is imperative to have a thorough knowledge of wrist anatomy in order to correlate anatomical knowledge with specific pathologies. When performing a physical exam of the wrist it is helpful to approach the exam in a systematic way and compartmentalize the wrist based on anatomic regions. There are several physical exam manoeuvres and tests that can be performed to help in the diagnosis of specific wrist conditions. An anatomically based approach to wrist palpation will be presented followed by a discussion of common wrist problems that present to hand surgeons.

Radial-sided wrist pain

Palpation of the wrist should not be a neglected or generalized part of the exam. Palpation of the wrist should be very specific and correlate with anatomy. When examining the radial side of the wrist, pain on palpation along the tendons of the first dorsal compartment (abductor pollicis longus and extensor pollicis brevis) as they exit at the level of the radial styloid is indicative of de Quervain's tenosynovitis. A hypertrophied tendon sheath is also a common finding. Palpating just distal to the radial styloid in the anatomic snuff box can elicit pain from a scaphoid fracture. The distal pole of the scaphoid can also be palpated immediately volar to the snuff box at the base of the thenar eminence. Continuing the exam by palpating distal to the snuff box will bring the examiner to the scaphoid, trapezium and trapezoid (STT) joint and the carpometacarpal (CMC) joint. Palpation of this area can elicit pain from common arthritic pathologies such as CMC arthritis.

Ulnar-sided wrist pain

Ulnar-sided wrist exam examination should start with palpation of the ulnar head and ulnar styloid. Pain along the ulnar styloid can be indicative of triangular fibrocartilage complex

(TFCC) pain or a distal ulna fracture. Palpation of the soft spot on the ulnar side of the wrist just distal to the distal ulna will elicit pain from the TFCC and can indicate pathology in this area. Immediately dorsal to the ulnar soft spot is the extensor carpi ulnaris (ECU) tendon, which is a frequent site of ulnar-sided tendonopathies. Palpation of the ECU tendon and sixth dorsal compartment sheath, especially in ulnar deviation, will help in diagnosing ECU pathologies. Volar-sided palpation can elicit pain from a fractured pisiform or hook of the hamate, as well as piso-triquetral arthritis.

Central wrist pain

Palpation of the wrist more central or midline is often less specific but still very helpful. The wrist should be palpated along the dorsal aspect and examination for pain at the lunate or scapholunate interval can be indicative of scapholunate (SL) ligament tear or Kienböck's disease. Palpation just proximal to the carpal bone can elicit pain from the distal radioulnar (DRU) joint.

Kienböck's disease

Pest first described avascular necrosis of the lunate based on cadaveric dissections. Avascular necrosis of the lunate is named after the radiologist Robert Kienböck.¹ Although he is best remembered for his paper 'Concerning traumatic malacia of the lunate and its consequences', he published over 250 scientific papers, an eight-volume edition on radiology and was the first to develop a film dosimeter to measure exposure to radiation.² Despite Kienböck's disease being described over 100 years ago, the aetiology and natural history are not clearly understood.

Kienböck's disease presents in patients as wrist pain, usually located centrally over the dorsum of the wrist. The pain usually affects one wrist and is rarely bilateral. Varying degrees of pain are present, with many patients going years before they seek medical attention. Pain is usually present with wrist motion but

as the disease progresses, pain at rest is common. It is commonly seen in men 20–40 years of age.

Traditionally, it was thought that fractures of the lunate would lead to Kienböck's disease because of its poor blood supply, but there is no clear consensus on the relationship. Long-term follow-up studies have shown that that lunate fractures do not reliably develop avascular necrosis, even with an ulnar negative variance.³ Ulnar negative variance has been associated with Kienböck's disease, prompting many to point to this anatomical feature as a possible aetiology. Hulten's classic study in 1928 showed an association between ulnar negative variance and Kienböck's disease but more recent studies do not demonstrate a clear correlation.^{4,5}

Clinical evaluation

The clinical evaluation of the patient with Kienböck's disease starts with a thorough physical exam of the wrist. However, the exam findings for Kienböck's disease are usually not diagnostic because they are non-specific. Dorsal wrist pain, often with motion or activity, is the main physical exam finding. The differential diagnosis will include dorsal wrist ganglion, scapholunate ligament injuries, DRU joint pathology or ulnar impaction syndrome just to name a few.

Radiographic evaluation is followed by the physical exam. Kienböck's can be diagnosed by correlating the physical exam with radiographs, however imaging using MRI is often necessary for definitive diagnosis (Figure 62.1). Radiographs are evaluated

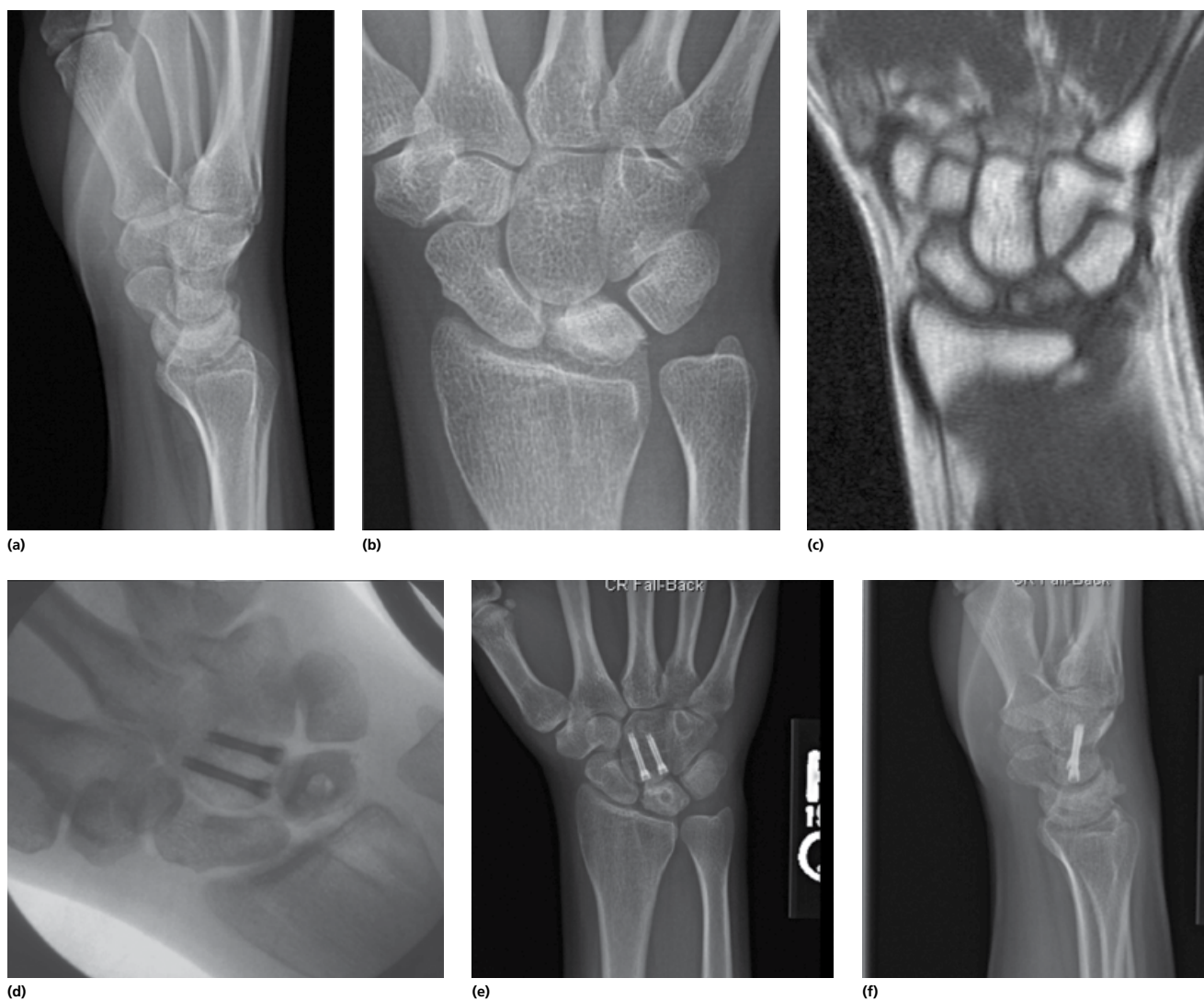


Figure 62.1 (a, b) Lateral and postero-anterior views of a wrist with sclerosis of the lunate and mild collapse of the lunate without general carpal collapse. Also note the ulnar positive variance. (c) MRI showing T1 with low signal intensity of the lunate and fracture line through the lunate. (d) Intraoperative X-ray showing capitatal shortening osteotomy and revascularization of the lunate using capsular-based vessels from the fourth extensor compartment as described by Sotereanos *et al.*¹³ (e, f) Three months postoperative radiographs showing revascularization of the lunate, no further collapse, and patient returning to pain-free wrist motion.

for sclerosis of the lunate and ulnar variance should be noted. The lack of lunate sclerosis does not rule out the disease. If radiographs are not diagnostic, then an MRI should be considered.

Kienböck's staging and treatment

Radiographs and MRI are used to stage Kienböck's disease. The staging system as presented by Lichtman and associates is the most popular and helps guide treatment and determine prognosis.^{6,7} The treatment of Kienböck's disease is based on the staging and will be discussed together.

Stage I

Symptoms of stage I Kienböck's disease are usually non-specific and mimic wrist sprains or synovitis. Radiographs are usually normal in stage I Kienböck's and represent a precollapse and early appearance of the disease. MRI will demonstrate low signal on T1 and T2 weighted images. It is important to recognize if there is low signal of the entire lunate. If there is only focalized low signal on T1 weighted images then other diagnoses, such as ulnar abutment, fractures, osteoid osteoma or enchondromas, become much more likely. If T2 weighted images show increased signal, this can be a sign of revascularization and MRI can be used to monitor resolution of disease.

Stage I disease is best treated non-operatively with a splint. Most people advocate for at least three months of splint use. Some authors advocate for night-time splinting; nevertheless, non-operative management is the mainstay of treatment.⁸

Stage II

Radiographs become diagnostic or at least suggestive of Kienböck's disease during stage II. One typically finds increased sclerosis of the lunate with possible fracture lines or early collapse. MRI continues to be diagnostic with the entire lunate showing low T1 and T2 signal intensity. Lunate height is maintained and symptoms usually mimic chronic wrist synovitis.

Stage IIIA

Stages II and IIIA are usually discussed together because the treatment options are the same. Stage IIIA Kienböck's disease demonstrates collapse of the lunate; however, there is maintenance of carpal height and no signs of arthritis. The anterior-posterior distance of the lunate is usually increased on the lateral radiograph. Sclerosis is present and MRI findings of low signal intensity on T1 and T2 weighted images remain consistent.

The treatment of stage II and stage IIIA disease should start with initiating supportive care and immobilization. A surgeon should not rush to surgery during this stage of treatment and a course of non-operative treatment should be initiated. The severity of the patient's symptoms should dictate whether to proceed with surgical treatment or continue with non-operative treatment. The surgical treatment options differ based on ulnar variance.

Stage II and IIIA with ulnar negative variance

When ulnar variance is noted to be negative, it is thought that increased pressure from the lunate fossa of the distal radius is the cause of the symptoms. Unloading and decreasing the pressure on the lunate through a radial shortening osteotomy is usually the treatment of choice. One study demonstrated 93% of patients had diminished pain after a radial shortening osteotomy.⁹ Ulnar lengthening osteotomy has been described for stage II and IIIA disease with ulnar negative variance but this is generally considered a more difficult procedure with higher complications. Ulnar lengthening requires bone grafting and healing at two osteotomy sites, both the proximal and distal ends.

Stage II and IIIA with ulnar positive or neutral variance

Revascularization procedures are most appropriate in stage II and IIIA with ulnar positive or neutral variance. A number of revascularization procedures have been described, including vascularized pedicle transfer from the distal radius, vascularized pisiform transfer, vascularized bone graft from the metacarpal and free vascularized grafts from the iliac crest or medial femoral condyle.¹⁰ The most popular vascularized pedicle transfer is from the distal radius and is the 4,5 extracompartmental artery.^{11,12} Sotereanos *et al.* have described a capsular-based vascularized bone graft (see Figure 62.1d) based on the fourth extensor compartment artery for the treatment of scaphoid avascular necrosis, which has been adapted for the treatment of stage II and IIIA Kienböck's disease because of its surgical simplicity.¹³ These procedures may be accompanied by joint levelling procedures such as capitate shortening, radius osteotomy or external fixation.

Stage IIIB

Stage IIIB is characterized by lunate collapse, rotation of the scaphoid and loss of carpal height. Revascularization procedures are usually not considered after lunate collapse and loss of carpal height because of the destruction of normal anatomic architectures. A good result from a revascularization procedure would not be expected. There is some controversy about the best way to treat stage IIIB disease and some authors have had good results with limited wrist arthrodesis, including scapho-capitate arthrodesis and scaphotrapezium-trapezoid arthrodesis. Although controversial, other authors have had good results with radial shortening osteotomies.¹⁴ Lunate excision with interposition arthroplasty or proximal row carpectomy are additional salvage procedures that have been described.

Stage IV

Stage IV is characterized by collapse of the lunate and degenerative changes in the radiocarpal and midcarpal joints. Joint levelling or revascularization procedures are not performed once degenerative changes appear. Many patients can be managed non-operatively with supportive care such as splints, anti-inflammatory medications and activity modification. In patients who have failed non-operative management, salvage procedures

can be considered such as limited or total wrist arthrodesis or proximal row carpectomy. For a proximal row carpectomy to reliably provide pain relief, the proximal capitate must be free from degenerative changes since it will eventually articulate with the lunate fossa of the distal radius.

De Quervain's tenosynovitis

De Quervain's tenosynovitis causes pain on the radial side of the wrist, centred over the first dorsal compartment. Active radial deviation of the wrist (such as a mother picking up a small child) or passive ulnar deviation causes severe pain over the tendons of the first dorsal compartment. The first dorsal compartment tendon sheath can be up to five times its normal size due to fibrous tissue deposition. De Quervain's tenosynovitis is a myxoid degeneration of the first dorsal compartment. Although non-steroidal anti-inflammatory medications are a first line treatment, it is not an acute inflammatory process. Often the tendon sheath can be easily palpated due to its enlarged size.

Intersection syndrome should be ruled out during the diagnosis of de Quervain's tenosynovitis. Intersection syndrome causes pain in a similar area but there are subtle differences. It is caused when pain, swelling and sometimes crepitus occur because the outcropping muscle bellies of the abductor pollicis longus and extensor pollicis brevis intersect with the long wrist extensors on the dorsum of the forearm.

Diagnosis of de Quervain's tenosynovitis is usually done with a patient history and physical exam. Imaging studies are usually not required. Pain over the first dorsal compartment on the radial aspect of the wrist, which is made worse with palpation, is the first physical exam findings. A thickened tendon sheath and pain with active radial deviation are also common findings. Finkelstein's manoeuvre (Figure 62.2) is the classic physical exam manoeuvre used to aid in the diagnosis.

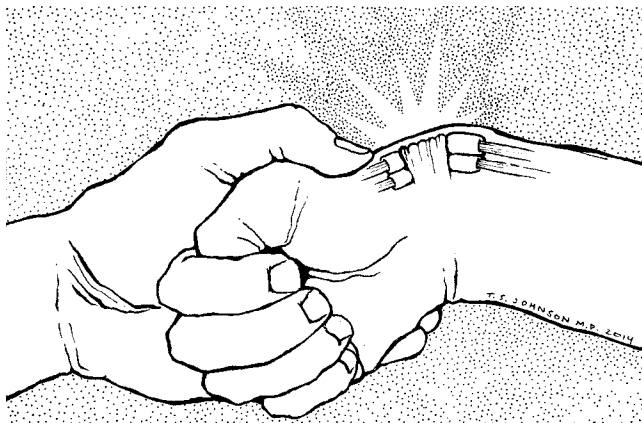


Figure 62.2 Finkelstein's manoeuvre. Pain is generated on the radial aspect of the wrist over the first dorsal compartment when the patient clasps his or her thumb into the palm and the examiner pulls the wrist into ulnar deviation.

Initiation of non-surgical treatments such as watchful waiting, splinting, non-steroidal anti-inflammatory medication and steroid injections into the second dorsal compartment should be considered. Multiple studies have shown successful treatment with each of these non-surgical techniques. The authors recommend non-surgical treatment be initiated upon diagnosis. We recommend a removeable thumb spica splint and non-steroidal anti-inflammatory medications. Other non-operative modalities include rest, ice, activity modification, iontophoresis and corticosteroid injections. Cases that are refractory to non-surgical management should be considered for surgical release.

Surgical release of the first dorsal compartment is very successful and produces reliable, long-term pain relief. Open surgery through a small horizontal incision is the authors' preferred method of treatment. One study showed complete pain relief after surgical release in 19 of 20 patients.¹⁵ Branches of the radial sensory nerve are in or near the operative site and should be carefully identified and/or avoided. If a branch of the radial sensory nerve is cut during surgery, a painful neuroma can result. Endoscopic methods of treatment have also been described and are successful.

Scaphoid fractures

The scaphoid is most commonly injured by a fall on an outstretched hand, resulting in radial-sided wrist pain and, classically, snuff box tenderness. Scaphoid fractures represent 60% of all carpal bone fractures. Because of the distally based scaphoid blood supply, fractures are notoriously difficult to treat with a high incidence of delayed union, non-union and avascular necrosis. Eighty per cent of the scaphoid is covered in articular cartilage, which limits its blood supply. The majority of the blood supply to the scaphoid comes from the dorsal scaphoid branches of the radial artery, with a minority of the blood supply from the volar scaphoid branches. These vessels enter the scaphoid relatively distally, resulting in purely intraosseous perfusion of the proximal pole. The tenuous blood supply of the proximal pole explains the reported avascular necrosis that occurs in 13–50% of scaphoid fractures. These percentages increase for proximal pole fractures. Early diagnosis and treatment is important since scaphoid fractures that are left untreated have a higher incidence of non-union and malunion. Scaphoid non-union is difficult to treat and if left untreated will progress to carpal collapse and degenerative arthritis, specifically, scaphoid non-union advanced collapse (SNAC).

Diagnosis of scaphoid fractures can be surprisingly difficult. Diagnosis begins with a good history and physical exam. Patients typically have pain on the radial side of the wrist, specifically in the anatomic snuff box, decreased range of wrist motion, decreased grip strength, and pain with thumb motion. Adequate radiographs, including postero-anterior (PA), lateral, pronated oblique, supinated oblique and a PA with ulnar deviation (scaphoid view), are essential. However, X-rays often

show no evidence of fracture. When there are signs of scaphoid fracture on history and physical exam but no radiographic evidence, the traditional algorithm would be to cast the patient in a short arm thumb spica cast and repeat the X-rays in two weeks. After two weeks there is usually sufficient resorption at the fracture site to visualize a scaphoid fracture on radiographs. However, following this algorithm will overtreat and subject many patients who do not actually have a scaphoid fracture to two weeks of casting, which can be costly in terms of loss of work and productivity. Advanced imaging studies, such as MRI and CT scans, can help in the diagnosis and in the surgical decision making. MRI is the preferred test if the diagnosis of a scaphoid fracture is in question. The sensitivity and specificity of diagnosing a fracture is 90–100% with MRI. CT scans show bone structure better than MRI and are therefore better in determining displacement of a fracture. However, a CT scan should be used with caution in diagnosing an occult scaphoid fracture because there is a high rate of overdiagnosis with CT (false-positives), most likely due to normal vascular foramina appearing as a fracture. Therefore, a CT scan is better used in surgical decision making to determine if a fracture seen on radiographs is displaced or not. MRI is the better test for determining if a fracture is actually present.

Cast immobilization for 8–12 weeks can successfully treat most non-displaced or minimally displaced scaphoid fractures. There is no consensus on whether to use a short arm or long arm cast. A long arm cast will prohibit wrist rotation and will better immobilize the scaphoid. If treating a scaphoid fracture non-operatively, the authors recommend using a long arm cast for the first 4–6 weeks and then changing to a short arm cast for the duration of the treatment. The disadvantages to cast treatment are increased time to healing, frequent office visits, increased need for radiographs, potential for skin breakdown, and stiffness.

Although controversial, some authors have advocated immediate percutaneous internal fixation of minimally displaced and even non-displaced scaphoid fractures. The reasons behind the trend to immediate percutaneous fixation of these fractures are a decreased time to healing, faster return to sports or work, and a favourable economic analysis in terms of social life and return to work.^{16–20} There appear to be similar outcomes at 2 years of cast versus percutaneous treatment of non-displaced or minimally displaced scaphoid fractures.

Internal fixation is indicated in scaphoid fractures with displacement of at least 1 mm on any radiographic view. Scaphoid fractures with displacement have a much higher probability of progressing to a non-union with cast immobilization compared with internal fixation. The headless compression screw is the most accepted technique for fixation of scaphoid fractures. It has the advantage of being recessed below the articular surface of the scaphoid. The scaphoid is a curved bone, shaped much like a kidney bean, although there can be significant variability. The shape of the scaphoid makes screw fixation challenging; for the best results, the screw should be placed down the central

axis of the scaphoid. Breaching of the scaphoid with the screw during fixation should be avoided since it will lead to early degenerative arthritis. Healing rates of 95% have been reported with adequate screw fixation.

Scaphoid non-unions

Scaphoid non-unions develop in about 5% of scaphoid fractures and are usually the result of inadequate immobilization, displacement or delayed diagnosis. Scaphoid non-unions are difficult because of the tenuous blood supply of the proximal pole and resultant avascular necrosis, the tendency for the scaphoid to collapse into a 'humpback deformity', and the systematic progression to degenerative arthritis and SNAC.

Successful treatment of scaphoid non-unions without collapse, using open and percutaneous techniques has been described. Percutaneous fixation of scaphoid non-unions without collapse or displacement has been described with a cannulated compression screw.^{21,22} Even with resorption of bone at the fracture site, the union rates of percutaneous screw fixation have been as high as 100%. The Matti–Russe technique has traditionally been the treatment of choice for scaphoid non-unions without collapse.²³ This technique uses corticocancellous pieces of bone taken from the iliac crest and placed into the scaphoid after the fibrous tissue from the non-union has been removed. Modifications of the Matti–Russe technique have taken the graft from the distal radius instead of the iliac crest²⁴ or placed a corticocancellous graft with either screw or K-wire fixation. The Matti–Russe technique has been reported to provide union in up to 90% of patients. The majority of patients who did not achieve union were reported to have avascular necrosis of the proximal pole, and this technique is not generally recommended for patients with avascular necrosis.

Treatment of scaphoid non-unions with collapse ('humpback deformity') is more difficult since this requires a need for reduction of the deformity, replacement of the lost bone and stable fixation. Volar wedge fixation with a tricortico-cancellous bone graft taken from the iliac crest has been a successful way to correct a humpback deformity and replace lost bone.²⁵ This technique is usually augmented with screw fixation down the central axis of the scaphoid. Recently, there have been published reports of treating scaphoid non-unions with humpback deformity without structural bone graft and only placing cancellous bone graft. The reduction is performed open through a volar approach and assisted with K-wires, while a screw fixation provides a stable construct for healing. Bone graft is then harvested from the distal radius and packed around the screw to replace the bone lost from the collapse.²⁶ The good results reported with these techniques have not been duplicated when the proximal pole has gone onto avascular necrosis.

Treatment of scaphoid non-unions with avascular necrosis of the proximal pole have been most successfully treated with vascularized bone grafts. The most popular vascularized bone graft is the 1,2 intercompartmental suprapretinacular artery

(1,2 ICSRA) bone graft taken from the dorsal aspect of the distal radius.²⁷ The vascular anatomy of the 1,2 ICSRA is consistent, but very small. Recent reports have shown about a 75% union rate with use of the 1,2 ICSRA vascularized graft. A capsular-based vascularized bone graft has been described by Sotereanos *et al.* and based off the artery of the base of the fourth dorsal compartment.²⁸ This technique has been praised for its simplicity. However, both of these techniques provide only a small amount of bone for reconstructing a humpback deformity and the vascular pedicles are tenuous. Recently, a

free vascularized bone graft taken from the medial femoral condyle (Figure 62.3) and based off the descending geniculate artery has been described.²⁹ This graft has the advantage of replacing articular surface to the scaphoid. The free vascularized medial femoral condyle flap can be taken with or without articular cartilage, with or without a chimaeric skin paddle, and a relatively much larger piece of bone can be harvested. The vascular pedicle of the medial femoral condyle flap is sufficiently robust and is usually anastomosed end-to-side into the radial artery.



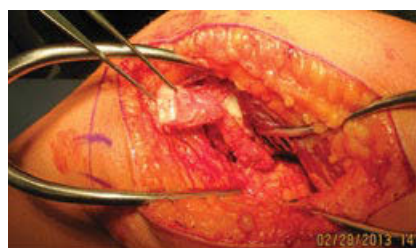
(a)



(b)



(c)



(d)



(e)



(f)

Figure 62.3 (a) MRI showing scaphoid non-union with humpback deformity and avascular necrosis of the proximal pole. (b) Intraoperative radiograph with radiopaque die injected into the scaphoid showing the large defect needed to be filled after all necrotic bone removed. (c) Medial femoral condyle free flap bone with its associated vascular pedicle. (d) Medial femoral condyle free flap being harvested from the knee. (e) Medial femoral condyle free flap being inset into the scaphoid defect. (f) Radiograph showing scaphoid one month after treatment with a medial femoral condyle free flap and microvascular anastomosis end-to-side to radial artery.

Scapholunate dissociation

Injuries to the scapholunate joint are part of a broader category of pathology termed carpal instability dissociative (CID). CID is a dissociation between two bones of the same carpal row. Common injuries such as scapholunate dissociation, lunotriquetral dissociation, unstable scaphoid fractures, inflammatory arthritis or advanced Kienbock's disease can cause CID. Carpal instability non-dissociative (CIND) describes pathology between the bones of two separate carpal rows.

Scapholunate dissociation (SLD) is caused by an injury to the scapholunate ligament that connects the scaphoid and the lunate. It represents a wide spectrum of pathology, ranging from minor sprain of the scapholunate ligament to total perilunate dislocation. With complete SLD the lunate-triquetrum rotate into an extended position while the scaphoid assumes a flexed position as it rotates around the radioscapohcapitate ligament. This classic pathologic position of the scaphoid and lunate-triquetrum is termed dorsal intercalated segmental instability (DISI) and is best identified on a lateral radiograph with an increased scapholunate angle. The DISI deformity caused by SLD is in contrast to the resultant volar intercalated segmental instability (VISI) caused by luno-triquetral dissociation. SLD is part of a process of carpal destabilization that can lead to scapholunate advanced collapse (SLAC) and severe degenerative arthritis.

SLD is usually caused by a fall on a hyperextended and ulnar deviated hand. A low index of suspicion is needed for diagnosis since many of these injuries go undiagnosed because of the subtle physical exam and radiographic findings. Low-grade (predynamic) sprains to the scapholunate ligament may have no discrete physical exam or radiographic finding and are usually diagnosed on arthroscopy. Patients with acute or chronic SLD usually have pain localized at the scapholunate ligament when palpated over the dorsal wrist just distal to Lister's tubercle. Some patients will also have pain in the anatomic snuff box and over the scaphoid tubercle.

Scaphoid shift test is a dynamic test that aids in the diagnosis of complete scapholunate tears. The scaphoid shift test, or Watson test, produces abnormal scapholunate motion and pain when positive. The examiner places the thumb over the scaphoid tubercle (volar) and the other four fingers on the distal radius (dorsal) and pushes the scaphoid in a dorsal direction with the thumb. The examiner then moves the patient's wrist from ulnar deviation (scaphoid extended) to radial deviation (scaphoid flexed). If there is a tear in the scapholunate ligament with instability, the scaphoid will be unable to be flexed and will instead be subluxated from the dorsal aspect of the distal radius. When the pressure is released from the scaphoid, the scaphoid will reduce back into anatomic position with a classic 'clunking' sound. This test is helpful in diagnosis but one should be aware of the low specificity of the manoeuvre.

The scapholunate ballottement test is performed by holding the lunate firmly with thumb and index finger of one hand and

the scaphoid with the thumb and index of the other hand. The scaphoid and lunate are then shifted volar and palmar opposite to each other. An excessive amount of motion, pain and crepitus are signs of a positive result.

The classic radiographic sign of SLD is an increased distance between the scaphoid and the lunate. An increased scapholunate distance on radiographs has been called the 'Terry Thomas' sign (in reference to the English film comedian's space between his two front teeth). However, the normal three views of the wrist (PA, oblique, lateral) may not show an increased distance and as a result there is a high false-negative incidence. Special radiographic views have been described that center the X-ray beam tangentially to the scapholunate joint and therefore show the true extent of the scapholunate distance. The clinched fist view and the Moneim view (PA with 20° of hyperpronation)³⁰ are both helpful in better visualizing the scapholunate distance. A distance of 5 mm or more is diagnostic of SLD.

The lateral radiograph should be examined carefully for DISI. An increase in the scapholunate angle compared to the contralateral side or a unilateral scapholunate angle of 70° (normal range is 30–60°) usually indicates increased flexion or rotatory subluxation of the scaphoid. A DISI deformity is seen in advanced stages of SLD and may not be present in occult or predynamic SLD.

Often, advanced imaging is needed to confirm a suspected SLD. MRI is the advanced imaging of choice used by the authors. With newer 3 Tesla magnets, the sensitivity and specificity of detecting a SLD is 89% and 100%.³¹

Treatment

The treatment of SLD is based on the stage of the injury at the time of treatment. SLD is separated into six different stages which progress from occult scapholunate injury to SLAC. When determining the severity of the injury and subsequent treatment, there are five questions proposed by Garcia-Elias *et al.*³² that help the surgeon understand the pathology and provide a framework for treatment.

- 1 Is the dorsal scapholunate ligament intact?
- 2 Does the dorsal scapholunate ligament have enough tissue to repair?
- 3 Is the scaphoid posture normal?
- 4 Is any carpal malalignment reducible?
- 5 Is the cartilage on the radiocarpal and midcarpal surfaces normal?

Stage I SLD presents as an occult scapholunate injury with only a partial tear of the scapholunate ligament. The dorsal scapholunate ligament is usually intact. Many stage I SLD injuries are diagnosed late, but if they are diagnosed early they may benefit from non-operative treatment including casting, splinting, non-steroidal anti-inflammatory medications and therapy. Arthroscopic debridement and arthroscopic thermal shrinkage have also had reported success.³³ Also temporary scapholunate pinning has been advocated for treatment of occult tears.³⁴

Stage II SLD is characterized by dynamic instability, a complete tear of the scapholunate ligament and enough scapholunate tissue to repair. Patients typically have pain and instability under loading conditions. By definition, there is no fixed deformity. Patients are treated with open repair of the scapholunate ligament, which can be accomplished by a number of methods. Commonly, the scapholunate ligament tears off of the scaphoid and remains attached to the lunate. Direct repair can be done with suture using transosseous suture holes, bone tunnels or suture anchors. K-wires are helpful in reducing the scaphoid and lunate and temporary pinning or headless screw fixation of the scapholunate joint is often helpful.

Stage III SLD is characterized by a dynamic instability, complete tear and an irreparable scapholunate ligament. For a patient to be a candidate for the reconstructive surgeries required to address this problem, there must not be collapse of the carpal bones or degenerative changes. The modified Brunelli procedure has become a popular ligament reconstruction that has had some success,^{35,36} but the preservation of motion and the maintenance of alignment continue to be a problem. A technique called the reduction and association of the scaphoid and lunate (RASL) attempts to make a pseudarthrosis between the scaphoid and lunate.³⁷ There have been some positive short-term results, but no long-term results are available. Loosening of the screw and symptomatic hardware have been a problem with this technique.

The scaphoid will naturally fall into a flexed position, or a DISI deformity, when it is dissociated from the lunate. To counteract this flexion deformity of the scaphoid, a dorsal capsulodesis is performed. The traditional capsulodesis was performed by Nathan and Blatt³⁸ and is proximally based. There have been a number of modifications to the original Blatt technique,^{39,40} which theoretically will allow for more motion. A drawback of the Blatt dorsal capsulodesis is the common 20° loss of flexion.

Stage IV SLD is characterized by a complete tear of the scapholunate ligament, an irreparable ligament and a DISI deformity that is reducible. Rotatory subluxation with the scaphoid in flexion and the lunate in extension is the hallmark of stage IV. SLD with associated DISI deformity represents a severe acute injury, as seen in perilunate dislocation, or a chronic attritional process that has destroyed the secondary stabilizers of the scaphoid. If left untreated, these injuries will predictably progress to degenerative arthritis and SLAC. The progression to SLAC starts with degeneration of the radial styloid, progresses to the entire scaphoid fossa, and then begins to affect the midcarpal joint. However, the time course to SLAC is unpredictable and in many patients is very slow. Some patients will choose to be treated with splinting and activity modification instead of a ligament reconstruction which limits motion. Treatment of stage IV SLD is similar to stage III with the use of a modified Brunelli ligament reconstruction, RASL and/or dorsal capsulodesis.

Stage V SLD is characterized by a complete tear of the scapholunate ligament, an irreparable ligament and a fixed DISI

deformity. There have been a number of limited intercarpal fusions recommended for a symptomatic SLD with DISI deformity. However, few patients fall into this category since degenerative changes usually accompany symptomatic, fixed DISI deformities. There are limited trials or results that support the use of scaphoid-sparing intercarpal arthrodesis. Scapholunate arthrodesis has largely been abandoned because of the low fusion rate.

Stage VI SLD is represented by carpal collapse and degenerative arthritis. SLAC wrist is the term used to describe this deformity. Patients should first be given a trial of non-operative treatment consisting of activity modification, splinting, steroid injections and non-steroidal anti-inflammatory medications. If non-operative intervention fails, then surgery may be indicated. Radial styloidectomy, radioscapoid arthrodesis, scaphoid excision four-corner fusion and proximal row carpectomy have all been described for SLAC. If the midcarpal joint is involved in the arthritic process, then a proximal row carpectomy usually has poor results. Also if the lunate facet and midcarpal joint show degenerative changes, then a total wrist arthrodesis may be the best option for the patient.

Perilunate dislocation and fracture dislocation

Perilunate dislocations (PLDs) are a spectrum of injuries represented by the carpal bones dislocating around the lunate. These injuries start on the radial aspect of the wrist with a scapholunate ligament tear, then progress to the articulation between the lunate and the capitate. The spectrum of injury severity progresses with tearing of the lunotriquetral ligament, dislocation of the capitate from the lunate (usually the capitate migrates dorsally), then finally the lunate dislocates into the carpal canal and possibly rotates on its own axis. These injuries are often missed because they are rare and the X-ray findings are not often seen by general medical providers. PLD can be purely ligamentous as noted above, or can have associated fractures (usually the scaphoid). Lesser and greater arc injuries refer to PLDs that are purely ligamentous or associated with a fracture respectively.

Radiographic evaluation of a PLD starts with standard, high-quality images. A decrease in carpal height, disruption of Gilula's lines (Figure 62.4), increased distanced between carpal bones, and fractures of the carpal bones or distal radius may be seen on the PA radiograph. Ulnar migration of the carpus can also present with PLD since these injuries are such high energy and contain both intercarpal ligament and intracarpal ligament disruption (carpal instability complex). The lateral may show the capitate dislocated from the lunate or possibly the lunate dislocated volarly. MRI and CT scans are often helpful in diagnosing ligamentous injuries and the extent of carpal fractures, respectively.

Non-operative treatment of PLDs or fracture dislocations has been shown to have poor results and is not recommended.



Figure 62.4 Postero-anterior radiograph of a wrist showing the three Gilula's lines. The first line represents the smooth arc connecting the scaphoid, lunate and triquetrum of the proximal carpal row at the radiocarpal joint. The second line represents the smooth arc of the proximal carpal row at the midcarpal joint. The third line represents the smooth arc around the capitate and hamate of the distal carpal row. A break in any line may represent wrist pathology.

Urgent closed reduction to relieve pressure on the median nerve is the first line of treatment. The median nerve must be monitored for progressive symptoms of dysfunction. If closed reduction cannot be obtained, then open reduction in the operating room should be performed. Both dorsal and volar approaches have been described for treatment. The dorsal approach gives good access to the proximal carpal row and the midcarpal joint. The volar approach has the advantage of being able to decompress the carpal tunnel if needed and allows visualization with possible repair of the volar ligaments. Greater arc injuries with fractures of the scaphoid usually have an intact scapholunate ligament and a disruption of the lunotriquetral ligament. Open fixation of the scaphoid and repair of the lunotriquetral ligament has been shown to be a successful treatment strategy for these injuries.⁴¹ Whenever PLD are associated with fractures, most authors recommend fixation with headless compression or cannulated screws. K-wire stabilization

of ligamentous injuries are advocated by some, with or without augmentation by primary ligamentous repair. Intercarpal screws have been compared to K-wire stabilization of ligamentous injuries without significant difference in outcome, except for fewer pin site-related complications with intercarpal screws.

The outcome of PLD treated after a delay in diagnosis (around four weeks or more) is worse than that in those treated soon after injury. There is no surgical procedure that is recommended above another, but the surgical procedure chosen should provide adequate visualization, repair of ligamentous injuries and fixation of fractures. Primary repair of ligaments, suture anchor repair, K-wire stabilization or intercarpal screws are all accepted treatments of unstable ligamentous injuries.

Lunotriquetral dissociation

Lunotriquetral dissociation is an often missed tear of the ligament connecting the lunate and triquetrum. Lunotriquetral dissociation is usually caused by a fall backwards with an outstretched hand in supination and radial deviation. This position concentrates the force on the pisiform, which acts as a punch against the triquetrum. The resulting ulnar-sided wrist pain is often misdiagnosed as a TFCC tear. Lunotriquetral dissociation can also be part of a more severe perilunate dislocation injury.

Lunotriquetral dissociation is part of a category of wrist pathology called carpal instability dissociative (meaning dissociation between bones of the same carpal row) or CID. It follows a familiar pattern of wrist deformity, with the scaphoid and attached lunate rotating into flexion and the triquetrum rotating into extension. The deformity is called volar intercalated segmental instability (VISI). The lunate follows the scaphoid and rotates into flexion (opposite to DISI deformity).

Physical exam findings of lunotriquetral dissociation are often sensitive but not very specific. Pain in the ulnar side of the wrist can represent many different problems. Lunotriquetral ballottement and a triquetral shear test are often positive. Radiographic evaluation will often show a subtle disruption of Gilula's lines and a possible increased lunotriquetral distance. Dynamic radiographs with the wrist in ulnar and radial deviation can be helpful in showing the disruption between the lunate and triquetrum. At times the radiographs will be normal and MRI or arthroscopic evaluation of the ligamentous structures will show evidence of lunotriquetral ligament injury.

If non-operative treatment of an acute lunotriquetral dissociation is initiated, a long arm cast should be applied to inhibit wrist supination and pronation. Failure of non-operative management is not uncommon. The indications for surgical treatment of lunotriquetral dissociation are varied and not very clear. There are many ways to address lunotriquetral dissociation surgically, including arthroscopic debridement, ligament repair, capsulodesis, K-wire stabilization and arthrodesis. Lunotriquetral ligament injuries are often associated with other injuries that may need addressing at the time of surgery.

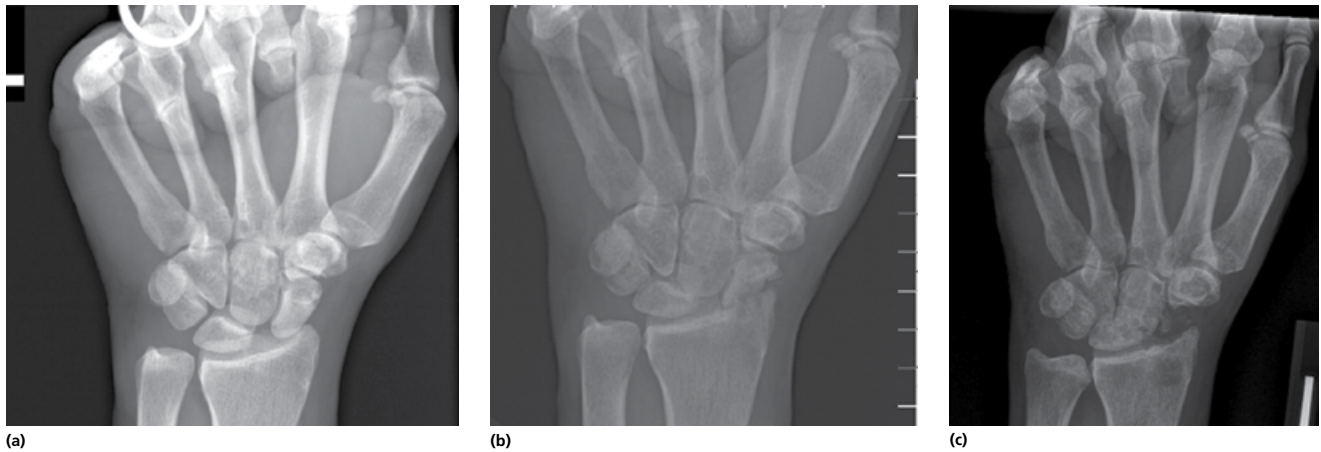


Figure 62.5 (a) Radiograph showing scapholunate widening. The patient had wrist pain, popping and decreased range of motion but chose to treat non-operatively. (b) Postero-anterior wrist X-ray 4 years later of the same patient. He now has severe pain even with rest. There are advanced degenerative changes at the radioscaphoid joint and significant degeneration at the capitulunate joint. The radiolunate articulation is spared. (c) A scaphoid excision and four-corner fusion was performed. The patient was able to return to work as a construction worker.

Scapholunate advanced collapse and scaphoid non-union advanced collapse

Scapholunate advanced collapse (SLAC) and SNAC are arthritic changes that take place in the wrist with a typical progression. They are both caused by trauma, but SLAC can also be caused by non-traumatic chronic changes of the wrist as seen in gout. Watson and Ballet demonstrated that wrist arthritis progressed in a predictable pattern.⁴² Stage I arthritic changes begin at the radial styloid and then progress to (stage II) the proximal radioscaphoid articulation. Arthritic changes next advance to the midcarpal joint between the capitate and the lunate (stage III). The lunate fossa rarely shows arthritic changes.

The diagnosis of SLAC and SNAC is made through history, physical exam and radiographs. The patient usually has a painful wrist with decreased range of motion. Radial-sided wrist pain predominates and dorsal wrist swelling is common. Bilateral radiographs are helpful for comparison. Staging the arthritic changes will help in determining treatment options.

Non-operative treatment will be beneficial for many patients. There are no studies delineating the natural history and many patients will have minimal symptoms for years. Symptomatic treatment with splints, non-steroidal anti-inflammatory medications and steroid injections is the first line of treatment. If a patient fails non-operative treatment and continues to have a painful, arthritic wrist, surgery is an option (Figure 62.5). Surgical options for SLAC and SNAC include partial wrist arthrodesis (scaphoid excision and four-corner fusion), complete wrist arthrodesis, proximal row carpectomy, denervation and radiostyloidectomy. SNAC can also be treated with excision of the distal scaphoid fragment. Traditional teaching states that a proximal row carpectomy should not be performed in patients with capitate arthritis. However, outcomes comparing scaphoid

excision four-corner fusion with proximal row carpectomy show similar results.^{43,44} Proximal row carpectomy has the added benefit of lower cost, no need for hardware, no need to wait for fusion and no extended postoperative immobilization. There has been some question about the lasting durability of proximal row carpectomy with younger patients (<35 years) and patients who are very active or manual labourers.^{45,46} Total wrist arthrodesis reliably relieves pain in the arthritic wrist but sacrifices wrist motion. Total wrist arthrodesis can be performed after a failed proximal row carpectomy or partial wrist fusion.

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CHAPTER 63

Rheumatoid arthritis of the hand and wrist

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Definition

Rheumatoid arthritis is a chronic inflammatory disease of synovial joints. It is characterized by a symmetrical polyarticular arthritis, typically affecting the wrist and small joints of the hand and feet. Inflammation progresses to joint destruction with pain, deformity and disability.

Non-articular manifestations include rheumatoid nodules, pulmonary fibrosis, renal amyloidosis, pericarditis, endocarditis, atherosclerosis, Felty's syndrome, keratoconjunctivitis sicca, haemolytic anaemia and generalized systemic symptoms such as malaise, anorexia, anaemia and weight loss. The course of rheumatoid arthritis is variable and unpredictable but premature mortality is well recognized.¹⁻³

The incidence of rheumatoid arthritis is generally quoted across European and North American literature as affecting 1% of the population. According to the 2009 National Institute of Health and Clinical Excellence (NICE) guidelines about 12 000 people are diagnosed with rheumatoid arthritis each year in the United Kingdom. Rheumatoid arthritis is two to four times more common in females than males, with a peak age of incidence in the seventh decade.⁴

Rheumatoid arthritis may have a significant social and economic impact on the people afflicted with this disease. According to NICE,⁴ approximately one third of people stop work within 2 years of diagnosis and total cost to the UK economy is £3.8 to £4.75 billion per year.

Diagnostic criteria

In 2010 the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR)⁵ revised the classification schema for rheumatoid arthritis (Table 63.1). Diagnosis is confirmed by synovitis in at least one joint, not explained by another cause, and a total score of 6 or more (out of 10) in four domains: joint involvement, serology, acute phase reactants and duration of symptoms. A key aim was to

identify patients with rheumatoid arthritis early, especially those with persistent or erosive disease, and to commence early pharmaceutical intervention.⁶ Assessment may be repeated as symptoms progress.

The classification system is designed to identify new cases of rheumatoid arthritis but patients with erosions typical of rheumatoid arthritis on X-ray, or patients who historically fulfilled the criteria may, of course, still be diagnosed with rheumatoid arthritis.

Pathophysiology

The causes of rheumatoid arthritis are unknown and tremendously complex. Antigen-presenting cells (APCs) bind and present an as yet unknown arthritogenic antigen to T cells. The binding of co-stimulatory ligands, also on the APCs, such as CD80 and CD86, assist in activation of T cells. T cells, fibroblasts and macrophages secrete numerous proinflammatory cytokines, such as the interleukins IL-1 and IL-6 and tumour necrosis factor alpha (TNF- α). These recruit and activate neutrophils, lymphocytes, macrophages, synovial cells, fibroblasts and B cells. Inheritance of certain HLA-II loci may mediate antigen presentation, T-cell reactivity and likely other immune relationships.⁷

B cells differentiate into plasma cells that secrete antibodies and autoantibodies. This process is abetted by IL-6. Autoantibodies include those to immunoglobulin G (IgG) (rheumatoid factor, RF) and citrullinated peptides (anticitrullinated protein antibody, ACPA), among others. The autoantibodies form immune complexes that drive the secretion of proinflammatory cytokines, augmenting the inflammatory process. Activated B cells also act as APCs. In conjunction with cells of the innate and acquired immune system there are numerous other chemokines that guide and control this intricate process.⁸

The final stage in the pathogenesis is a swollen synovium full of macrophages, T cells, B cells, plasma cells and synovial fibroblasts. This synovial pannus then erodes cartilage, bone

Table 63.1 The 2010 American College of Rheumatology/European League Against Rheumatism classification criteria for rheumatoid arthritis

Patients with at least one joint with clinical synovitis, not explained by another disease	Score (out of 10)
A. Joint involvement	
1 large joint	0
2–10 large joints	1
1–3 small joints	2
4–10 small joints	3
>10 joints (at least one small joint)	5
B. Serology	
Negative RF and negative ACPA	0
Low positive RF or low positive ACPA	2
High positive RF or high positive ACPA	3
C. Acute phase reactants	
Normal CRP and normal ESR	0
Abnormal CRP or abnormal ESR	1
D. Duration of symptoms	
<6 weeks	0
≥6 weeks	1

RF, rheumatoid factor; ACPA, anti-citrullinated protein antibody; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.

and supporting soft tissues, leading to joint destruction, deformity and pain. Synovial proliferation around tendons can cause tenosynovitis or tendon destruction and rupture. Joint destruction begins early and erosive changes may occur within months.

Rheumatoid nodules are typically cutaneous and contain a centre of fibrinoid necrosis surrounded by macrophages and fibroblasts and finally a cuff of connective tissue with lymphocytes and plasma cells. These nodules are typically found on areas of repeated mechanical stress or over bony prominences such as the olecranon, the calcaneum and the metacarpophalangeal (MCP) joints. Rheumatoid nodules are associated with more aggressive disease.

Due to a generalized dysfunction of the immune system rheumatoid arthritis patients have an increased susceptibility to infection.⁹

Management principles and operative options

Assessment and evaluation

A rheumatology referral should be made for any patient with a persistent and unexplained synovitis. This includes patients with normal inflammatory markers and those who are RF-negative. Referral should be expedited if the synovitis affects multiple joints, or if the duration of symptoms has been longer than three months. There is an urgency to diagnose rheumatoid arthritis; prompt treatment has been shown to reduce permanent joint damage.¹⁰

Rheumatoid arthritis should be managed by a multidisciplinary team, which may include a rheumatologist, occupational therapist, orthopaedic or plastic surgeon and other allied health professionals.

Box 63.1 Initial assessment of rheumatoid arthritis

- Clinical evaluation: joint synovitis and extra-articular manifestations
- Blood work: ESR, CRP, RF, ACPA, FBC, liver and renal biochemistry
- X-rays of hand and feet



Figure 63.1 Various rheumatoid deformities of the hand including a swan neck deformity of thumb, subluxation of the index MCP joint and generalized arthritic change throughout the hand and wrist. Note fusion of the DIP joint of the ring and the PIP joint of the little fingers.

General assessment

Assessment includes examination for joint synovitis and extra-articular manifestations (Box 63.1). Blood tests should include inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Serology should include RF and ACPA, a positive ACPA being more specific for rheumatoid arthritis. Baseline full blood count (FBC), liver and renal biochemistry are useful for planning treatment. X-rays of hands (Figure 63.1) and feet are appraised for early bony erosions.

Radiological features

X-rays provide a record of the permanent damage of rheumatoid arthritis (Box 63.2). Changes include surface erosions, cystic changes, cortical thinning, osteoporosis and progressive joint narrowing through to frank ankylosis, subluxation and dislocation.

Box 63.2 Radiological features of rheumatoid arthritis

- Joint narrowing and destruction progressing to ankylosis
- Erosions
- Cortical thinning
- Osteoporosis
- Subluxation/dislocation
- Bony destruction (mutilans)

Box 63.3 Poor prognostic features on diagnosis of rheumatoid arthritis

- Positive RF or ACPA
- Early functional limitations
- Multiple swollen or tender joints
- Early bony erosions on X-ray
- Extra-articular manifestations
- High ESR or CRP

In mutilans arthropathy there is bony destruction, which may progress rapidly.

There are a number of radiological classification systems; the most well known are the Sharp and Larsen systems. They score radiographs of the hands, wrists and feet; the higher the score the more severe the bony destruction. The Sharp method scores bony erosions and joint space narrowing separately over 31 areas to give a score from 0 to 448. The Larsen method assesses 20 joints over the hands, wrists and feet, to give a score out of 100. The Larsen method is easier to utilize, but the Sharp method is more sensitive for detecting changes over time. The Sharp and Larsen assessments have significant correlation with each other¹¹ and with the extent of joint and cartilage destruction.¹²

MRI is increasingly used to detect pre-erosive synovitis, and early bone damage not seen on standard radiography. Fat-suppressed gadolinium-enhanced T1 weighted images will demonstrate pre-erosive synovitis, tenosynovitis and bony erosions. The presence of bone marrow oedema, seen on T2 weighted images, is also predictive of the development of bony erosions.¹³ Identification and treatment of subclinical synovitis and prevention of bony erosions are the aims of more aggressive medical management regimes.^{14,15}

Identifying patients with aggressive disease

One of the key goals of initial assessment is to identify those patients with signs that suggest an aggressive disease process (Box 63.3). These patients are monitored closely; a poor response to first-line medical treatment may warrant early use of biological therapies to gain early control of the disease.

Assessment of disease activity

People with newly diagnosed rheumatoid arthritis require regular assessment of disease activity until the disease is stable. Usually this consists of a monthly to tri-monthly review including CRP and a Disease Activity Score (DAS28). The DAS28 is a measure of disease activity in rheumatoid arthritis; an online

tool collates a clinical assessment of 28 specified joints, serum inflammatory markers and a visual analogue scale (0–10) of the patient's global function. A score greater than 5.1 indicates active disease and less than 3.2 indicates well-controlled disease. A score of less than 2.6 indicates remission. Alteration in the DAS28 can be used to assess response to treatment. Until the disease is well controlled, repeat radiography of the extremities is recommended every 6–12 months. Persistent erosive disease despite good symptom control may warrant a change in medication regime.¹⁶ Stable disease requires annual review.

Assessment of disability status and health status

Rheumatoid arthritis is a chronic illness and it is crucial to document both the disability and health status of each patient.

The most common measure of disability status is the Stanford Health Assessment Questionnaire Disability Index (HAQ-DI), which has eight categories covering dressing, arising, eating, walking, hygiene, reach, grip and activities. For each domain, a score of 0–1 represents mild to moderate disability, 1–2 moderate to severe disability and 2–3 severe to very severe disability.

Health status is frequently assessed via the Short Form 36 (SF-36). The SF-36 consists of eight sections covering vitality, physical functioning, bodily pain, general health, ability to perform social roles, ability to perform physical roles, ability to perform emotional roles, and mental health. Each of the eight sections are scored from 0–100, with 100 being normal health status.

Surgical options

The benefits of surgery include relief of pain, recovery of function and cosmetic improvement. Rheumatoid deformities are frequently multifactorial and they must be assessed within the context of the patient's rheumatoid pathology and global function. The decision to offer surgery requires evaluation of the surgical problem and functional limitations. There are general principles when creating a rheumatoid management plan; operate on the lower limb before the upper limb, operate proximally then distally, offer reliable procedures with the highest chance of success first. Souter presented his personal reflections on the relative merits of different rheumatoid hand procedures and felt the three most efficacious procedures were extensor synovectomy and excision of ulnar head, wrist stabilization and thumb MCP joint fusion.¹⁷ However critical analysis of the different surgical procedures is lacking across the literature. Randomized controlled trials comparing different surgical treatments are rare. There is also considerable discrepancy between the views of surgeons and rheumatologists so the benefits of managing rheumatoid patients within the confines of a multidisciplinary team are obvious.^{18,19}

Synovectomy and tenosynovectomy

Synovectomy is the excision of inflamed synovial tissue within a joint and tenosynovectomy implies removal of inflamed synovial tissue surrounding a tendon. The benefits and roles of

synovectomy and tenosynovectomy are quite different; refractory tenosynovitis benefits more clearly from surgical treatment.

Synovitis is increasingly managed by alteration of medications. Isolated synovitis may benefit from intra-articular steroid injection. Recalcitrant synovitis or incidental synovitis discovered during the course of a surgical procedure may be excised but there is no evidence that synovectomy alters disease course or reduces intra-articular cartilage destruction. It may provide temporary pain relief.^{20,21} Possible side effects include discomfort and postoperative stiffness.

Tenosynovitis may affect any tendon surrounded by synovium. Extensor tenosynovitis creates a swelling over the dorsum of the hand, extending beneath and restrained by the extensor retinaculum. The synovial pannus may cause tendon attrition and rupture. Extensor tenosynovectomy is recommended to prevent tendon rupture when synovitis persists for more than 3–6 months despite aggressive medical management. A dorsal longitudinal incision allows exposure of all extensor compartments. The extensor retinaculum is elevated from the sixth to second compartments, ulnar to radial; the tendons are examined for rupture and tenosynovectomy performed. The distal radioulnar (DRU) joint should be examined and treated (synovectomy/capsular repair/Darrach procedure) if required. The retinaculum may then be split in half transversely, with one half being placed beneath the tendons to provide protection from the DRU joint and the other used to reconstitute the extensor retinaculum and prevent bowstringing. Brumfield *et al.*²² reviewed 78 rheumatoid patients, with 102 dorsal wrist tenosynovectomies, intra-articular synovectomies and a Darrach (ulnar head) resection. With an average follow-up of 11 years pain was improved in 83% of wrists, with a 13° loss in wrist movement. Synovitis recurred in 16% of patients, subsequent tendon ruptures occurred in 5% of patients but radiologically the disease progressed unchecked. A total of 27 wrists required revision surgery including tendon transfers, partial and total wrist fusion and wrist arthroplasty.²²

There are three principal types of flexor tenosynovitis: isolated carpal tenosynovitis, palmodigital tenosynovitis and diffuse tenosynovitis. Synovial proliferation within the carpal tunnel may cause carpal tunnel syndrome. Wrist tenosynovectomy is performed through an extended carpal tunnel incision, preserving the palmar cutaneous branch of the median. Bony protrusions on the floor of the carpal tunnel should be smoothed.²³ Palmodigital tenosynovitis typically causes diffuse swelling, triggering and restriction of flexion, and may progress to rupture of flexor tendons due to erosive tenosynovitis. Again, if there is no improvement after three months of intensive medical treatment then tenosynovectomy is indicated. Palmar synovectomy requires broad exposure and may necessitate extension into the digits. Digital triggering may be improved by synovectomy, nodule excision and A1 pulley release. Flatt recommended release of the radial side of the A1 pulleys and cautioned against the release of multiple A1 pulleys, which may potentiate ulnar drift deformity. In this situation resection of the one slip of flexor digitorum superficialis (FDS) is preferred.²⁴

Wrist

The wrist is the major load-bearing joint of the hand and therefore pain or instability may cause significant impairment of upper limb function. Wrist involvement affects up to 50% of patients within 2 years of disease onset, increasing to 90% after 10 years;²⁵ wrist surgery is both common and beneficial to rheumatoid patients.

Pathogenesis of rheumatoid wrist disease

The wrist is inherently unstable and relies on an extensive collection of extrinsic and intrinsic ligaments to preserve stability between the forearm bones and carpus. Extrinsic ligaments extend from the radius and ulna to the carpus; palmar ligaments are stronger than dorsal ligaments. Intrinsic ligaments link the carpal bones only. The scapholunate and lunotriquetral ligaments secure the proximal carpal row and are key in maintaining the mechanics of force transmission through the wrist. Further stability is provided by the flexor and extensor retinaculi and the 23 extrinsic extensor and flexor tendons that surround the wrist. The triangular fibrocartilage (TFC) complex provides stability to the DRU joint. Normally 80% of load is transmitted through the capitate, scaphoid and lunate to the radius. Pathological weakening of ligaments distorts the biomechanics of the wrist, initially during loading and later at rest. Weakening of the important volar radiocarpal ligaments (radioscaphocapitate and radiolunotriquetral ligaments) contributes to ulnar translation and volar subluxation of the carpus. The ulna remains in its original position and becomes relatively prominent as the position of the carpus alters. This collection of signs is termed a caput ulnae deformity (Box 63.4). Bony destruction and intrinsic ligament failure, especially scapholunate ligament disintegration, leads to carpal dissociation and loss of carpal height. With ulnar translation of the carpus the metacarpals fall into radial deviation.

X-ray assessment of the rheumatoid wrist

Standard postero-anterior (PA) and lateral wrist X-rays should be assessed for features of rheumatoid arthritis and caput ulnae deformity. Two further aspects should be assessed: carpal height ratio and ulnar translation. The key features to document are:

- general features of rheumatoid arthritis (osteoporosis, erosion, diminished joint space);
- focal area of arthritis (radiocarpal joint, midcarpal joint);
- features of caput ulnae deformity;
- carpal height;

Box 63.4 Classic patterns of rheumatoid wrist deformity

- Caput ulnae deformity
- Ulnar translation and radial deviation of the carpus
- Palmar subluxation of carpus
- Supination of carpus
- Prominent ulnar head

- ulnar translation; and
- status of the DRU joint/ulnar head.

The carpal height ratio as described by McMurtry *et al.* is the distance from the third carpometacarpal (CMC) joint to the lunate fossa divided by the length of the third metacarpal. The average carpal height ratio is 0.53 (range 0.45–0.60).²⁶ Other methods of calculation are also available.

A rapid semi-quantitative measure of ulnar carpal translation can be made by assessing the position of the lunate relative to the radius; approximately 50% of the lunate should reside over the radius. A formal measure, described by Chamay and Della Santa, can be made by calculating the distance from the centre of the capitate head to the long axis of the ulna, divided by the length of the third metacarpal; the average is 0.3 (range 0.27–0.33).²⁷

Classifications of rheumatoid wrist disease

There have been several classification systems for rheumatoid arthritis of the wrist. The Larsen classification (1973) is a radiographic descriptive classification (Table 63.2).²⁸

The Wrightington classification (1989) is another radiographic classification system, designed to codify wrist pathology and guide treatment (Table 63.3). This scheme lacks the ability to anticipate the progression of wrist disease.²⁹

Simmen and Huber proposed an alternative classification system to try to predict the pattern of disease development (Table 63.4).³⁰ Identifying the broad progression of rheumatoid arthritis can be of assistance in planning surgical procedures, especially the type III patients who the authors felt should be followed closely and offered reliable stabilization of the wrist with the onset of ulnar translation or loss of carpal height.

Partial wrist arthrodesis

A partial wrist arthrodesis in rheumatoid arthritis may be performed for two reasons: isolated radiocarpal arthritis and ulnar translation. Options include radiolunate fusion and radioscapulolunate (RSL) fusion. The benefit of a partial arthrodesis is to preserve wrist movement at the midcarpal joint; most activities of daily living can be accomplished with a range of wrist movement from 5° flexion through 30° extension.³¹ Should it be necessary, subsequent conversion of partial arthrodesis to total arthrodesis is not technically difficult.³²

Chamay and Della Santa introduced radiolunate fusion in 1983 after it was observed that spontaneous ankylosis of the radiolunate joint prevented continued distortion of the wrist and preserved a reasonable range of movement.³³ Partial fusion may be combined with synovectomy and ulnar head resection. The radiolunate joint is denuded of remaining cartilage and the lunate is positioned in a neutral position of 10–15° flexion and secured with K-wires, screws, staples or plates. It is important to place the lunate in a neutral position to allow maximal extension of the wrist. Bone graft may or may not be used. Radiolunate fusion is one of the classic and reliable operations for rheumatoid wrist pathology; it provides pain relief and maintains wrist

Table 63.2 The Larsen classification for rheumatoid arthritis of the wrist

0	No changes
1	Soft tissue swelling, demineralization
2	Marginal erosions, radial deviation
3	Articular erosions. Joint space narrowing, mild radiocarpal instability
4a	Midcarpal ankylosis, major radiocarpal instability
4b	Radiocarpal ankylosis, stable
5a	Destruction of carpus, radiocarpal dislocation
5b	Destruction of carpus, complete ankylosis

Table 63.3 The Wrightington classification of rheumatoid wrist X-rays

Grade 1	Wrist architecture is preserved Peri-articular osteoporosis Early cyst formation Mild rotatory instability of scaphoid
Grade 2	Ulnar carpal translation Marked scaphoid flexion Radiolunate arthritis
Grade 3	Midcarpal arthritis Radioscaphoid arthritis Volar subluxation of the carpus on the radius
Grade 4	Significant loss of bone stock from distal radius

Table 63.4 Simmen's classification for rheumatoid arthritis of the wrist

Type I – Ankylosis	Spontaneous tendency to fuse Stable
Type II – Arthrosis	Articular loss and arthrosis (similar to osteoarthritis) Stable
Type III – Disintegration	Progressive destruction Loss of alignment Unstable

motion.³⁴ Linscheid *et al* reviewed 19 wrists, an average of 28 months postradiolunate fusion; the average wrist extension and flexion were 25° and 30°, respectively, and pain and grip strength were generally improved.³⁵ Progression of arthritis at the midcarpal joint may still occur; this may be due to alteration of carpal kinetics or disease progression.³⁶ Borisch and Haussmann retrospectively reviewed 91 wrists postradio-lunate fusion with a mean follow-up of 60 months. Radiolunate fusion was often accompanied by synovectomy, ulna head resection and division of the posterior interosseous nerve. Radiologically, ulnar translation was effectively reduced. Most patients were pain free and content with the overall result, despite disease progression in 37% of midcarpal joints. There was an 8% complication rate, comparable with other studies; this consisted mainly of problems with osteosynthesis, either displacement or malplacement of metalware or subsequent fractures. Of note the authors recommend that in wrists with a fixed carpal collapse, repositioning of the lunate and restoration of carpal height should not be attempted as this seemed to precipitate secondary arthrosis and cause midcarpal dislocation.³⁷

RSL arthrodesis is a less commonly performed operation, but may be appropriate for patients with extensive involvement of the radioscaphoid joint. Gordon and King reported the first RSL arthrodesis in 1961. Via a dorsal midline incision and through the fourth extensor compartment, the wrist joint is entered, preserving the dorsal wrist ligaments; the articular surfaces of the radius, scaphoid and lunate are denuded to cancellous bone and osteosynthesis performed with or without bone graft. RSL arthrodesis reduces pain and preserves some motion, however the incidence of serious complications is significant, including non-unions, subsequent scaphoid fractures and midcarpal arthritis,³⁸ probably a consequence of altered carpal kinematics. Distal scaphoid resection may improve postoperative wrist flexion-extension; McCombe *et al.* demonstrated an 86% increase in passive midcarpal motion in a cadaver model following distal scaphoid excision with RSL fusion.³⁹

Total wrist arthrodesis

Total wrist arthrodesis is a reliable option for patients with severe painful rheumatoid wrist destruction, flexion deformity secondary to extensor tendon rupture and as a salvage procedure following failed arthroplasty or partial wrist fusion. The radius, scaphoid, lunate, capitate and third CMC joint should be fused.⁴⁰ The suggested position of arthrodesis ranges from 0° to 30° extension, with or without a small amount of ulnar deviation. Reliable arthrodesis was established in the 1960s, when Hazewinkel *et al.* introduced wrist fusion with a Rush pin and bone graft⁴¹ and Clayton used a Steinmann pin.⁴² Mannerfelt *et al.* modified this with the addition of staples⁴³ and Benkeddache *et al.* used staples in isolation.⁴⁴ Internal fixation with an intramedullary nail through the third metacarpal is well known for being a fast and reliable method of fixation, especially in the presence of osteopenic bone; bone grafting is not essential.

In 1971 Meuli *et al.* introduced a nine-hole plate for wrist fusion;⁴⁵ Heim and Pfeffer used a 3.5 mm dynamic compression plate.⁴⁶ Currently a straight or precontoured limited contact dynamic compression plate, with 3.5 mm screws proximally and 2.7 mm screws distally is used. Plate fixation absolutely relies on decent bone stock, it is suitable for osteoarthritic wrists and seldom used in rheumatoid arthritis.

Solem *et al.* reviewed 42 wrist fusions, 34 for rheumatoid wrist disease, over a mean 10.5-year period.⁴⁷ There were 21 Mannerfelt fusions (Rush pin and staples) and 21 plate fusions. There were two non-unions in the Mannerfelt group. The mean position of the wrist following Mannerfelt and plate fusions was 0° and 8° of extension respectively ($p < 0.02$). There was no significant difference in key pinch, grip strength, pain in the wrist and patient satisfaction between the two methods of fusion, although scores in the plate fixation group were slightly better.⁴⁷

There is no clear consensus on the position of fusion in bilateral arthroplasty. Suggestions within the literature range from 10° extension of both wrists, neutral position both wrists

or 5–10° flexion of non-dominant wrist and a neutral position of dominant wrist.⁴⁸ Slight flexion does assist with some activities of daily living (e.g. perineal hygiene).

Total wrist arthroplasty

Total wrist arthroplasty (TWA) is an attractive option for rheumatoid patients with painful wrists, reasonable range of movement and well-preserved hand function. It is especially appealing in patients requiring surgery for bilateral wrist disease. Contraindications include an active rheumatoid arthritis flare, infections, high-demand patients, patients requiring the use of walking aids, poor hand function, poor bone stock or previous carpal resections and inadequate wrist control, especially poor wrist extension.⁴⁹ The surgeon should document the current disease activity, status of the skin and extensor tendons (especially extensor carpi radialis brevis), presence or absence of carpal tunnel syndrome and range of movement.

The first wrist replacement implant was designed by Swanson and consisted of a simple silicone spacer.⁵⁰ Despite similar implants being successful within the small joints of the hand, Swanson wrist replacements are no longer used due to a high rate of complications. Jolly *et al.* reviewed 23 Swanson wrist implants, an average of 72 months following surgery.⁵¹ Complications included implant fracture (58%), revision surgery (30%), radiographic changes consistent with particulate synovitis (30%) and prosthetic settling and bone resorption (75%).⁵¹

A number of wrist surface replacement arthroplasty implants have been introduced since Swanson's silicone spacer. The Menon prosthesis or Universal Wrist Implant was developed by Jay Menon in the 1980s. Titanium-stemmed prostheses are inserted into the radius and carpus, with a broad ellipsoid articulation between a contoured cobalt-chrome surface on the radial component and a polyethylene carpal component. Limited resection of the carpus and intercarpal fusion aids in prosthetic stability. Radial inclination and volar offset improve joint stability and wrist extension. The ulnar head may be retained or a Darrach procedure performed. The Universal-2™ Total Wrist Implant System is a later modification with reduced radial inclination and a porous coating to encourage osseous integration.⁵²

Kailash *et al.* reviewed 92 Universal-2 wrist replacements, 83 for rheumatoid wrist disease.⁵³ With a mean follow-up of 51 months, 91% of patients had satisfactory pain relief with 23° extension and 21° of flexion. Complications included joint stiffness (10%), wrist pain (9%) and superficial infection (2%). Almost 5% of patients required revision surgery and 3.5% needed salvage arthrodesis.⁵³ Salvage options for failed total wrist joint replacement include revision surgery or arthrodesis. Beer and Turner⁵⁴ followed 12 patients who underwent salvage arthrodesis after TWA using tricortical bone graft and an intramedullary Steinmann pin. Seven patients united and five had a painless pseudo-arthritis, with 17 complications in nine patients.⁵⁴

For patients with severe bilateral wrist involvement, wrist arthroplasty of the non-dominant wrist and fusion of the

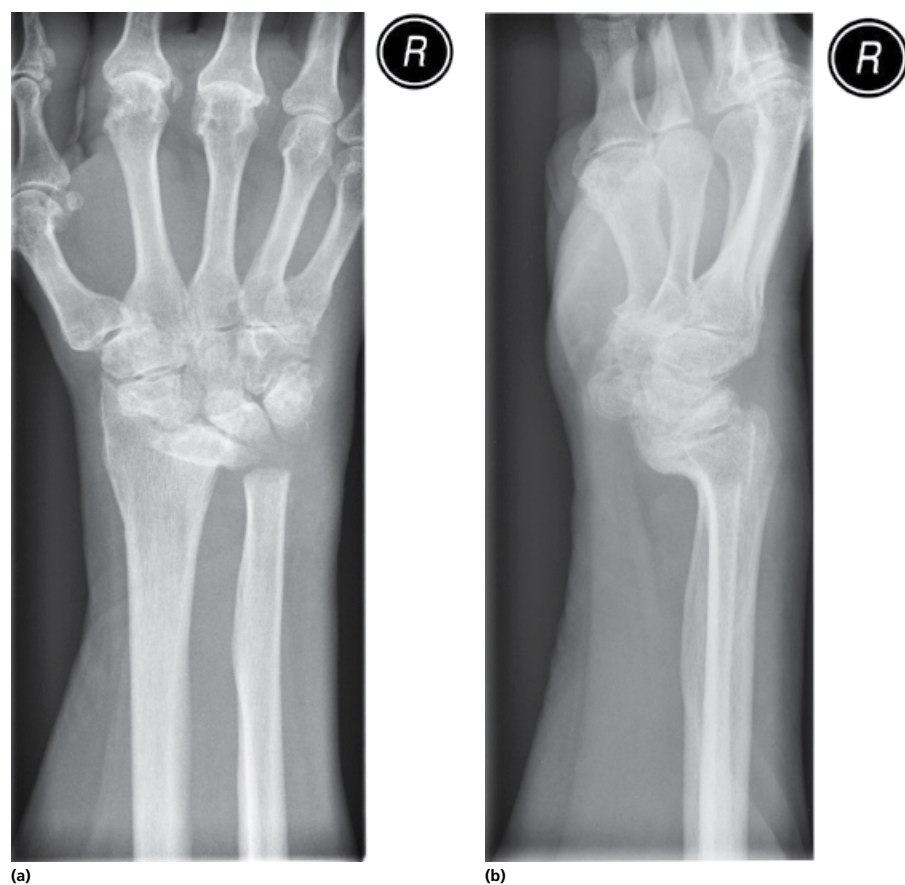


Figure 63.2 (a) Antero-posterior view of wrist post Darrach procedure with marked degenerative change within carpus. (b) Lateral view of wrist demonstrating palmar subluxation of the carpus.

dominant wrist may confer a functional advantage. Vicar and Burton⁵⁵ noted that patients who had a wrist fusion on one side and a wrist arthroplasty on the other prefer the arthroplasty.

Distal radioulnar joint

The DRU joint is often affected early in rheumatoid arthritis. Caput ulnae syndrome is present in 25% of patients; the head of the ulna appears prominent due to carpal subluxation and supination plus DRU joint subluxation. Clinically this generates the piano key sign on examination: downward pressure on the head of the ulna temporarily relocates the ulnar head. DRU joint arthritis occurs in 31–75% of patients and causes pain on pronosupination and compression of the distal forearm.

Ulnar head prominence and associated tenosynovitis can cause tendon attrition and rupture (typically EDM is affected first). Surgical treatment requires synovectomy and either stabilization or excision of the ulnar head.

Ulnar head resection, as described by Darrach in 1912, has good results in the majority of low-demand rheumatoid patients (Figure 63.2). It is easily combined with other procedures on the dorsum of the wrist, such as synovectomy, partial or total wrist arthrodesis and TWA. The surgical approach is dorsal via the fifth compartment, preserving the ECU subsheath of the sixth

compartment. There is minimal bone resection, just proximal to the sigmoid notch; this preserves the attachments of the pronator quadratus, which acts as a dynamic stabilizer. There is no evidence that surgical stabilization of the stump with part of pronator quadratus muscle or flexor carpi ulnaris or extensor carpi ulnaris tendons improves results.⁵⁶ Ulnar carpal translation is not a complication of ulnar head resection.⁵⁷ Thirupathi *et al.* reported that 95% of patients had excellent pain relief.⁵⁸

Rasker *et al.* followed 40 patients with rheumatoid arthritis who had a Darrach procedure.⁵⁹ After 35 months, 38 patients had complete or almost complete resolution of pain. Functional use, supination, pronation and strength all improved.⁵⁹ It is clear this symptomatic benefit applies to rheumatoid patients rather than patients with posttraumatic DRU joint pathology. Fraser *et al.* reviewed patients following resection of the distal ulna for rheumatoid and posttraumatic conditions.⁶⁰ Although 83% of rheumatoid patients were pain free, only 36% of patients with posttraumatic DRU joint problems had similar benefit.⁶⁰

The Sauvé–Kapandji procedure involves DRU joint arthrodesis and segmental resection of the ulna.⁶¹ It is seldom indicated in rheumatoid arthritis. Via a dorsal incision, the DRU joint is exposed, cartilage removed and an arthrodesis is performed with compression screws or K-wires. The ulnar head is placed in

a position of neutral or slight ulnar negative variance. A narrow segment of ulna is removed just proximal to the arthrodesis site to create a psuedoarthrosis. Complications include radioulnar impingement, non-union, tendon rupture and union of the ulna.

Ulna head arthroplasty is a recent option for rheumatoid DRU joint pathology although it is mainly used in osteoarthritis; there are hemi or total ulnar head implants available. Currently it is mainly used as a salvage procedure for failed distal ulna resection or Sauvé–Kapandji procedures, but its indications may expand with time. The benefits of arthroplasty include improved stability of the forearm axis and prevention of impingement. The contraindications to ulna head arthroplasty include poor bone stock, infection, advanced joint destruction and significant instability of the DRU joint. The surgical approach is similar to that for a Darrach or Sauvé–Kapandji; there is careful preservation of the soft tissue stabilizers such as the DRU joint ligaments, ECU and dorsal capsule. A cutting guide marks the amount of ulna resection and after broaching the canal the implant is placed. Sometimes the sigmoid notch is deepened to facilitate stability of the DRU joint. The soft tissues are carefully repaired to provide support.⁶² Berger and Cooney used the Avanta U-Head prosthesis in 22 patients, including some with rheumatoid arthritis and followed them for 2 years.⁶³ Clinical results were good to excellent in 18 of 22 patients. Two patients were successfully revised after prosthetic loosening.⁶³

Digital extensor deficiency

Loss of digit extension is a common problem in rheumatoid arthritis and has a number of possible causes: extensor tendon rupture, posterior interosseous nerve (PIN) palsy, subluxation of extensor tendons or volar subluxation of metacarpal heads (Box 63.5).

Extensor tendon rupture always occurs at the wrist and is due to tendon attrition by proliferative synovium. Vaughan-Jackson first described sequential rupture of digital extensor tendons at the level of the DRU joint. Usually the extensor digiti minimi (EDM) tendon ruptures first, followed by other digital and wrist extensors. Surgery has a reparative and prophylactic role and involves tenosynovectomy and surgical treatment of the ulnar head/DRU joint plus tendon repair or transfer as required.

Due to gradual attrition and retraction of tendon ends, direct repair is usually not feasible. For isolated ruptures an end-to-side transfer or intercalated grafting are options. Multiple ruptures require intercalated grafts or tendon transfers of extensor indicis proprius (EIP) or adjacent EDC tendons (Table 63.5). If all the digital extensors are ruptured then an FDS transfer is required.⁶⁴ The FDS tendons to the third and fourth digits are transferred through the interosseous membrane or around the side of the radius and into the digital extensors.⁶⁵ Interposition tendon grafts can be used with reasonable results in recent ruptures;⁶⁶ excursion of greater than 2 cm of the proximal musculo-tendon unit is required for this to be a feasible option.⁶⁷ Results are always best in patients who maintain good wrist mobility.

Rupture of the extensor pollicis longus (EPL) tendon on Lister's tubercle is common, but the functional deficit depends

Box 63.5 Possible causes of loss of digital extension

- Extensor tendon rupture
- PIN palsy
- Subluxation of extensor tendons
- Volar subluxation of metacarpal heads

Table 63.5 Suggested extensor tendon transfers

Ruptured tendon	Transfer
EPL	EIP to EPL
EDM	Tenosynovectomy and treatment DRU joint
EDM & EDC 5	EIP to EDC 5 or EPL to EDC 5 or EDC 5 to EDC 4
EDM & EDC 4 & 5	EIP to EDM, EDC 4 to EDC 3
EDM & EDC 3–5	EIP to EDM & EDC 4, EDC3 to EDC 2
Multiple EDC, EDM & EIP	FDS 4 to EDM & EDC4, FDS 3 to EDC 2 & 3 & EIP
Thumb & all fingers	FDS 4 to EDC 3–5, FDS 3 to EPL & EIP

on the function of the extensor pollicis brevis (EPB) and the interphalangeal joint. Often EPB and the intrinsic thumb muscles are too weak to fully extend the MCP joint and this causes difficulties with opening the hand for grasp. The treatment of choice is EIP transfer, however with multiple extensor ruptures the EIP may be needed to reconstruct digital extension and so other options need to be considered. An interposition tendon graft may be used for fresh ruptures with sufficient excursion of the proximal musculo-tendon unit.⁶⁸ EPL to EPB transfer provides simultaneous extension of the interphalangeal and MCP joints and the functional limitations are minimal.⁶⁹

Extension of digits is achieved by combined action of the EDC tendons and sagittal bands; sagittal bands are crucial for maintaining the position of the extensor tendons over the metacarpal heads. Radial sagittal band attenuation or rupture causes rheumatoid digits to move into a position of ulnar drift. A zigzag deformity of the wrist and digits is common as rheumatoid arthritis progresses; the metacarpals fall into radial deviation and the digits into ulnar deviation.⁷⁰ Subluxation of extensor tendons between the heads of the metacarpals ultimately causes them to behave as MCP joint flexors.

Extensor tendon subluxation requires rebalancing of the extensor tendons. The extensor hood is opened to allow access to the joint and for synovectomy, the radial sagittal band is then imbricated to centralize the extensor tendon over the head of the metacarpal. A crossed intrinsic transfer may be performed; the tight ulnar lateral band is transferred to the adjacent radial lateral band or MCP joint collateral ligament.⁷¹

Flexor tendon rupture

Flexor tendon rupture is also due to synovial infiltration and attrition and may occur within the wrist, palm or digits. The Mannerfelt lesion is a rupture of the flexor pollicis longus

(FPL) tendon at the wrist and is frequently followed by ruptures of the long digital flexors.⁷² Surgical treatment involves an extended carpal tunnel exposure, synovectomy and debridement of bony prominences. The tendon ruptures are reconstructed on an individual basis and after consultation with the patient. An isolated rupture of the FPL may be treated by interphalangeal joint fusion, tendon graft or FDS transfer. FDP ruptures may be treated with distal interphalangeal (DIP) joint fusion, formal tendon reconstruction or side-to-side transfer. Isolated FDS ruptures may be left untreated and used as tendon grafts.⁷³

Ertel *et al.* reviewed 115 flexor tendon ruptures in 36 rheumatoid patients; 80% of ruptures occurred at the wrist, 17% within the digits and 3% within the palm.⁷⁴ At the wrist level 67% of ruptures were due to attrition on bony spurs and 34% were due to infiltrative tenosynovitis, whereas all ruptures within the palm and digits were due to infiltrative tenosynovitis. Although active motion was achieved in 88% of tendon reconstructions, the average motion was poor; average movement was 23° at the thumb interphalangeal joint and 55° at the digital PIP joints. Isolated ruptures within the palm or wrist had the best results. The authors highlighted the importance of early surgery, if only to debride tenosynovium and bony spurs that may cause further ruptures.⁷⁴

Rheumatoid digital joint replacements

The classic end-stage MCP joint deformity is one of flexion, ulnar deviation and subluxation or dislocation. Proliferative synovitis attenuates or destroys supporting capsuloligamentous structures around the joint. Proximal deformities, such as carpal translation and radial deviation of the metacarpals plus the relatively ulnar pull of long flexor and extensor tendons and the intrinsic musculature all contribute to the ulnar drift. The established surgical practice has been to offer MCP joint arthroplasty only when function is severely limited. Recently the benefits of pain relief and cosmetic improvement, as well as range of movement has altered the indications for MCP joint arthroplasty.⁷⁵ Indications include pain, a significant extensor lag, ulnar deviation or functional impairment, although some patients with advanced changes may remain pain free and function well. These patients may still consider surgery for cosmesis but should be warned that function may not be improved and grip strength may be weakened.⁷⁶ Correction of proximal wrist deformities and extensor tendon ruptures should be performed prior to arthroplasty.

Silicone interposition implants are still the preferred choice due to their intrinsic stability and durability. Swanson's implants encapsulate in scar like all implants; they are not fixed to bone, a fact that almost certainly contributes to their long-term survival.⁷⁷ Encapsulation may mean that implant failure does not necessitate revision of the arthroplasty but implant failure may be associated with increased ulnar drift and poorer outcome.⁷⁸ Pain relief is good but the subsequent range of motion, of about 40°, is not significantly changed. Improvement in extension allows the MCP joint to function

in a more useful arc and facilitates opening of the hand for grasp.⁷⁹ Complications include recurrence of deformity, implant failure, implant dislocation, osteolysis, instability, stiffness and silicone synovitis.⁸⁰

Rothwell *et al.* confirmed significant improvement in hand function following silicone arthroplasty; benefits were maintained for at least 3–4 years following surgery.⁸¹ Chung *et al.* prospectively followed 16 patients following Swanson MCP joint arthroplasty.⁷⁵ At 6 and 12 months functional measures such as grip and pinch strength were unchanged but significant improvements in subjective hand function, activities of daily living, pain, aesthetics and patient satisfaction were reported. Ulnar drift significantly improved too (mean 24°). The arc of motion (mean 46°) was unchanged. The initial benefits of arthroplasty diminish with time. Goldfarb and Stern assessed long-term follow-up of 208 silicone MCP joints arthroplasties over a mean of 14 years.⁷⁸ The mean arc of movement improved from 30° preoperatively to 46° postoperatively but this decreased to 36° at final follow-up. The MCP joints were placed in a better position for grip with improved extension lag; preoperative extension lag was 57°, postoperative extension lag was 11° but this decreased to 23° at final follow-up. Initial improvement in ulnar drift was not maintained. Implant fractures were reported in 63% of arthroplasties and were associated with increased ulnar drift. Only 27% of patients were pain free at the time of final follow-up. Different silicone implants may have different longevity and outcome measures. Sutter implants used a modified design and had a 45% failure rate by 3 years.⁸² Analysis demonstrated that the stress of flexion was concentrated, rather than distributed diffusely as with a Swanson implant, resulting in a high rate of implant failure at the central portion of the hinge.⁸³

A newer option is surface replacement arthroplasty. The indications for these implants are as above but their use is typically contraindicated by inadequate soft tissue stabilizers and poor bone stock.⁸⁴ Cook *et al.* followed 71 pyrolytic carbon unrestrained MCP joint arthroplasties for a mean of 11 years in 26 patients, 19 with rheumatoid arthritis.⁸⁵ The replacements were all used in patients with minimal deformity of the MCP joint. At follow-up, 94% of implants were in a reduced position, with an improved range of motion and 13–16° reduction in extensor lag. There was no change in ulnar deviation. The 10-year implant survival rate was 81%.⁸⁵ Pyrocarbon arthroplasty is a suitable and wear-resistant implant for mild MCP joint deformity in patients complaining of pain.

Flexible silicone implant arthroplasty can also be performed at the PIP joint. Ashworth *et al.* followed 99 Swanson PIP joint replacements. After an average 5.8 year follow-up 67% of patients were pain free. The average postoperative arc of motion was 29°, which was less than the preoperative arc of motion of 38°, and 19% of the implants required revision at 9 years.⁸⁶ Surface replacement arthroplasty for the PIP joint of rheumatoid patients is rare and typically contraindicated due to instability.^{87,88} Murray *et al.* reviewed 67 prostheses in 47 patients with a mean follow-up of 8.8 years.⁸⁹ Seventy-five per cent of patients

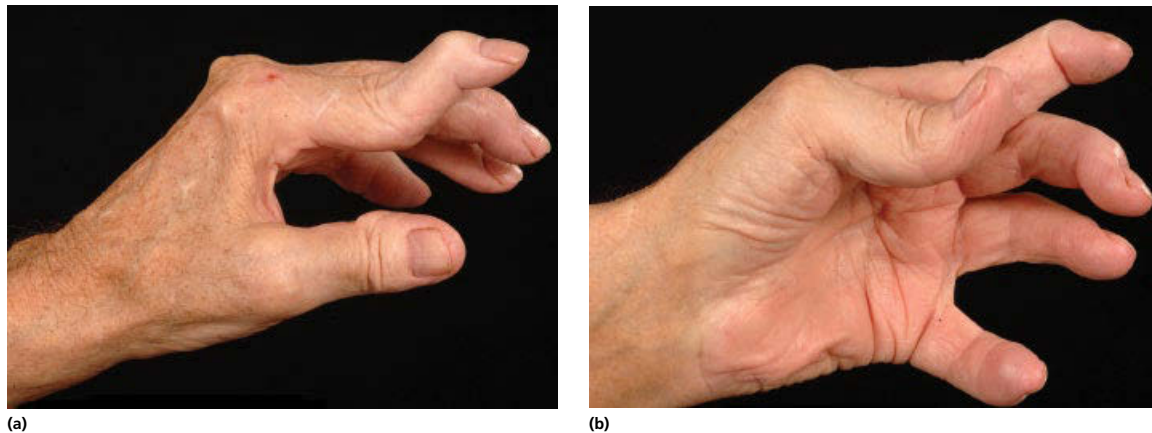


Figure 63.3 (a) Swan neck deformity of index finger. (b) Boutonnière deformity of the thumb.

had osteoarthritis and 25% rheumatoid arthritis. The average range of movement was 40°. There was a 15% failure rate by 15 years, and there were 22 complications requiring four PIP joint fusions and two amputations. The failure rate was not different in osteoarthritis and rheumatoid patients.⁸⁹ The alternative and reliable option is fusion of the PIP joint.

Rheumatoid digital deformities

The two classical digital deformities are boutonnière deformity and swan neck deformity (Figure 63.3). In established rheumatoid arthritis the approximate incidence of boutonnière deformity is 36% and swan neck deformity 14%.⁹⁰ Both conditions have PIP joint deformity due to rheumatoid synovitis but the mechanical aetiology is quite different. Deformities may be classified as fully correctable, partially correctable or fixed. Surgery for rheumatoid digital deformities usually occurs towards the end of the rheumatoid treatment sequence.

Boutonnière deformity is characterized by flexion of the PIP joint and hyperextension of the DIP joint. The primary pathology is synovitis of the PIP joint leading to attenuation or rupture of the central slip insertion, and with time, volar subluxation of the lateral bands; the latter then act to flex the PIP joint and hyperextend the DIP joint. Secondary contracture of the accessory collateral ligaments, transverse retinacular ligaments, oblique retinacular ligament and volar plate creates a fixed deformity. Although patients often dislike the appearance of boutonnière deformity, PIP joint flexion and grasp are not usually compromised.

Formal boutonnière deformity repair is often disappointing and surgeons should not swap a bent digit for a stiff and extended digit. A fully correctable boutonnière deformity usually has an extensor lag of less than 15° and is best treated with splintage. The PIP joint is held straight in the splint and flexion of the DIP joint is encouraged to allow lateral band glide. If the DIP joint cannot be flexed with the PIP joint in neutral then an extensor tenotomy (Fowler tenotomy) may be required; this divides the terminal extensor tendon over the mid portion of the middle phalanx, while preserving the oblique retinacular

ligament (ORL) to enable extension. A partly correctable boutonnière deformity usually has an extensor deficit of 30–40°. Splintage is used initially to convert the partly correctable deformity back to a flexible deformity; extensor tendon reconstruction may then be attempted.

There are a number of methods of extensor reconstruction available, including plication of the central slip and using a distally based FDS slip passed through the middle phalangeal base and woven into the extensor over the proximal phalanx.⁹¹ A fixed boutonnière deformity may require fusion in an improved position.⁹² If X-rays show PIP joint destruction, arthrodesis is usually recommended.⁹³ Kiefhaber and Strickland performed 21 reconstructions for rheumatoid boutonnière deformities. Tendon reconstructions included either reefing of the central slip, coaptation of the lateral bands, use of a free tendon graft, a Matev procedure or flexor digitorum sublimis transfer.⁹⁴ At a mean follow-up of 2 years the results of were variable and unpredictable; the mean final extensor deficit was 39° and the average loss of flexion was 12°.⁹⁵

Swan neck deformity is characterized by hyperextension of the PIP joint and flexion of the DIP joint. Although the hyperextension of the PIP joint is the most obvious clinical finding, the primary pathology may occur at the MCP, PIP or DIP joints. Determining which joint is responsible for the swan neck deformity guides treatment. Secondary contracture of intrinsic muscles may also contribute to the deformity. Swan neck deformity causes difficulty in initiating grasp and can prove a functional problem for patients.

Treatment of swan neck deformity depends on the joint pathology that initiates the deformity (Table 63.6). As with other rheumatoid conditions it is important to treat proximal deformities first. Correctable swan neck deformity requires treatment of the abnormal anatomy. Thus MCP joint dislocation or ulnar subluxation of tendons should be corrected and tight intrinsic muscles should be released. At the PIP joint a palmar restraint to prevent hyperextension may be achieved by either FDS tenodesis or a lateral band transfer. FDS tenodesis involves division of the radial slip of FDS proximal to the A1 pulley, which is then

Table 63.6 Summary treatment guide for swan neck deformity

	Fully correctable	Partially correctable	Fixed
MCP joint	Correction ulnar deviation Arthroplasty and/or Soft tissue correction	Soft tissue release and Arthroplasty and/or Intrinsic release	As for partially correctable
PIP joint	FDS tenodesis or Lateral band transfer	Soft tissue release and FDS tenodesis or Lateral band transfer	Arthrodesis or arthroplasty
DIP joint	Arthrodesis	Arthrodesis	Arthrodesis

secured to the proximal phalanx or to the A1 or A2 pulley,⁹⁶ keeping the PIP joint in 20° of flexion and preventing hyperextension. The other option is a lateral band transfer as described by Zancolli; the lateral band is mobilized from the midpoint of the proximal phalanx to the midpoint of the middle phalanx, the flexor sheath is opened between the A2 and A4 pulleys and the radial lateral band is translocated palmarward around a sling created between the FDS tendon slip and palmar plate.⁹⁷ Lateral band transfer has the benefit of correcting both PIP joint hyperextension and DIP flexion. Tonkin *et al.* performed lateral band transfer in 30 digits with swan neck deformity, the majority of patients having rheumatoid arthritis.⁹⁸ Mean preoperative hyperextension of 16° was corrected to a mean flexion contracture of 11°; all deformities were corrected with no recurrences or complications.⁹⁸ Deformities that are not passively correctable may imply contracture of soft tissues around the joint, which require release before surgical correction of the primary deformity may be performed. This typically involves release of the dorsally subluxed lateral bands off the central slip and dorsal capsulectomy or capsulotomy.⁹³ Absence of movement may indicate articular damage, which requires either arthrodesis or arthroplasty.

Rheumatoid thumb deformities

The thumb is commonly involved in rheumatoid arthritis; 60–80% of patients are affected.⁹⁹

Nalebuff developed a classification system for the rheumatoid thumb by identifying the joint primarily responsible for deformity (Table 63.7).^{100,101} In type I (boutonnière deformity) the primary pathology is synovitis at the MCP joint with attenuation of the EPB insertion and subluxation of the EPL ulnarly. Extensor force concentrates on the interphalangeal joint, causing hyperextension. It is the most common thumb deformity.¹⁰²

In type II thumbs, a boutonnière deformity is also present but originates due to pathology at the CMC joint. In a type III thumb (swan neck deformity) the problem originates with synovitis at the CMC joint, causing dorsal and radial subluxation of the metacarpal base, adduction of the first metacarpal, narrowing of the first web and reciprocal MCP joint hyperextension and interphalangeal joint flexion. Belt *et al.* evaluated X-rays over a 20-year period and found that 81% of rheumatoid

Table 63.7 Nalebuff classification of rheumatoid thumb deformity¹⁰⁰

I	Boutonnière deformity
II	Boutonnière deformity with CMC joint involvement
III	Swan deformity
IV	Gamekeeper's thumb
V	Swan neck deformity due to MCP joint involvement
VI	Arthritis mutilans

patients with greater than 4 mm CMC joint subluxation developed swan neck deformity of the thumb.¹⁰³

In a type IV thumb (gamekeeper deformity) attenuation of the ulnar collateral ligament creates lateral instability of the MCP joint. Type V deformity is a swan neck deformity due to volar plate laxity at the MCP joint with a normal CMC joint. Type VI deformity is arthritis mutilans with extensive bony resorption.¹⁰⁴

Although the Nalebuff classification provides a mechanical explanation for the zigzag deformities found in rheumatoid thumbs, it is the two concepts of boutonnière deformity and swan neck deformity that are of value to surgeons.

A boutonnière deformity classically requires treatment at the MCP joint and occasionally at the interphalangeal joint. Terrono *et al.* followed 53 rheumatoid patients with boutonnière deformities of the thumb over 7 years. The authors classified type I deformities into early, moderate and advanced to aid assessment and treatment. Early deformities had fully correctable MCP and interphalangeal joints; treatment was MCP joint synovectomy and EPL–EPB rerouting, as described by Nalebuff.¹⁰³ In moderate deformities the MCP joint was destroyed and fixed but the interphalangeal joint was correctable; surgical options were MCP joint arthroplasty or arthrodesis. Advanced deformities were fixed at both joints, which were radiologically destroyed and both joints required treatment; options included MCP joint arthroplasty or fusion and interphalangeal joint release or fusion. For moderate deformities MCP joint arthrodesis provided a high degree of satisfaction (4.7/5), and a pinch-strength of 6 pounds (2.7 kg); 13% had subsequent interphalangeal joint collapse. MCP joint arthroplasty patients experienced a lower satisfaction rate (4.1/5), and a 3.5 pound (1.6 kg) pinch-strength; 23% subsequently had interphalangeal joint collapse. In advanced cases 85% of interphalangeal joint releases failed. Combined MCP joint and interphalangeal joint fusion were recommended for high-demand patients and MCP joint arthroplasty and interphalangeal joint fusion for low-demand patients.

EPL rerouting can be performed with numerous variations. Nalebuff's original description divides the EPL just distal to the MCP joint and attaches it either into the dorsal capsule of the MCP joint or to the proximal phalanx, EPL then extends the MCP joint. The EPB is reattached to the EPL and extension of the interphalangeal joint is performed by the intrinsic muscles via the lateral bands.¹⁰⁴ Another option is EPB-plasty: the thinned or ruptured EPB is dissected free from the extensor hood and reattached to the proximal phalanx. The extensor hood is plicated over the tendon

repair.¹⁰⁵ Both procedures required immobilization of the MCP joint for 4–6 weeks postoperatively.

MCP joint arthroplasty is performed through a sinusoidal incision with capsulotomy between EPB and EPL. A minimal amount of bone is resected, preserving the collateral ligaments. The largest silicone implant that fits is inserted, capsular structures are repaired and the attenuated EPB and EPL are restored to their anatomical position.¹⁰⁶ Average range of movement is about 25°, with an improved flexion arc of 15–40°.¹⁰⁷ Complications include implant failure and instability. Arthroplasty is not suitable for high-demand patients or patients with gross instability around the MCP joint. Conversion to arthrodesis may require bone graft.

Arthrodesis of the MCP joint is a reliable procedure for disorders of the rheumatoid thumb, especially if CMC or interphalangeal joints are intact. The position of fusion is usually 15° flexion, 5° abduction and 20° pronation.¹⁰¹ Fusion rates range from 80 to 100%. Bone grafting may be required, particularly for arthritis mutilans.

Surgical treatment of swan neck deformity usually involves trapeziectomy to position the thumb better; ligament reconstruction and tendon interposition as described by Burton and Pellegrini¹⁰⁸ is optional. Persistent hyperextension of the MCP joint may warrant sesamoid arthrodesis¹⁰⁹ or fusion depending on X-ray appearances. Very occasionally, restriction of the first web may require deepening or release.¹¹⁰ Arthrodesis of the CMC joint is not recommended.

Fortunately, rheumatoid arthritis does not usually affect all three thumb joints to the same degree. Soft tissue procedures often do not provide long-term correction but may be performed cognizant that further surgery may be required. When more than one joint is affected it is best to preserve mobility in as many joints as possible: arthroplasty is preferable at the CMC joint, the MCP joint may have either arthroplasty or arthrodesis, depending on patient demand, and the interphalangeal joint is usually fused.¹¹¹

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CHAPTER 64

Lesions of the hand

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Introduction

Hand tumours represent a substantial proportion of the workload for hand surgeons. Most tumours are benign and arise from soft tissues. Ganglions, giant cell tumours of tendon sheath and inclusion dermoids make up the bulk of soft tissue lesions seen within the hand, whereas malignant tumours are much less common. Bone tumours are seen less often than soft tissue lesions; the vast majority are benign and of these enchondromas predominate. Primary malignant bone tumours are extremely rare within the hand. Metastases occur and most frequently involve the terminal phalanges.

Despite the relative rarity of malignant lesions, the surgeon must remain alert and keep the possibility of malignancy in mind whenever dealing with any hand tumour, as this diminishes the chance of missing or mistreating a malignancy. The principles of management of malignant tumours, including appropriate staging and biopsy, have been well described and must be conscientiously adhered to.

Approach

It is necessary to ask about the duration and progression of symptoms (slow-growing lesions are more likely to be benign, rapid growth implies a higher chance of malignancy), the presence of pain, particularly night pain, and response to medication (osteoid osteomas are said to respond readily to aspirin) and symptoms of neurological dysfunction. Systemic features such as unexplained weight loss and respiratory symptoms should always be sought.

Physical examination should include: site and size of the lesion (lesions greater than 5 cm in diameter are more suspicious for malignancy;¹ consistency; colour; an attempt to define the tissue level of the lesion (e.g. skin, subcutaneous tissues, deep to tendon); fixity to surrounding structures such as bone or tendon; range of motion of the adjacent joints (e.g. dorsal wrist ganglions often impair dorsiflexion); the presence of nerve or vascular dysfunction distal to the lesion; the presence of a

Tinel's sign on tapping upon the lesion; and an examination of draining lymph nodes (epitrochlear and axillary).

Investigation

Plain radiographs

A plain radiograph of the hand is the starting point for investigation of bone tumours. Considerable information about the biology and behaviour of the tumour can be obtained from the margin between bone and tumour and the reaction of periosteum.

If there is a very clear, distinct margin between the tumour and the bone this is described as 'a well defined zone of transition'. This implies a slow-growing, usually benign lesion where the host bone has time to respond to the lesion. This kind of margin is typically seen in enchondromas. If the margin is indistinct the lesion can be described as permeative and this implies a more rapidly growing and potentially sinister lesion.

A periosteal reaction is a worrisome feature and implies infection or an osteosarcoma. Speckling of the interior of the tumour, known as matrix calcification, implies a cartilaginous tumour.

CT scan

CT scans are helpful in assessing complex regions of the upper limb, particularly the carpal bones, where plain radiographs may be difficult to interpret. They can also be useful in searching for the nidus that characterizes an osteoid osteoma, and defining the relationship of the cortex of a tumour and the adjacent normal cortex, which is beneficial in distinguishing between an osteochondroma and less common surface lesions such as juxta-cortical chondromas and Nora's disease.

Ultrasound and MRI

Soft tissue tumours are best imaged with ultrasound, if a skilled technician and radiologist are available, and/or MRI. The principal use of an ultrasound is in distinguishing between solid and cystic lesions, especially in confirming the presence of a ganglion. Dynamic imaging is possible with ultrasound and the

capacity to evaluate blood flow is particularly helpful in the evaluation of glomus tumours.

An MRI provides excellent definition of soft tissue lesions and is useful in determining the anatomical location of the lesion and involvement of surrounding structures. It is mandatory in the setting of suspected malignancy. An MRI must be performed prior to a formal biopsy as the soft tissue planes will be disturbed by the surgical procedure and interpretation of the images will be adversely affected. It is good practice to notify the radiologist about any concerns regarding the potential for malignancy because an infusion of gadolinium will often be necessary; if this is not done initially the patient may need to be recalled for further imaging.

Blood tests

A full blood count and serum electrophoresis (EPG) should be performed when malignancy is suspected to assess for leukaemia and myeloma. Calcium, phosphate, urea, electrolytes and creatinine and liver function tests should also be checked in suspicious cases.

Biopsy

Many lesions of the hand are treated with excisional biopsy; for example suspected ganglions, giant cell tumours and small lipomas are usually definitively treated with excision through the reactive zone immediately surrounding the tumour. For lesions where there is a suspicion of malignancy an incisional biopsy is performed as the first stage of treatment, followed by definitive surgery when the tumour has been characterized.

The principles of incisional biopsy must be considered in all cases:

- Perform appropriate local imaging prior to surgery.
- Use a tourniquet and exsanguinate by prolonged elevation, rather than an Esmarch bandage as there is a theoretical risk of proximal seeding.
- Site the incision to allow inclusion in any subsequent definitive surgery.
- Use a longitudinal incision.
- Confine the incision to one compartment, avoid spreading with scissors and contamination of surrounding tissues.
- Obtain a substantial amount of material for analysis; include the periphery of the tumour as the centre of the tumour may contain only necrotic material.
- Infection must always be considered and swabs and specimens for microbiology should be taken as a routine.
- Employ exacting haemostasis and try to avoid use of a drain; if one is necessary it should exit in the line of the incision, close to the incision and ideally distal to the incision.

It is always prudent to approach the lesion with the question 'If this turns out to be malignant, will the current operation compromise any future operation?' Ideally, an incisional biopsy should be performed by the surgeon who will go on to definitively manage the lesion. It is good practice for the responsible

surgeon to physically convey the specimen to the pathologist to ensure that it does not go astray.

Soft tissue lesions

Skin

Epidermal inclusion cyst

These are common lesions, comprising approximately 10% of all hand tumours.² They tend to occur in the glabrous skin of the palm and fingers. A minor penetrating injury, which is usually not remembered by the patient, results in the implantation of some epidermal cells in the dermis, where they continue to produce keratin. The resulting collection of keratin within an epithelial lined cyst is a painless, smooth round tumour, not mobile relative to the skin but not fixed to underlying structures. Alternate theories of their origin have been proposed, including occlusion of pilosebaceous follicles or an arrest of keratinocyte migration.³ These cysts do not necessarily require treatment, but can interfere with function or grip, and can be complicated by infection. Treatment is by surgical excision, which should aim to remove the entire cyst wall, without penetrating the cyst, to reduce the chance of recurrence. If urgent incision and drainage is required for an infection, complete excision should not be attempted. A further procedure should be planned for at least six weeks after infection has settled.

Warts

Common warts are the most common neoplasm in the hand, and are caused by the human papilloma virus (HPV). They are generally self-limiting and will spontaneously regress within 2 years. Treatment to remove warts is best focused on stimulating immunity to HPV, with use of topical irritants such as salicylates. Cryotherapy can be used as first line but care should be exercised when underlying structures are at risk, for example the germinal matrix if the lesions are over the dorsum of the distal phalanx. On rare occasions, warts are resistant to non-surgical therapy and excision and grafting may be necessary. Recurrence at the margins of surgery is not unusual.

Actinic keratosis

Actinic or solar keratoses are pink, scaly macular lesions arising mainly on the sun-exposed dorsum of the hand. They have a modest risk of malignant transformation to squamous cell carcinoma (SCC), although this is difficult to quantify. They can be satisfactorily treated by curettage of the lesion, although recurrent lesions should be biopsied to exclude malignant transformation.

Squamous cell carcinoma

SCCs comprise approximately 75% of skin malignancies of the hand, and are the most common malignancy of the hand overall.⁴ They are more common in men, by a ratio of 4:1. There is an overall rate of metastasis of 3%, although SCC of the hand has higher rates of metastasis and recurrence. They present as



Figure 64.1 Squamous cell carcinoma on the dorsum of the hand.

pink or red nodules or plaques which can exhibit scale, ulceration or induration (Figure 64.1). Worrying features for both metastasis and local recurrence are ulceration, greater size of the lesion, lymphatic or perineural invasion. Keratoacanthoma can present as a similar lesion, clinically very difficult to distinguish from SCC. It can exhibit rapid growth and then spontaneous resolution, however prompt excision and histopathological examination is strongly recommended given the obvious risk of observing a potentially malignant lesion.

Treatment of SCC is by surgical excision. The lesion should be excised with one tissue plane below the tumour, and the appropriate margin depends on the size of the lesion. Tumours smaller than 2 cm in maximum diameter should be excised with a 5 mm margin, whereas larger tumours should have a 1 cm margin.⁵ Appropriate margins create defects that often need coverage with full-thickness skin grafts. Metastasis of SCC is by lymphatic spread and the antecubital fossa and axilla should always be examined for evidence of nodal spread. Where axillary lymph node metastases are confirmed, an axillary lymph node dissection should be performed.

Basal cell carcinoma

Basal cell carcinoma (BCC) occurs more commonly in sun-exposed areas, and is therefore prevalent on the dorsum of the hand. It occurs more commonly in men. Metastasis is extremely rare, and the treatment is by excision with clear margins. A 3 mm margin of excision is recommended for the more common nodular and superficial BCCs, although a 5 mm margin is necessary for morpheiform BCC where the margin is less distinct.⁵ Superficial BCC proven on biopsy can be treated with topical immunotherapy in the form of imiquimod, but the inconvenience of ulceration and dressings on the hand leads us to recommend excision in almost all cases.

Melanoma

Melanoma comprises approximately 3% of hand malignancies, and subungual melanoma is as common as the cutaneous variety. As with pigmented lesions elsewhere, suspicious lesions



Figure 64.2 Subungual melanoma with Hutchinson's sign.

should be biopsied promptly. Even where the index of suspicion is high, an excisional biopsy should have a small (2 mm) margin so as not to disrupt the lymphatic drainage of the area should sentinel node biopsy be necessary.

Subungual melanoma typically presents with longitudinal melanonychia, a band of brown/black in the nailbed as a result of melanin pigment deposition. Hutchinson's sign is the spread of pigmentation into the proximal nail fold and is an important though not pathognomonic sign of subungual melanoma (Figure 64.2). Subungual melanomas can mimic subungual haematomas, and unless there is a clear history of trauma and evidence of progression of the pigment with nail growth, the nail should be removed and the area biopsied.

Definitive management of melanoma is guided by histological analysis of the lesion. Melanoma *in situ* requires a 5 mm margin; tumours with a Breslow thickness of less than 1 mm should be excised with a 1 cm margin, those with a greater than 2 mm thickness require a 2 cm margin, and intermediate thickness melanoma should have a margin of at least 1 cm, with additional margins at the surgeon's discretion.⁶ Subungual melanoma cannot be given a Breslow thickness and treatment should be by amputation through the distal interphalangeal (DIP) joint.⁷

Use of sentinel lymph node biopsy (SLNB) varies widely. There is some controversy in the literature as to whether SLNB has a survival benefit either as a treatment itself or when it leads to regional lymph node clearance. It can yield useful prognostic information to patients as to their likelihood of metastasis and survival, and if done should be performed at the time of wide local excision.⁸

Other malignant skin lesions

Tumours of the eccrine sweat glands are rare, and consist of a variety of tumours including mucinous carcinoma, adenoid cystic carcinoma, porocarcinoma, malignant mixed tumour, cylindrocarcinoma and spiradenocarcinoma. They can be locally aggressive, and metastasis has been described, although its rate is difficult to estimate given the rarity of the conditions. Treatment is by surgical excision with a wide margin. Chemotherapy and radiotherapy have not been shown to be effective.

Merkel cell tumours are aggressive and carry a poor prognosis. They present as a painless, purple nodule of variable size. They arise from the Merkel cells of the basal layer of the epidermis. They are very radiosensitive and while neoadjuvant radiotherapy has not been shown to improve survival, it reduces the rate of local recurrence. Surgical treatment should be by excision with wide margins.

Fat

Lipoma

Lipomas are very common soft tissue tumours, although they are relatively rare in the hand. They are rarely symptomatic except when they grow sufficiently large to act as a space occupying lesion, for example in the carpal tunnel⁹ or Guyon's canal (Figure 64.3). Lipomas are typically soft, non-tender masses and can be multilobulated. The Bufalini sign is a relatively radiolucent area within the soft tissues on plain radiographs due to the transparency of the fatty tissue of the lipoma in relation to muscle. Preoperative imaging with MRI is recommended to delineate the extent of the tumour and also to confirm the absence of suspicious features of a soft tissue malignancy. There is typically a well-circumscribed mass with the features of normal fat on T1 and T2 images. Treatment is by surgery or observation. Complete excision, without requiring a margin of normal tissue, is generally curative. Malignant transformation is extraordinarily rare.



Figure 64.3 Large lipoma in the palm extending into Guyon's canal and causing ulnar nerve compression.

Lipoblastoma

Lipoblastomas are a distinct type of fatty tumour, most often occurring in children under 3 years old. They are composed of lobulated tissue with immature fat cells separated by fibrovascular septae and areas of myxoid matrix. Treatment is again by surgical excision.

Connective tissue

Ganglion cyst

This is the most common tumour of the hand and wrist. Ganglions are not true cysts, as there is no epithelial lining to the cavity. They are collections of mucin with a connective tissue wall, often with a stalk leading to a point of origin. Their pathogenesis is unclear, but may relate to mucinous degeneration of the capsule of a joint or the fibrous flexor sheath. There are four common sites of ganglion cysts in the hand: volar and dorsal wrist, DIP joint and the palm.^{10,11}

Ganglia tend to wax and wane in size (sharing this characteristic only with some vascular lesions), particularly in lesions around the palm, are soft to firm but compressible and may transilluminate.

Volar wrist ganglion cysts generally occur on the radial side and typically arise from the scaphotrapezium-trapezoid (STT) joint. They are often adherent to the adjacent radial artery, which is at risk during excision. It is not uncommon to mistake the radial artery for the stalk of the ganglion and the artery should be defined prior to excision. Dorsal wrist ganglia usually arise from the scapholunate ligament, and result in dorsal wrist pain, which is exacerbated by forced dorsiflexion as in pushing up from a table or performing pushups.

Ganglia over the dorsum of the DIP joint are conventionally called mucous cysts. They commonly arise in the context of DIP joint osteoarthritis, particularly with dorsal osteophytes. They can cause a pressure effect on the germinal matrix, leading to a characteristic concave deformity of the nail distal to the lesion. Palmar ganglion cysts occur over the metacarpophalangeal (MCP) joints and can arise from the volar capsule of the joint or the flexor sheath (Figure 64.4).

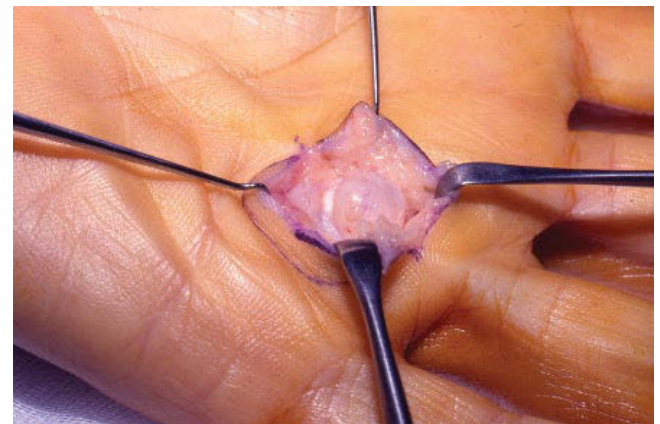


Figure 64.4 Ganglion of the flexor sheath.

As ganglion cysts frequently spontaneously resolve, and are essentially harmless masses, treatment is required only where they persist and cause discomfort or where the patient finds the lump unsightly. On rare occasions they can cause problems from a mass effect – for example a ganglion in Guyon's canal resulting in an ulnar nerve compression. Discharge of a mucous cyst through the nail fold or through breakdown of the skin over the cyst is a strong indication for surgery.

Treatment of ganglia is by complete excision of the lesion and excision and cautery of the point of origin. When designing incisions to approach ganglia, particularly those in atypical positions about the wrist, one must be aware that the stalk of origin may be long and lead some distance away from the palpable lesion. Sometimes a second incision is required to provide adequate access to the origin. Another important point is to avoid closure of the wrist capsule as this will only result in stiffness.



Figure 64.5 Mucous cyst with planned excision and local flap for closure.

Excision of dorsal osteophytes at the DIP joint and the joint capsule between the extensor tendon and the collateral ligaments can help reduce recurrence of mucous cysts. Occasionally the ganglion can result in overlying skin of poor quality, in which case a small local flap is required for skin closure (Figure 64.5). Recurrence rates are in the order of 10% with surgery. More conservative treatment options such as closed rupture or needle aspiration have higher recurrence rates and are not recommended, particularly as they may lead to septic arthritis of the DIP joint.

Giant cell tumour of tendon sheath

These are slow-growing firm lesions arising from the fibrous flexor sheath or joint. They are typically seen in the third to fifth decade of life, and present with a long history of a gradually enlarging, firm, non-compressible painless mass on the finger, commonly around the DIP joint. They do not transilluminate, which can differentiate them clinically from mucous cysts. They have no malignant potential, but can be locally aggressive, invading bone and tendon. Complete excision should be curative but is difficult particularly in extensive lesions, and recurrence is common, occurring in 20–50% of cases. A higher recurrence rate of giant cell tumour associated with downregulation of the *nm23-H1* gene has been reported¹² and immuno-histochemical analysis of nm23 expression within the tumour has been advocated for prognostic information. However this has been challenged in a more recent larger series.¹³ Incomplete excision is the greatest determinant of recurrence, and with a benign condition the need to avoid morbidity from radical excision of flexor tendons or annular pulleys makes it difficult to be confident of complete excision (Figure 64.6).

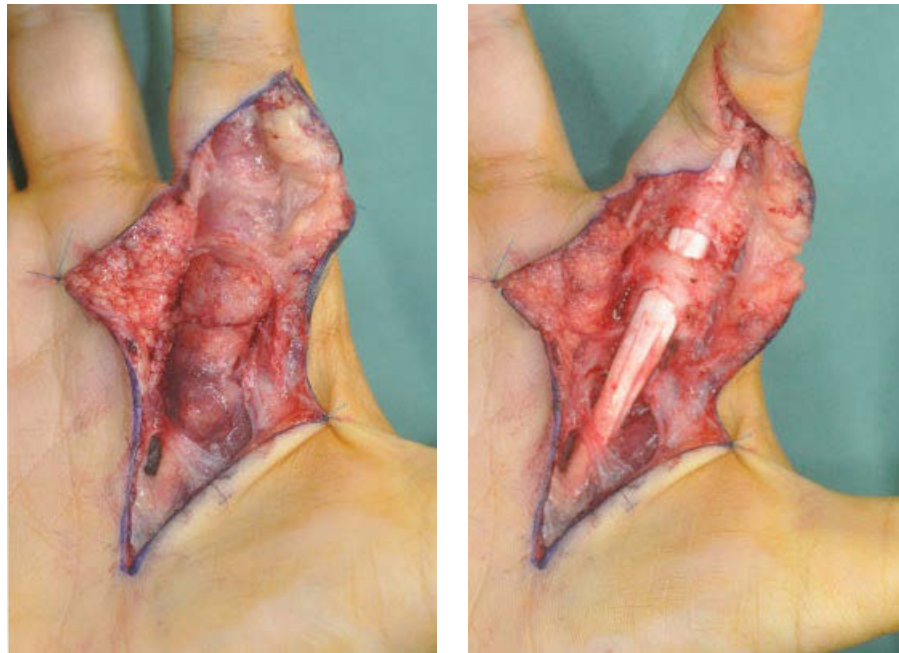


Figure 64.6 Giant cell tumour of flexor sheath before and after excision. Source: Prof. Christopher Coombs. Reproduced with permission.

Fibromas

Digital fibromas in adults present as painless firm masses, often in and around the fibrous flexor sheaths. They can be clinically difficult to differentiate from giant cell tumours. Treatment is by surgical excision. Infantile digital fibromas, or fibromas of Reye, are nodular proliferations more typically found on the dorsal or lateral aspect of the finger, occurring in the first 2 years of life (Figure 64.7). Many will spontaneously resolve, however recurrence is common after removal. It is therefore recommended to manage these conservatively and operate only in cases of functional impairment.

Juvenile aponeurotic fibromatosis is a condition of nodules in the palms (and soles) of young children. They can become calcified and be visible on plain X-ray imaging. These are benign lesions which are managed by excision in the case of masses causing a functional problem. Recurrence is common and excision with a margin of normal palmar aponeurosis is recommended.

Vessels

Historically the terminology used to describe vascular anomalies was confusing, with lesions grouped together inappropriately solely on their appearance. Mulliken and Glowaki¹⁴ proposed a classification based on the histological characteristics of the lesions which forms the basis of current terminology as adopted by the International Society for the Study of Vascular Anomalies (ISSVA).¹⁵ Vascular lesions can be broadly divided into tumours and malformations.

Haemangioma

This is a vascular tumour of infancy. It is not present at birth, and a reddish-purple mass develops at around 4–6 weeks. Deeper lesions appear blueish and superficial lesions red. They tend to grow rapidly in the first year and then undergo a long period of gradual involution, with 50% resolving at 5 years and 70% at 7 years. A variant exists which is present at birth, which will either rapidly involute in the first year (rapidly involuting congenital haemangioma or RICH), or can persist without

involuting at all (non-involuting congenital haemangioma or NICH). Haemangiomas on the hand can mostly be left to spontaneously involute. Indications for treatment are persistent ulceration, bleeding or infection. The basis of treatment of haemangiomas is now medical treatment with propranolol, although surgical excision – when able to be performed simply with minimal scarring and primary closure – may be more straightforward and still has a role.

Pyogenic granuloma (lobular capillary haemangioma)

The terminology of this non-pyogenic, non-granulomatous lesion has become so ingrained that it is unlikely to change. They are friable, pedunculated lesions with a shiny surface (Figure 64.8). They commonly arise at the site of minor trauma, where they grow rapidly and bleed easily. They tend not to resolve spontaneously and should be excised, although multiple treatments with silver nitrate have also been shown to achieve resolution in some cases. Tissue should always be sent for histological analysis as pyogenic granulomas can be mimicked by malignancies such as amelanotic melanoma.

Glomus tumours

The glomus body is a neurovascular structure involved in temperature regulation. Glomus tumours arise from the glomus body and present as blue/red lumps, usually in the fingertips, or as a hard-to-see subungual mass which can cause nail discoloration and ridging. They are more common in women, and often exhibit cold intolerance, severe paroxysmal pain and exquisite tenderness. Love's sign is point tenderness elicited by systematic palpation with a paperclip or similar; Hildreth's test involves inflating a tourniquet – eradication of the pain under tourniquet is highly suggestive of a glomus tumour. A plain X-ray may show a small lytic area in the distal phalanx, and ultrasound and/or MRI can be useful if the diagnosis is still in doubt. Because glomus tumours are multicentric in up to 30% of cases we usually obtain an ultrasound or MRI prior to surgery.

Treatment is by surgical excision. In subungual glomus tumours the nail plate is removed, and the nail fold is reflected



Figure 64.7 Infantile fibromatosis.



Figure 64.8 Pyogenic granuloma.

with the aid of bilateral incisions. A longitudinal incision is made in the germinal matrix to expose the tumour, which is excised to periosteum. Meticulous closure of the germinal matrix is essential to reduce the chance of ridging of the nail.

Malformations

Vascular malformations can be classified by their flow characteristics (into high-flow and low-flow lesions) and subclassified by their histology.

Slow-flow malformations

These can be classified as capillary, lymphatic, venous or combined malformations. They are present at birth but often not obvious. They can become manifest in the first few years of life and will grow commensurately with the patient. In addition to problems specific to their subtype, morbidity in slow-flow lesions of the hand can also be from the bulk and weight of the lesion impairing function, and from a mass effect of the lesion causing peripheral nerve compression.

Capillary malformations exhibit a red discoloration of the skin due to small vessels within the dermis. They are rarely the cause of any functional problem, although very large capillary malformations can lead to overgrowth phenomena in the affected limb. They can simply be observed for growth disturbance and for the possibility of a larger vessel anomaly. The cutaneous discoloration can be addressed with pulsed dye laser therapy.

Venous malformations can vary greatly in size, and often are not limited by tissue planes. They are soft and diffuse, and will decompress with pressure or elevation. Pain is a common feature relating to intralésional thrombus formation. Compression garments can give symptomatic relief by alleviating the heaviness associated with distension of the lesion. Low-dose aspirin in conjunction with compression is useful in preventing intralésional thrombosis. Sclerotherapy can also help to reduce the size of the malformation.

Lymphatic malformations can also vary in size and are sometimes characterized by excessive swelling of the extremity. They do not decompress with elevation, and are frequently complicated by recurrent episodes of infection. Compression and sclerotherapy is also useful for lymphatic malformations.

Surgery for slow-flow lesions is reserved for when conservative measures have failed, and where there is functional impairment, compression neuropathy or other problems due to a mass effect of the lesion. Excision should be staged where the lesions affect multiple anatomical areas.

Fast-flow malformations

These lesions are arterial or arteriovenous malformations. They are warm, occasionally painful lesions that are difficult to decompress. There is often a palpable pulse or thrill. In extremity arteriovenous malformations there can be signs and symptoms of distal ischaemia with shunting through arteriovenous fistulas, and where extensive the patients can exhibit signs of high-output congestive heart failure. There can also be a mass effect

causing peripheral nerve compression. Although the malformation is present at birth, a third of lesions will not be evident until after the age of 10 years.

There is a hormonal influence on the progression of both fast-flow and slow-flow lesions, with around a third to a half of females experiencing an exacerbation in puberty, pregnancy or with use of the contraceptive pill. There is a much smaller rate of progression in puberty in males.

Upper limb fast-flow malformations have been classified into types A, B and C.¹⁶ Type A fast-flow lesions are either single or multiple arteriovenous fistulas, aneurysms or ectasias of the arterial circulation. Type B are arteriovenous anomalies with macro- or micro-fistulas localized to a single axial artery of the limb, hand or digit. Type C malformations are diffuse arteriovenous anomalies with arteriovenous fistulas involving all tissues of the limb. They expand into previously uninvolved areas and often exhibit a distal vascular steal phenomenon.

Doppler ultrasound is useful to differentiate between fast-flow and slow-flow malformations, and MRI is used to identify the extent and exact position of the lesion. Fast-flow lesions can be helped with compression therapy. Arterial embolization can be useful, although failure to eradicate the lesion may lead to more rapid enlargement stimulated by the resulting relative ischaemia.

Where conservative measures do not alleviate symptoms or where there is recurrent bleeding or ulceration, surgical excision or debulking is indicated. It is of critical importance to maintain meticulous haemostasis during the resection, and keep the debulking of large lesions within a well-defined anatomical area. The excision of types B and C fast-flow malformations is aided by preoperative embolization. Type C malformations have a grave prognosis, with the majority of patients eventually having an amputation.

Nerves

Nerve tumours account for about 12% of all benign and 8% of all malignant soft tissue neoplasms.^{17,18} They comprise only 5% of upper extremity tumours in adults and 2% in children.^{19–22} Most are benign. These include pseudotumours such as neuromas, and true tumours, most of which arise from the peripheral nerve sheath and are designated as peripheral nerve sheath tumours (PNSTs). Neurilemmomas (schwannomas) and neurofibromas are most common. The granular cell tumour, neurothekeoma and nerve sheath myxoma may be found in the hand and upper limb less commonly. The perineurioma arises from the perineurium. Other nerve-associated tumours include the fibrolipoma and peripheral nerve sheath ganglion which derive from non-specific neural or peripheral tissue.

Malignant PNSTs arise *de novo* or from malignant degeneration in a benign tumour, and are often associated with neurofibromatosis type 1 (NF1).

Neurilemmoma

These benign PNSTs are the most common of the nerve tumour group, most often in patients of 30–60 years of age, with men and women equally affected.^{23–25} Common sites include the



(a)



(b)

Figure 64.9 (a, b) Neurilemmoma.

brachial plexus and the peripheral nerves of the upper limb. Occasionally there is no apparent nerve association but most are found tethered in the longitudinal axis of the limb in the line of a peripheral nerve. They are usually solitary with non-aggressive features, including slow growth and small size. However, multiple schwannomas may occur, occasionally in association with NF1.¹⁸

Schwannomas are fusiform, eccentrically sited in nerve trunks with apparent entry and exit of small nerve fibres at each end. Pain and neurological symptoms and signs of neurological deficit are unusual, even when the lesions are palpable. In some cases there is a sensory deficit and a positive Tinel's sign.²⁶

The schwannoma and the affected nerve lie within a true capsule: the epineurium. In most instances, at surgery, they can be enucleated from within the epineurium with no nerve deficit unless surrounding nerve fibres are injured at the time of removal (Figures 64.9). Large schwannomas that have been present for a longer time commonly undergo degenerative changes, including cyst formation, calcification, haemorrhage and fibrosis. These are referred to as 'ancient schwannomas'.^{18,27} These do not shell out easily at surgery and damage to nerve fibres is a greater risk.

The histological hallmark of schwannomas is identification of Antoni A and Antoni B regions. The former occur in hypercellular areas with elongated spindle cells with nuclear palisading forming occasional Verocay bodies. Antoni B regions are hypocellular and less organized, containing more myxoid loosely arranged tissue with high water content. The cells stain strongly for S-100 and moderately for Leu-7.²⁶⁻²⁸

Malignant degeneration of a true schwannoma is extremely rare.¹⁸

Neurofibroma

Neurofibroma is a benign PNST of Schwann cell origin, although perineural cells and fibroblasts are also present. It is differentiated from a neurilemmoma in that the tumour mass and the nerve fascicles are intertwined²⁸ (Figure 64.10). Clinical symptoms and signs are as for neurilemmoma.^{19,20,29} Three types are recognized: localized, diffuse and plexiform.^{26,28} Most are of the localized

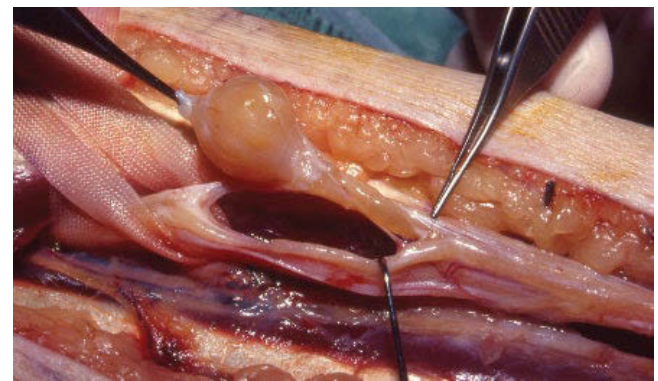


Figure 64.10 Neurofibroma.

variety and usually affect superficial cutaneous nerves, although larger nerves in deep-seated regions may be involved. They are more likely to be multiple and deep-seated when in conjunction with NF1. Diffuse neurofibromas occur in the young, mainly in the head and neck. Plexiform neurofibromas are pathognomonic of NF1 and usually involve a long segment of a major nerve trunk extending into the nerve branches. These may be associated with massive and deforming enlargement of an extremity. The estimated prevalence of malignant transformation varies from 2 to 29%, with a sudden increase in size of a previously stable neurofibroma and the presence of pain and neurological defects leading to suspicion of malignant change.^{17,18,28}

A localized solitary neurofibroma is composed of interlacing fascicles; they do not contain Antoni A and B regions and myxoid areas and degenerative regions are not as prominent as they are in schwannoma. They stain less strongly for S-100 protein and Leu-7 than do schwannomas.^{17,18,28}

Granular cell tumour

Between 10 and 20% of these are found in the upper extremity, appearing as small, non-tender, subcutaneous nodules; 10–25% are multifocal. They may be found in association with a nerve trunk, a digital nerve or with no nerve association. They may or

may not be intimately associated with nerve fascicles. They stain moderately for S-100 and Leu-7, suggesting that their origin is the Schwann cell.^{28,30–34} Malignant change is reported as being less than 3%.^{30,35} Following biopsy, a granular cell tumour may be excised or not. However, malignant change is rare and resection may result in neurological deficit requiring reconstruction.

Perineurioma

The soft tissue perineurioma has been recently recognized as a distinct benign peripheral nerve tumour, arising from perineural cells.³⁶ There are two types, usually presenting in young adults. In both the cells do not stain for S-100 and Leu-7 proteins but they do stain for epithelial membrane antigen (EMA).³⁷ The soft tissue perineuriomas arise as slow-growing painless nodules in the trunk or extremities, often without a clear association with a nerve. Some report that tumours of perineural origin with malignant change may arise from benign soft tissue perineuromas, but this is not proven.^{38,39}

The intraneural perineurioma (localized hypertrophic neuropathy) is a benign neoplasm composed of perineural cells surrounding nerve fibres and is restricted to the boundaries of peripheral nerves. A motor deficit of nerve function is common, often with only mild sensory involvement. It occurs most commonly in the sciatic nerve but presents equally in upper and lower limbs. In approximately 17% of cases more than one nerve is involved.⁴⁰ These tumours are static or slowly progressive. Malignant degeneration has not been reported. Treatment may be indicated for a nerve deficit. Resection may be difficult because of the length of nerve involved. Nerve grafting, nerve transfers, tendon transfers or joint stabilizations may be considered following resection.

Fibrolipoma

This is an uncommon tumour-like lipomatous process involving peripheral nerves and their branches in which increased fatty tissue is interspersed among thick nerve bundles.^{18,41,42} Males and females are equally affected with presentation typical before 30 years of age, often at birth or early childhood. The median nerve is most commonly affected. Clinical symptoms are associated with slow growth of the soft tissue tumour, often in association with symptoms of a compression neuropathy. In 27–60% of cases the nerve lesion is associated with macrodactyly – macrodystrophica fibrolipomatosis – often involving the index and middle fingers.¹⁸ Multiple digits may be affected. The precise relationship between the fibrolipomatosis and the macrodactyly has not been established.

The affected nerve is diffusely enlarged. Currently the tumour is designated as a fibrolipomatosis of a nerve rather than arising from neural tissue.⁴¹ Those with a fibrolipomatosis hamartoma of nerve, most often the median nerve, may respond to a limited soft tissue excision and carpal tunnel release as appropriate. Recurrence of symptoms may occur.

Other benign nerve tumours

Neurothekeoma and nerve sheath myxoma both present as isolated, small dermal lesions in young adults. They may arise, at least in part, from the Schwann or perineural cell. They do not undergo malignant degeneration and excisional biopsy is usually curative.^{43–49}

Another neuromuscular hamartoma (the benign triton tumour) is exceedingly rare but may involve the brachial plexus or the sciatic nerve and other large nerve trunks of infants and young children. They are a multilobed proliferation of mature skeletal muscle and neural elements.²⁸

A Pacinian neuroma is a proliferation of Pacinian corpuscles, often presenting as a painful nodule on the digit.²⁸

A nerve sheath ganglion may arise from within the epineurium or from adjacent joints, such as the tibiofibular joint. A compression neuropathy may result.^{18,28}

Malignant peripheral nerve sheath tumours

These account for 5–10% of all soft tissue sarcomas, usually affecting adults aged 20–50 years, with an equal sex distribution. Twenty per cent occur in the upper extremities.⁵⁰ They constitute approximately 3% of all malignant tumours of the hand.^{51,52} These lesions are associated with NF1 in 25–70% of cases and most commonly involve major nerve trunks.¹⁸ The incidence of malignant PNST in patients with NF1 has been reported as 2–5%, although a recent longitudinal study suggests a lifetime risk as high as 13%, particularly for plexiform neurofibromas.^{51,52} Malignant change from a benign schwannoma or a solitary neurofibroma is rare. Malignant change can occur secondary to previous radiation therapy.

These are high-grade sarcomas. Presentation is with pain, increasing size of the tumour and neurological symptoms of motor weakness and sensory deficits. Regrettably the symptoms such as pain, weakness and numbness may occur late.

The tumour spreads along the affected nerve with thickening of epineurium and perineurium proximal and distal to the mass (Figure 64.11). The tumour cells are arranged in fascicles and areas of haemorrhage and necrosis are frequent.

Metastasis is through haematogenous spread, although perineural extension and lymphatic spread also occur.^{53–56}

Malignant peripheral nerve sheath tumours are reactive for S-100 protein in 50% of cases. Perineural cell differentiation may sometimes be seen and this could confer a better prognosis.

Investigations

The modalities of ultrasonography and MRI are particularly helpful in the characterization of nerve tumours and may assist in indicating the possibility of malignancy.²⁶ These may demonstrate: that the tumour is in the region of a major nerve; there may be evidence of tubular entry and exit of nerve, easier to detect in lesions affecting large deep nerves; fusiform shape of the tumour; a split-fat sign indicative of a ring of fat often present about neurogenic neoplasms; atrophic muscle changes with increased fat content or increased size of muscle, best seen on

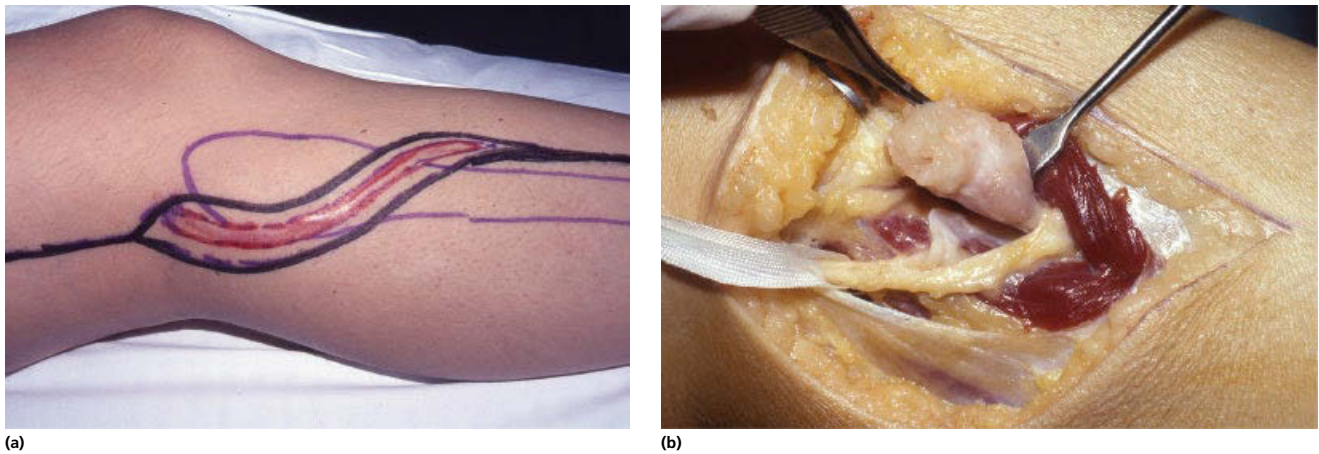


Figure 64.11 (a, b) Neurosarcoma of the peroneal nerve.

T1 weighted MR images but also can be evaluated with a finding of increased echogenicity of muscle on ultrasound; and the presence of target sign and fascicular appearance – the former corresponds to fibrous tissue in the centre of the nerve with myxoid tissue peripherally, the latter represents multiple small ring-like structures corresponding to fascicular bundles.

In schwannomas, the mass is more likely to be eccentric and separable from the nerve, whereas in neurofibromas the two structures are indistinguishable. The target sign is more characteristic of neurofibroma and the fascicular sign is perhaps more commonly associated with schwannomas. Schwannomas are more frequently associated with central necrosis, cyst formation and calcification, particularly in long-standing schwannomas (ancient schwannoma). Central enhancement after gadolinium administration has been reported more frequently with neurofibromas.

Malignant tumours may demonstrate heterogeneous MRI signals with less well-defined margins; target and fascicular signs are rare; the enhancement pattern is predominantly peripheral and heterogeneous; a duplex ultrasound may exhibit sarcoma type anarchic hypervascularisation rather than the peripheral hypervascular changes of a benign tumour. Scintigraphic gallium 67 citrate imaging reveals a significant uptake and the positron emission tomography (PET) scan is positive.

It should however be emphasized that MRI differentiation between benign and malignant tumours, and among benign tumours such as schwannoma and neurofibroma, is not a reliable diagnostic tool. Formal biopsy remains the only reliable method of diagnosis.

Management

Surgical decision making and surgical technique must adhere to the principles of tumour surgery described in the introduction. The macroscopic appearance of a schwannoma is usually diagnostic and, in most instances, the tumour may be peeled from the nerve fibres with minimal or no neural damage and no subsequent deficit. This is not so for the ancient schwannoma,

when nerve fibres may be intimately involved with cystic areas which insinuate between and involve fascicles. Even when dissecting a typical schwannoma there may be an entering fascicle or exiting fascicle which is sacrificed, but usually with no discernible postoperative deficit. If the tumour does not dissect easily from the nerve, the surgeon must consider whether this might be a neurofibroma or a malignant lesion. A frozen section biopsy assists in making the diagnosis. Resection of a neurofibroma may leave some nerve deficit or amount to a debulking procedure with tumour remaining. If there is doubt about malignancy then a formal biopsy alone is appropriate. Subsequently, if the diagnosis is confirmed as a neurofibroma, the surgeon will discuss with the patient the advantages and disadvantages of further surgery, the possibility of nerve resection and grafting, and the possible consequences of leaving the tumour *in situ* without proceeding to secondary surgery.

If malignant, then a wide excision, preferably compartmental, is appropriate. However, spread along the nerve is possible and there may be multiple lesions. Limb-sparing surgery is most commonly advised. Compartmental excision is difficult in the hand but more applicable to the forearm. Vital vessels should be retained or reconstructed. Nerves may be reconstructed in a secondary manner with grafting or nerve transfers. Tendon transfers and joint stabilizations may be appropriate. Limb amputation may be necessary.

Local recurrence and metastasis are common. Radiotherapy may be helpful in controlling local disease. No chemotherapeutic regime has proven to be effective.^{50,53,57} Metastasis is by the way of haematogenous spread although perineural extension and embolization of lymphatics may also occur.²⁸ Those patients with neurofibromatosis and tumour size greater than 5 cm have a poorer prognosis. Ducatman *et al.* reported a median survival of 3 years from the time of diagnosis, with a 23% survival rate at 10 years.⁵³

Soft tissue sarcomas

Approximately 5% of soft tissue sarcomas occur in the hand. The commonest histological subtypes are epithelioid sarcoma, synovial sarcoma and malignant fibrous histiocytoma. Rhabdomyosarcoma

is a more common sarcoma in children. Patients typically present with a history of a rapidly enlarging painless mass in the hand.

When dealing with a suspicious soft tissue mass, tissue diagnosis is essential prior to definitive management. The principles of biopsy of soft tissue malignancies outlined above should be adhered to in obtaining tissue for histopathology. Patients should have MRI prior to biopsy, and if sarcoma is confirmed, it should be staged with imaging of the chest and possibly with PET scanning to identify metastases.

The traditional treatment of amputation for soft tissue hand sarcomas has given way to limb-sparing or hand-sparing surgery in many cases. The standard of care is now wide en-bloc excision with clear margins, followed by soft tissue reconstruction and postoperative radiotherapy and chemotherapy determined by the histological subtype. This approach has equivalent survival figures to amputation.⁵⁸ The histological grade of the tumour is the greatest determinant of survival, and guides the surgical and adjuvant management. Salvage of vital structures is a secondary goal to adequate resection of the tumour.

The goal of treatment is complete removal of the tumour with a surrounding cuff of normal tissue. Excision with a margin of 2–3 cm of normal tissue has been advocated. Radical excision with these margins in the hand will often necessitate extensive soft tissue reconstruction if any function is to be salvaged, and often a two-team approach with resecting and reconstructing teams will be useful. Functional outcomes are generally dependent on the amount of native tissue left behind, but even double ray amputations can leave a hand that is of superior function to a prosthesis.⁵⁹

Epithelioid sarcomas are epithelial tumours with a necrotic central area. They are most common in young adults, and are unusual in that they frequently exhibit lymphatic metastasis. Examination of the regional lymph node basins is therefore mandatory, and SLNB has been advocated. Synovial sarcoma is also most commonly a condition of young adults. The tumours are of epithelial and spindle cells. It usually presents with a soft tissue mass close to joints or bursae. There is some benefit to neoadjuvant chemotherapy in reducing the bulk of the tumours and increasing the feasibility of limb salvage. Rhabdomyosarcomas are mainly seen in children and adolescents. Preoperative and postoperative chemotherapy form part of the treatment protocol to reduce local recurrence and increase survival.

Bone tumours

Benign bone tumours

Enchondroma

Enchondromas are common, comprising up to 90% of all bone tumours within the hand. The proximal phalanges and metacarpals are most often affected. More than 50% of all enchondromas are located within the long bones of the hand.

These tumours frequently present as a pathological fracture. They can also manifest as a progressive, usually painless swelling, or as incidental findings on plain radiographs.

On plain radiographs matrix calcification suggests a tumour of cartilage. The calcification tends to lie in the centre of the lesion. There is a well-defined zone of transition. The cortical bone can be thinned and may appear to be scalloped from within. The lesion lies centrally within the metaphysis.

Treatment is with observation if the lesion is small and asymptomatic; if causing problems the tumour can be curetted from the bone, preferably via a midlateral incision. It is prudent to obtain radiographs intraoperatively to confirm that the curette has probed the limits of the tumour cavity in all directions. Bone graft is not necessary in most cases,⁶⁰ but judgement is used if the cortex is excessively thinned and the bone appears particularly fragile.

In the event of trauma the fracture is treated on its merits and once healed residual tumour can be observed or removed. Bone graft and internal fixation may be necessary when treating the fracture. Whenever graft is obtained, it should be via fresh instruments to avoid contaminating the graft site.

When the curetted specimen is sent to the laboratory, particularly in the setting of trauma it is necessary to provide an adequate history to the pathologist as it can be difficult to distinguish between an enchondroma and a low-grade chondrosarcoma (in the setting of elective curettage and no fracture) and between an enchondroma and an osteosarcoma (in the posttraumatic setting when osteoid is being produced). Following removal of the lesion some authorities recommend surveillance X-rays postoperatively.¹

Malignant change in a solitary enchondroma has been described but is very rare.

Multiple enchondroma

Ollier's disease

The name of Louis Ollier (1830–1900), a French surgeon, is attached to an acquired developmental abnormality characterized by multiple enchondromas involving multiple bones, and which can involve any or all of the epiphysis, metaphysis or diaphysis (Figure 64.12).

These lesions are of note for their propensity to cause significant growth disturbance, shortening of the bones and joint disruption, and for their higher potential for malignant change. The propensity for malignant change is less for the hand than for more central lesions. As for solitary enchondromas, growth usually ceases with skeletal maturity.

Maffucci's syndrome

Angelo Maffucci (1847–1903), an Italian pathologist, described a syndrome characterized by multiple enchondromas and haemangiomas. It has an even higher rate of malignant transformation than Ollier's syndrome and can be associated with very marked growth disturbance. Chondrosarcomas and malignant vascular tumours may both occur; furthermore these patients are prone to tumours of other systems including the thyroid, breast, pancreas and liver, leading to some authorities estimating an overall rate of malignancy approaching 100%.⁶¹



Figure 64.12 Multiple enchondromas.

In both syndromes treatment is targeted at lesions causing functional problems; it is usually not possible or sensible to attempt to remove all lesions. Children with these syndromes may need to endure many operations over the years. A high index of suspicion for malignant change must be maintained.

Osteochondroma (also known as cartilage capped exostosis)

An osteochondroma is a relatively common bone tumour characterized by a pedunculated or sessile outgrowth from the bone which has both cortex and cancellous bone in continuity with the normal bone, and which is covered by a cartilaginous cap, which in turn is covered by a fibrous band.⁶² It is caused by a defect in the perichondral ring of the physis; the physal cartilage slips to an aberrant position and there continues to grow. Being produced by the physis, growth should stop with skeletal maturity.

Most cases present by the late teens and lesions are more commonly seen in males. Around 5% of all osteochondromas are seen in the hand.

Radiographs demonstrate the characteristics of cortical and medullary continuity. Pedunculated lesions are usually directed

towards the diaphysis of the bone. Because radiographs do not show the cartilaginous cap, which can be 2–3 cm in thickness,⁶² the usual tumour is significantly larger clinically than on X-ray. MRI scans demonstrate the cartilaginous caps well and are of use when planning surgery in anatomically complex regions such as around the brachial plexus.

These lesions usually present as long-standing masses, which slowly enlarge. They can cause pain by direct pressure upon the skin or by impinging on adjacent nerves or tendons. A bursa may develop over a prominent osteochondroma.

Transformation to a chondrosarcoma is rare but has been described and is heralded by continued growth after skeletal maturity has been reached.

Treatment consists of excision of the lesion through the base of the stalk, and is usually reserved for symptomatic lesions.

Multiple hereditary exostosis (diaphyseal aclasis, osteochondromatosis)

This is an autosomal dominant inherited condition characterized by multiple lesions, usually picked up within the first years of life. The chief points of interest in this condition are that malignant change is seen at a much higher rate than in solitary osteochondromas, and that deformities of the forearm are common because of growth disturbance and/or pressure from lesions within this complex two-bone system.

Giant cell tumours

Giant cell tumours make up around 20% of benign bone tumours, are usually seen in the third to fourth decades and are slightly more common in women (bony tumours as a whole are more common in men).

In the upper limb giant cell tumours predominantly affect the distal radius, which is the third most common site for a giant cell tumour after the distal femur and proximal tibia. It is very rare in the carpus or phalanges. Multicentric giant cell tumours are uncommon but do occur more frequently with the small bones of the hand (10% versus 1% for long bones).

The lesions are benign but local recurrence is common. They usually present with pain and swelling, and occasionally with pathological fracture. Distant metastasis is possible but seen in less than 10%.⁶³ Late sarcomatous degeneration has occurred but most cases have been exposed to radiotherapy prior to malignant change.

On plain radiographs the appearance is characteristic; the lesion involves the epiphysis, extends to subchondral bone and has a well-defined but non-sclerotic zone of transition. Matrix calcification is not seen. More aggressive tumours may expand and penetrate the bone to involve the soft tissues. CT scans may aid in the evaluation of tumours of the carpus or metacarpus (Figure 64.13).

The main differential diagnosis is of chondroblastoma, a benign cartilage tumour which also involves the epiphysis. This tumour occurs in younger patients prior to closure of the physes, and may display matrix calcification on plain X-rays. It is also much less common in the radius than a giant cell tumour.



Figure 64.13 Giant cell tumour distal radius.

Other differential diagnoses include aneurysmal bone cyst, osteomyelitis and brown tumours of hyperparathyroidism.

Giant cell tumours are staged according to Campanacci's scheme.⁶⁴ Grade I lesions have a cortex that may be thinned but is not deformed. Grade II lesions have an expanded cortex. Grade III lesions have a poorly defined border and can extend into the soft tissues.

In the radius, wide intralesional excision is preferred for grade I and II lesions. The margin is then extended with an adjuvant therapy such as bone cement, liquid nitrogen or phenol. Without adjuvant therapy recurrence is almost inevitable. Grade III lesions are treated with en-bloc excision and reconstitution with anatomically specific allograft or free vascularized fibula grafting. Carpal and tubular hand bones are best treated with resection if possible, as the rates of recurrence are higher for the small bones of the hand – around 20% when adjuvant therapy is used.⁶⁵ Recurrent giant cell tumour is thought to have a higher malignant potential.

Aneurysmal bone cyst

Aneurysmal bone cysts are benign expansile lytic lesions seen in younger patients and typically involving the metacarpals. Radiographs show cortical thinning and expansion and MRI scans show pathognomonic fluid levels within the lesion. The main differential diagnosis is giant cell tumour, and the treatment is similar, with intralesional curettage and adjuvant therapy to extend margins using liquid nitrogen or bone cement;

for larger lesions and/or those extending into soft tissue resection and reconstruction is necessary.

Osteoid osteoma

Osteoid osteomas, described by Jaffe in 1935, are benign tumours composed of central calcified osteoid surrounded by a vascularized stroma and then a sclerotic periphery of reactive bone. Prostaglandins are secreted by the tumour. Tumours with the same pathology more than 2 cm in diameter are known as osteoblastomas.

Hand tumours represent 10% of all osteoid osteomas. The tumours typically occur in the second or third decade and are twice as common in males.

Pain is a characteristic and prominent feature of osteoid osteoma and is said to be worse at night, and be very responsive to aspirin, although Marcuzzi found that only just under half his patients had pain relief from aspirin.⁶⁶ Osteoid osteomas are often implicated as a cause of unexplained or chronic pain (as are glomus tumours). They can result in significant enlargement of the affected bone and lie in the differential diagnosis of macrodactyly.

They may occur in any of the bones of the hand with a slight predilection for the phalanges. The classic radiological appearance is of cortical thickening perhaps with a central lytic area, containing a small central nidus of bone, but initial radiographs are often unremarkable.⁶⁶ The lesions are often better seen on a CT scan, and three-phase bone scan is particularly sensitive for the tumour (Figure 64.14).

Treatment can be with prolonged aspirin use, but it may take several years for the lesion to burn out and surgical treatment is usually preferred. Radiofrequency ablation is used in other parts of the body but heats up the surrounding structures and may be less suitable for the hand with its closely packed vital structures. Surgical removal with or without grafting using a burr down technique is preferred in the hand and is highly successful.⁶⁶

Malignant bone tumours

These are very rare; chondrosarcomas are seen most often, whereas osteosarcomas and Ewing's tumours are extremely uncommon in the hand. Bone sarcomas present with pain, swelling, pathological fractures and, in the case of Ewing's sarcoma, systemic symptoms. Adequate staging, both local and systemic, is critical in developing a management strategy, and treatment must involve a musculoskeletal oncology service.

Chondrosarcoma

These are the most common malignant bone tumours in the hand. Chondrosarcomas can arise *de novo* or from malignant change of a solitary enchondroma (very rare) or less uncommonly from the enchondromas of Ollier's or Maffucci's syndromes.

Hand chondrosarcomas make up 2% of all chondrosarcomas.⁶² They occur in an older age group with most patients in the sixth or later decade. This tumour usually involves the proximal phalanges and metacarpals.

The lesions are typically slow growing and expansile, and have areas of matrix calcification and lysis. The cortex may be



Figure 64.14 Osteoid osteoma with sclerosis of proximal phalanx and nidus indicated by long arrow.

disrupted and there may be an associated soft tissue mass (Figure 64.15).

Treatment is by wide excision. Chondromas are typically not radio- or chemo-sensitive, and metastasis is late; if it occurs, the lungs are most often affected.

Osteosarcoma

These lesions are even rarer than chondrosarcomas. They tend to occur in an older age group than osteosarcomas elsewhere in the body and present with a rapidly enlarging painful mass.

Plain radiographs may show areas of lysis and/or bony destruction associated with periosteal new bone formation.

Treatment is with radical excision usually combined with chemotherapy. Some oncologists use neoadjuvant therapy, beginning chemotherapy after the incisional biopsy but prior to definitive treatment in order to shrink the tumour, diminish the reactive zone around the tumour and to allow assessment of the tumour response to chemotherapy.

Ewing's sarcoma

Ewing's sarcoma presents in younger patients than chondro- or osteosarcoma. The metacarpals and phalanges are most commonly involved. Radiographs demonstrate a destructive, expansile lesion, often with a soft tissue extension that is confirmed on MRI. The presentation often resembles an infection, with rapidly increasing swelling, pain and systemic features, and misdiagnosis as osteomyelitis is possible. Treatment involves incisional biopsy, then neoadjuvant chemotherapy in conjunction with wide resection.



(a)



(b)

Figure 64.15 (a) Chondrosarcoma arising from distal radius. (b) Excision of distal radius and creation of one-bone forearm.

Metastatic disease

Metastasis to the hand is rare. It typically involves the terminal phalanges. The most common malignancy metastasizing to the hand arises from the lung. Hand metastasis occurs late in the disease and the prognosis is dismal, with average life expectancy of nine months post diagnosis.

The goal of management is symptom control, and amputation through healthy tissue proximal to the lesion is usual.

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PART VIII

Aesthetic surgery

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CHAPTER 65

Facial anatomy and ageing

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Introduction

Fundamental to safety when operating in the face is a firm grounding in the principles on which the facial soft tissue layers are constructed. This is also important because the pathogenesis of facial ageing is explained on an anatomical basis, particularly the variations in the onset and outcome of ageing seen in different individuals. This is most important in addressing the overriding concern of the course of the facial nerve branches. An anatomical approach to surgical rejuvenation of the face provides the way to obtaining a 'natural' result that is lasting and with minimal morbidity.

Regions of the face

The traditional approach to assessing the face is to consider the face in thirds (upper, middle and lower thirds). Although useful, this approach limits conceptualization as it is not based on the function of the face. From a functional perspective, the face has an anterior aspect and a lateral aspect. The anterior face is highly evolved for communication and facial expression. In contrast, the lateral face predominantly covers the structures of mastication. A vertical line descending from the lateral orbital rim is the approximate division between the anterior and lateral zones of the face. Internally, a series of facial retaining ligaments are strategically located along this line to demarcate the anterior from the lateral face (Figure 65.1). The mimetic muscles of the face are located in the superficial fascia of the anterior face, mostly around the eyes and the mouth. This highly mobile area of the face is designed to allow fine movement and is prone to develop laxity with ageing. In contrast, the lateral face is relatively immobile as it overlies the structures to do with mastication, the temporalis, masseter, the parotid gland and its duct, all located deep to the deep fascia. The only superficial muscle in the lateral face is the platysma in the lower third, which extends to the level of the oral commissure.

Importantly, the soft tissues of the anterior face are subdivided into two parts: that which overlies the skeleton and the

larger part that comprises the highly specialized sphincters overlying the bony cavities.^{1,2} Where the soft tissues overlie the orbital and oral cavities, they are modified as there is no deep fascial layer for support. Accordingly, support does not come from within the space immediately below, but from the rim of the cavities. The transitions between these areas, although not seen in youth, become increasingly evident with ageing.

Surgical anatomy of the face, superficial musculoaponeurotic system, facial spaces and retaining ligaments

The five concentric layers of the face are:^{1,2} (1) skin; (2) subcutaneous tissue; (3) musculoaponeurotic layer; (4) areola tissue; and (5) deep fascia. This five-layered arrangement is most clearly seen in the scalp and forehead due to expansion of the soft tissues as a result of evolutionary expansion of the underlying cranial vault necessary to accommodate the highly developed frontal lobe in humans. It is an excellent place to study the principles of the layered anatomy (Figure 65.2). Layer 4 (the loose areolar tissue) is the layer that allows the superficial fascia (defined as the composite flap of layers 1 through 3) to glide over the deep fascia (layer 5). The simplified anatomy over the scalp gives the basic prototype of layer 4.³ There are no structures crossing this plane, which is essentially an avascular potential space. At the boundaries of the scalp along the superior temporal line and across the supraorbital rim, the scalp and the forehead are firmly anchored by ligamentous attachments. Vital structures such as nerves and vessels are always located in close proximity to the retaining ligaments. In the face proper, although the principles of construction remain the same, there is considerably greater complexity. This is due to the compaction resulting from the absence of forward projection of the midface, as occurs in other species, and the predominance of the orbital and oral cavities that limit the availability of a bony platform for attachment of ligaments and muscles. To secure this five layers to the facial skeleton, an elaborate system of retaining ligaments

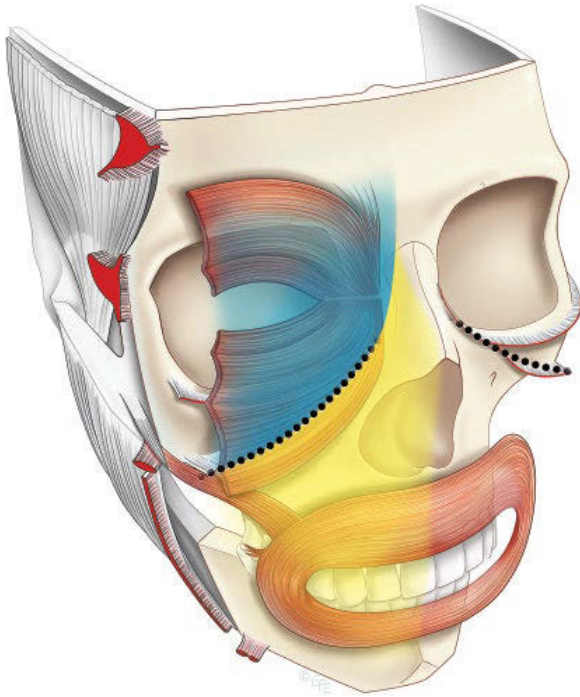


Figure 65.1 Regions of the face: The mobile anterior face is functionally adapted for facial expressions and is separated from the relatively fixed lateral face (shaded) which overlies masticatory structures. A vertical line of retaining ligaments (red) separates the anterior and lateral face. These ligaments are, from above: temporal, lateral orbital, zygomatic, masseteric and mandibular ligaments. In the anterior face, the midcheek is split obliquely into two separate functional parts by the midcheek groove (dotted line) related to two cavities: the periorbital part above (blue) and the perioral part below (yellow).

bind the dermis to the skeleton, the components of this system pass through all layers (Figures 65.3 and 65.4).⁴⁻⁶

Layer 1 – skin

The epidermis is a cell-rich layer composed mainly of differentiating keratinocytes and a smaller number of pigment-producing melanocytes and antigen-presenting Langerhans cells. A rich vascular plexus is an important component of the dermis. The thickness of the dermis relates to its function and tends to be inversely proportionate to its mobility. The dermis is thinnest in the eyelids and thickest over the forehead and the nasal tip. The thinner the dermis, the more susceptible it is to qualitative deterioration ageing changes.

Layer 2 – subcutaneous tissue

The subcutaneous layer has two components: the subcutaneous fat, which provides volume, and the fibrous retinacular cutis that binds the dermis to the underlying superficial musculoaponeurotic system (SMAS) of the face.²² Of note, the retinacular cutis is the name given to that portion of the retaining ligament that passes through the subcutaneous tissues. The amount, proportion and arrangement of each component vary in different

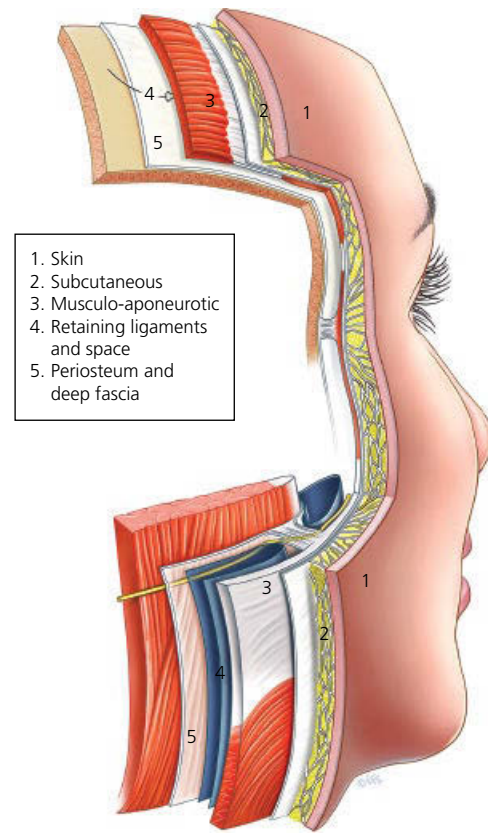


Figure 65.2 The face is constructed in five basic layers. This five-layered construct is most evident in the scalp but also exists in the rest of the face, with significant modification and compaction for functional adaptation. Layer 4 is the most significantly modified layer, with alternating facial soft tissue spaces and retaining ligaments. Facial nerve branches also transition from deep to superficial in association with the retaining ligaments through layer 4.

regions of the face. In the scalp, the subcutaneous layer has uniform thickness and consistency of fixation to the overlying dermis. In contrast, in the face proper, the subcutaneous layer has significant variation in thickness and attachments. In specialized areas such as the eyelids and lips, this layer is significantly compacted such that fat may appear non-existent. In other areas, such as the nasolabial segment, it is very thick.⁴ In areas with thick subcutaneous tissue, the retinacular cutis lengthens significantly, predisposing its fibres to weakening and distension with ageing.

Within the subcutaneous tissue, the overall attachment to the overlying dermis is stronger and denser than the attachment to the underlying SMAS.²² This is a result of the tree-like arrangement of the retinacular cutis fibres (see Figure 65.3), with fewer but thicker fibres deep as it rises through the SMAS that progressively divide into multiple fine microligaments as they reach the dermis. This explains why it is easier to perform subcutaneous dissection in the deeper subcutaneous level (just on the surface of the underlying SMAS) than more superficially nearer the dermis, as there are fewer retinacular cutis fibres and the

Figure 65.3 The retaining ligaments of the face can be likened to a tree. The ligaments attach the soft tissues to the facial skeleton or deep muscle fascia, passing through all five layers of the soft tissues. It fans out in a series of branches and inserts into the dermis. At different levels of dissection, it is given different names, such as the retinacular cutis in the subcutaneous layer and ligaments in the sub-SMAS level.

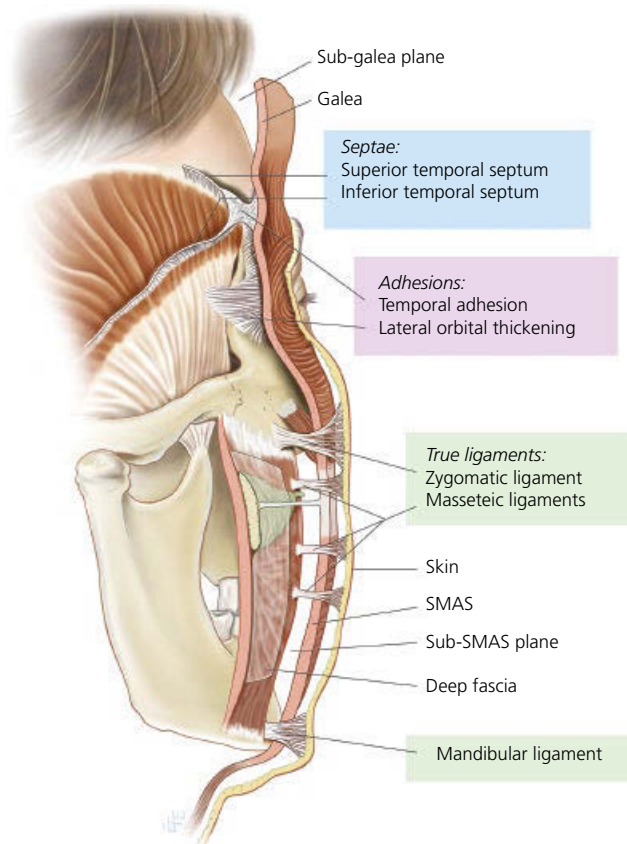
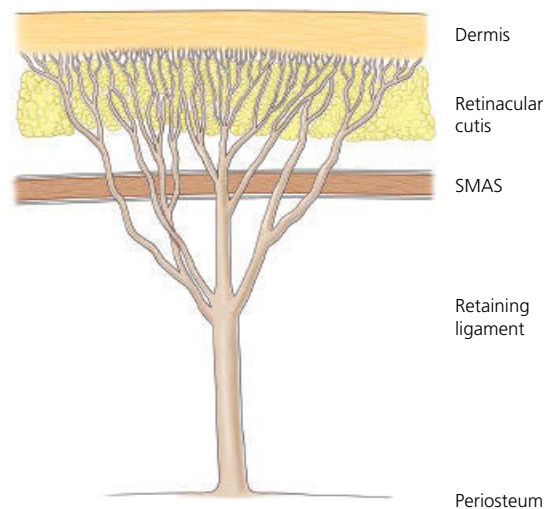


Figure 65.4 Three morphological forms of retaining ligaments of the face.

subcutaneous fat here does not attach directly to the outer surface of the underlying SMAS.

Furthermore, the retinacular cutis fibres are not uniform across the face, but vary in orientation and density according to the anatomy of the underlying deeper structures. As will be apparent when the anatomy of the underlying layer 4 is described, at the location of the retaining ligaments, the vertically orientated

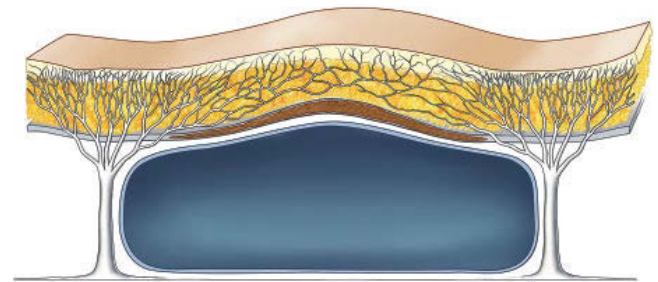


Figure 65.5 The density and strength of the retinacular cutis fibres in the subcutaneous layer vary in different areas of the face. Where they overlie the retaining ligaments, the fibres are denser and orientated more vertically. In these areas, sharp release is usually necessary to raise a subcutaneous flap. In contrast, in areas overlying a space, the fibres are less dense and orientated more horizontally. Here it is relatively easy to elevate a subcutaneous facelift flap.

retinacular cutis fibres are the most dense and are the most effective in supporting for the overlying soft tissues and, in so doing, form boundaries that compartmentalize the subcutaneous fat. These areas, such as the so-called *McGregor's patch* over the body of the zygoma, often require sharp release to mobilize. In between these retaining ligaments in layer 4 are located the soft tissue spaces of the face that facilitate the mobility of the superficial fascia over the deep fascia. Where the subcutaneous fat overlies a space, the retinacular fibres are less dense and orientated more horizontally, as a result of which, the tissues tend to separate with relative ease, often with just simple blunt finger dissection (Figure 65.5). This variation in the density and orientation of the retinacular cutis fibres in the subcutaneous fat is the anatomical basis for the compartmentalization of the subcutaneous fat into discrete compartments, only recently described.⁷

Layer 3 – musculoaponeurotic layer

The muscles of facial expression are unique and fundamentally different from skeletal muscles beneath the deep fascia because they are situated within the superficial fascia and they move the

soft tissues of which they are a part. All muscles of facial expression have either all or the majority of their course within layer 3 and they are predominantly located over and around the orbital and oral cavities. In the prototype scalp, the occipital-frontalis moves the overlying soft tissue of the forehead, while its under-surface glides over the subgaleal aponeurotic space (layer 4). Layer 3 is continuous over the entire face, although for descriptive purposes, different names are given to certain parts according to the superficial muscle within. It is called the galea over the scalp, the temporoparietal (superficial temporal) fascia over the temple, the orbicularis fascia in the periorbital region, the SMAS over the mid and lower face and platysma in the neck.⁸

Within layer 3, the facial muscles themselves have a layered configuration, with the broad, flat muscles forming the superficial layer that covers the anterior aspect of the face. The frontalis covers the upper, orbicularis oculi the middle and the platysma the lower thirds, respectively. The muscles of this layer have minimal direct attachment to the bone, stabilized to the skeleton at their periphery indirectly by the vertically orientated retaining ligaments as noted earlier. The frontalis is fixed along the superior temporal line by the superior temporal septum, the orbicularis oculi laterally by the lateral orbital thickening and the main zygomatic ligament at its inferolateral border and the platysma at its upper border by the lower masseteric ligament. The deeper muscles in layer 3 provide greater functional control of the sphincters over the bony cavities. For the upper third, these are the corrugators and procerus and around the oral cavity, the elevators (zygomaticus major and minor, levator labii superioris, levator angularis oris) and levator angul oris and the depressors (depressor angularis oris, depressor labii inferioris) around the oral sphincter and the mentalis.

Layer 4

Layer 4 is the plane in which dissection is performed in sub-SMAS facelifts. It is an area of significant complexity and contains the following structures: (1) soft tissue spaces, (2) retaining ligaments, (3) deep layers of the intrinsic muscles passing from their bone attachment to their more superficial soft tissue origin, and (4) facial nerve branches, passing from deep to superficial. Functionally, a series of soft tissue spaces exist in layer 4 to allow independent movement of the periorbital and perioral muscle of facial expressions over the deep fascia responsible for mastication directly beneath the muscles of facial expression. The retaining ligaments of the face are strategically placed within and reinforce the boundaries between the soft tissue spaces (Figure 65.6).¹

In the lateral face, immediately in front of the ear, extending 25–30 mm forward of the ear cartilage to the posterior border of the platysma, is a diffuse area of ligamentous attachment, described by Furnas as the platysma auricular fascia (PAF). As no facial expression occurs here, the dermis, subcutaneous tissue, SMAS and the underlying parotid capsule (layers 1–5) are bound together as an area of diffuse retaining ligament. Layer 4 is reduced to a layer of fusion here, without a soft tissue

space. In contrast, in the anterior face where there is considerable movement over and around the bony cavities, the ligaments are significantly compacted and arranged around the edges of the bony cavities. These boundaries provide the last position where there is underlying deep fascia for the mobile shutters of the lids and lips to be supported. Importantly for the surgeon, the retaining ligaments also act as transition points for the facial nerve branches to pass from deep to superficial on their way to innervate their target muscles.

Soft tissue spaces of the face are in two forms: (1) those overlying bony cavities, such as the preseptal space of the eyelid over the orbit and the vestibule of the oral cavity, under the lips and the lower nasolabial segment of the cheek and (2) those overlying bone, where the spaces allow the overlying superficial fascia to glide freely over the underlying bone.

Layer 5

The deep fascia, the deepest soft tissue layer of the face, is formed by the periosteum where it overlies bone. Over the lateral face, where the muscles of mastication (temporalis and masseter) overlie the bone, the deep fascia is instead the fascial covering of the muscles, the deep temporal fascia and masseteric fascia above and below the zygomatic arch, respectively. The parotid fascia is also part of the deep fascia. The investing deep cervical fascia is the corresponding layer in the neck where it covers the supraomohyoid muscles and splits to form the submandibular space that contains the submandibular gland. The deep fascia, although thin, is tough and unyielding and gives attachment to the retaining ligaments of the face. In the mobile shutters over the bony cavities,

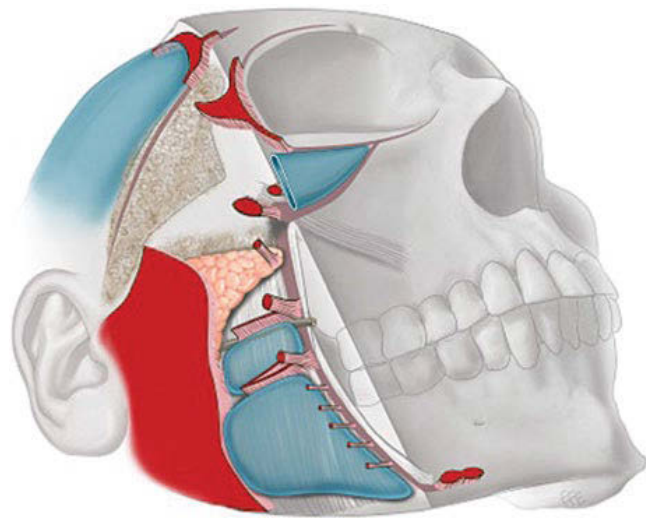


Figure 65.6 Topographical anatomy of layer 4 over the lateral face. Spaces (blue), ligaments (red) and the areas of important anatomy (stippled). The largest area of ligamentous attachment, the platysma-auricular fascia (PAF), dominates the posterior part of level 4 at the least mobile part of the face. The lateral face transitions into the anterior face at the vertical line of retaining ligaments. Immediately above and below the arch of the zygoma are the triangular-shaped areas that contain the important anatomy proceeding from the lateral into the anterior face.

the deep fascia is absent, being replaced by a mobile lining derived from the cavities, that of the conjunctiva or oral mucosa.

Anatomy over the cavities in the skeleton

The general pattern of the five-layered anatomy is modified where the orbital, oral and nasal cavities are present over the anterior face (Figure 65.7). Only the outer three-layer composite continues into the soft tissue overlying the cavities. The SMAS layer within this composite includes the sphincteric orbicularis muscles that extend to the free edge of the soft tissue aperture of the eyelids and lips. The retaining ligaments, which are such a key feature of the five-layered anatomy, are not present over the cavities. There are thus anatomical and functional transitions from the relative stability over fixed areas to the high mobility of the soft tissue shutters over the cavities. In order to support this composite, the retaining ligaments are condensed and compacted along the bony orbital rim (Figure 65.8). This is the anatomical basis for the periorbital ligament around the orbit, of which the lower lid part is the orbicularis-retaining ligament, which stabilizes the orbicularis oculi to the orbital rim periosteum. Around the oral cavity, the ligaments arise from the

platform of the body of the zygoma, from the deep fascia over the masseter and from the body of the zygoma.⁹

The deeper component of the eyelid and lips are derived from the origin of the cavity and are not an extension of the facial soft tissues. In the eyelid, the deeper lid muscles with their related aponeurosis (levator and capsulopalpebral fasciae) and fat are retained by the fascial system of the septum orbitale. The free edges of the upper and lower eyelids obtain their ligamentous support from the tarsal plates, with their canthal tendon attachments to the medial and lateral orbital rims. In the pretarsal area, the superficial and deep lid structures, the anterior and posterior lamellae, merge. But in between, the orbital rim and the pretarsal area of the lower lid the, lamellae remain quite separate (i.e. the preseptal orbicularis does not have an attachment to the septum orbitale). This is the anatomical basis for the surgically significant preseptal space of the lower lid. In the upper lids, there is not an equivalent space as the submuscular fat pad over the superior orbital rim continues on the outer surface of the septum orbitale where it is adherent to the overlying fascia on the underside of the orbicularis almost down to where the levator crosses into the orbicularis.

The extent of the vestibule of the oral cavity, covering the maxilla and the mandible, has a major impact on the susceptibility to ageing of the overlying soft tissue. The skeleton underlying the space is unavailable to provide ligamentous attachment for support of the soft tissues that cover this large area. The extreme mobility of the lip and adjacent part of the cheek renders it susceptible to ageing changes and the indication for a lower facelift is largely to correct ageing changes in this unsupported tissue.

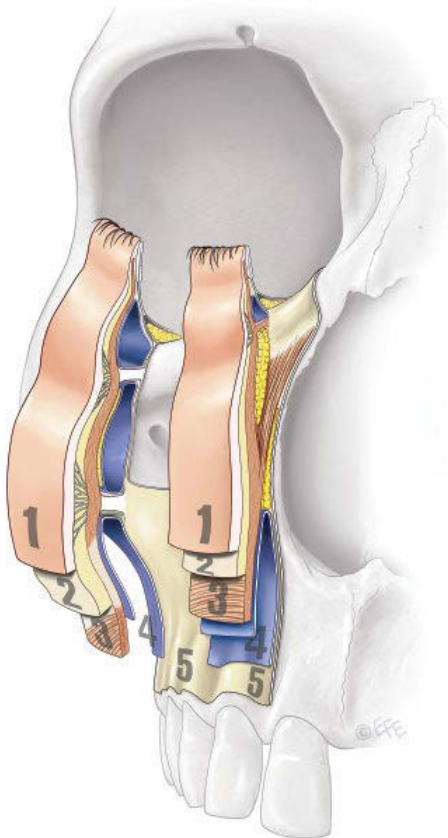


Figure 65.7 The anatomy over the skeleton and over bony cavities, showing the relationship of the soft tissue spaces to bony cavity spaces.

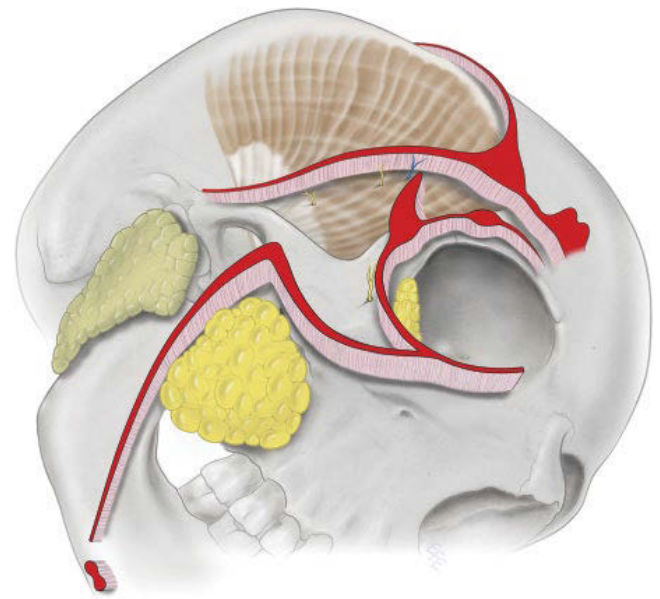


Figure 65.8 The system of retaining ligaments situated around the bony cavities stabilizes the soft tissue over the cavities.

Facial spaces

A large part of the sub-SMAS layer 4 consists of soft tissue 'spaces'. These spaces have defined boundaries that are strategically reinforced by retaining ligaments. Significantly, these areas are by definition anatomically 'safe spaces' to dissect, as no structures cross within and all branches of the facial nerve are outside these spaces. As the roof of each space is the least supported part, they are more prone to developing laxity here with ageing, compared to their ligament-reinforced boundaries. This differential laxity accounts for much of the characteristic changes that occur with ageing. Once a space has been surgically defined to its boundaries, the retaining ligaments in the boundary can then be precisely released under direct vision to achieve the desired mobilization, while preserving the vital structures closely associated with the ligaments. A brief description of surgically significant facial soft tissue spaces is given below.

Upper temporal space

The upper temporal space separates the temporoparietal fascia from the deep temporal fascia and is separated from the forehead by the superior temporal septum (STS) along the superior temporal line (Figure 65.9).³ Antero-inferiorly, the upper space is separated from a lower triangular temporal area that contains important anatomy, by the inferior temporal septum (ITS). These septi merge at the triangular-shaped zone of adhesion called the temporal (orbital) ligament. The upper temporal space provides safe surgical access to the lateral brow and upper midcheek. The space can be readily opened by blunt dissection to its boundaries. Once identified, the boundaries are then released by precise sharp dissection. The STS can be released sharply, taking care only to preserve the lateral (deep) branch of the supraorbital nerve, which runs parallel to the septum about 0.5 cm medial to it. The ITS provides a marker to the important anatomy here as the temporal branches of the facial nerve are located parallel to and immediately inferior to this septum. To release the ITS, the ceiling of the space is gently lifted off the deep temporal fascia floor, which three-dimensionalizes the septum in preparation for gentle release at the level of the floor, bearing in mind the frontal branches are located on the underside of the temporoparietal fascia in the ceiling, under the roof of the lower temporal area. Once released, the sentinel vein comes into view. The sentinel vein is not a good landmark for locating the temporal branches as they course cephalad to the vein, between it and the ITS, where they travel in a layer of fat suspended on the underside of the temporoparietal fascia.

Prezygomatic space

This triangular-shaped space overlies the body of the zygoma, its floor covering the origins of the zygomatic muscles. The space allows the independent displacement of the obicularis oculi (pars orbitale) in its roof from the zygomatic muscles

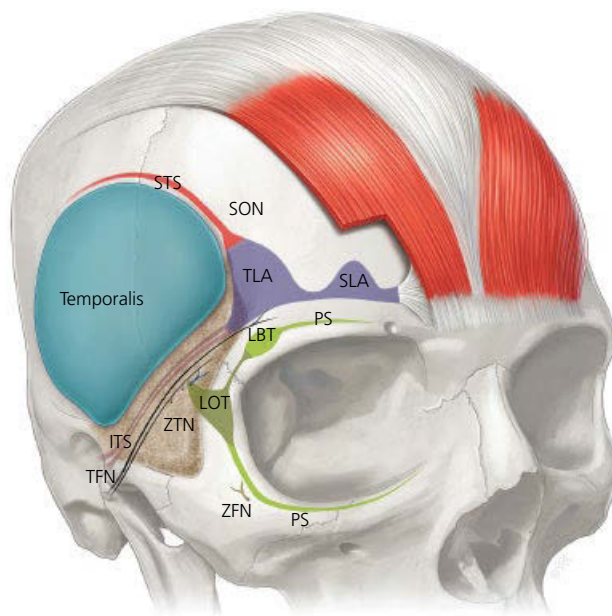


Figure 65.9 The upper temporal space and the retaining ligaments of the temple. The boundaries of the space are the superior temporal septum (STS) and the inferior temporal septum (ITS), which are extensions of the temporal ligament adhesion (TLA). No structures cross the temporal space. The TLA continues medially as the supraorbital ligamentous adhesion (SLA). Inferior to the temporal space is the triangular-shaped area of important anatomy (stippled). Crossing Level 4 in this area are the medial and lateral branches of the zygomatic temporal nerve (ZTN) and the sentinel vein. The temporal branches of the facial nerve (TFN) course on the underside of the temporoparietal fascia over the area immediately inferior to the ITS. The periorbital septum (PS, green) is on the orbital rim at the boundary of the orbital cavity. The lateral orbital thickening (LOT) and the lateral row thickening (LBT) are parts of the periorbital septum.

under the floor. Contraction of the overlying orbicularis elevates the prezygomatic soft tissues, which results in zygomatic smile lines (below the crows feet) (Figure 65.10).^{10,11} With ageing laxity the roof of the space rests at a lower level. As a result, there is a now a greater amplitude of movement on orbicularis contraction, exaggerating the zygomatic lines with ageing. This ageing of the prezygomatic space, with bulging over its roof accentuated by its well-supported boundaries, is the anatomical basis for the clinical entity variously described as malar mounds, bags or malar crescent. These deformities indicate the presence of significant laxity and the treatment is directed to tightening the laxity of the roof.

Premaxillary space

This quadrangular-shaped space (medial to the prezygomatic space) overlies the maxilla. Its floor is formed by the levator labii superioris. The space allows the independent displacement of the obicularis oculi in its roof from the lip elevators under the floor. Figure 65.11 details the anatomical relations of the premaxillary space.¹² This space is clinically significant in that laxity over its roof contributes to the deepening of the nasolabial fold with ageing.

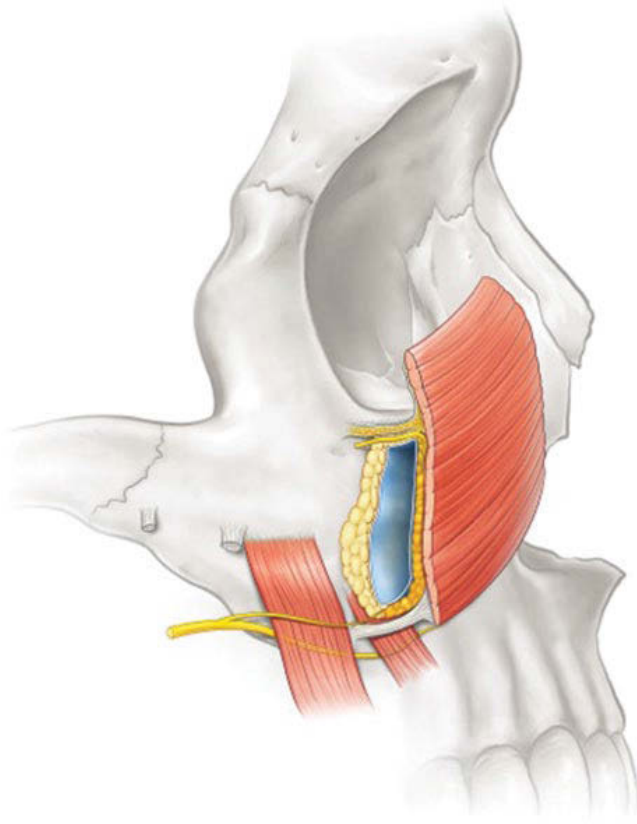


Figure 65.10 The prezygomatic space overlies the body of the zygoma. The origin of the zygomatic muscles extends under the floor. The roof is formed by the orbicularis oculi line by the SOOF (suborbicularis oculi fat). The upper ligamentous border formed by the orbicularis-retaining ligament is not as strong as the zygomatic ligament-reinforced lower border.

Lower premasseter space

This space overlies the lower half of the masseter and is analogous to the temporal space in that it overlies the deep fascia of a muscle of mastication (Figure 65.12).¹³ This gliding soft tissue plane allows opening of the jaw without restriction and avoids excessive distortion of the overlying soft tissues. The roof of this space is formed by the platysma. The lower premasseter space has profound clinical significance as it is the anatomical basis for the development of jowls with ageing. Laxity in the roof of the space, particularly where it has a weakened attachment to the anterior masseter by the masseteric ligaments and its inferior boundary where there is no ligament, manifests as the labiomandibular fold and jowl, respectively. The relatively stable fixation at the anterior-inferior corner of the premasseter space provided by the mandibular ligament accounts for the dimple that is commonly seen separating the labiomandibular fold above and the jowl below.

Middle premasseter space

The middle premasseter space is a rectangular space cephalad to the lower premasseter space (Figure 65.13).¹⁴ Its floor is formed by the masseteric facial and its roof by the SMAS. Its an important

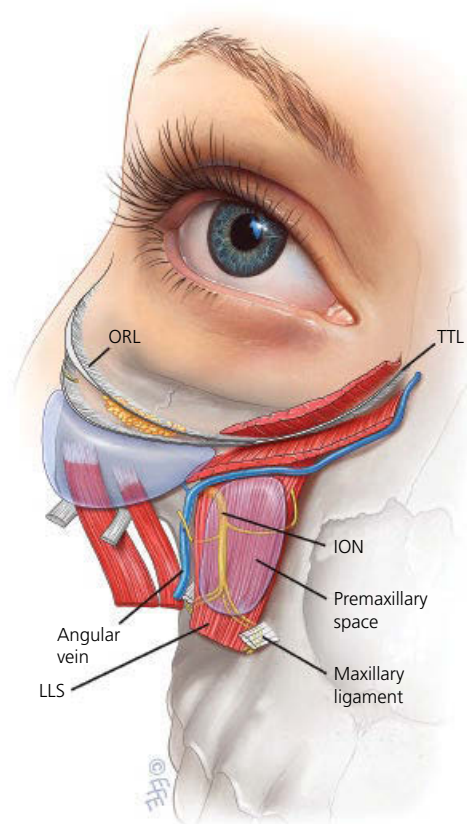


Figure 65.11 The anatomy and key relations of the premaxillary space. LLS, levator labii superioris; ION, infraorbital nerve, TTL, tear trough ligament, ORL, orbicularis-retaining ligament.

facial soft tissue space in sub-SMAS facelift in that it is an anatomically safe area to dissect, with facial nerve branches located in its superior and inferior boundaries. The parotid gland and the duct are closely associated with the upper boundary of the middle premasseter space.

Buccal space

This is one of the deep facial spaces, being like the submandibular space (which contains the submandibular gland), deep to the deep fascia (layer 5). The buccal space is located in the anterior face, medial to the anterior border of the masseter above the level of the oral commissure in youth. The space and its contents, the buccal fat, facilitates movement of the overlying nasolabial segment of the midcheek as well as buffering this area from excessive motion from jaw movement. Ageing and attrition of the boundaries, particularly of the masseteric ligaments inferiorly result in the platysma being less firmly bound to the masseter. This allows the space to enlarge and also allows the buccal fat to prolapse inferiorly, below the level of the commissure into the lower face. As the buccal fat comes to overlie the anterior border of the lower masseter it results in increased prominence of the labiomandibular fold and jowl.

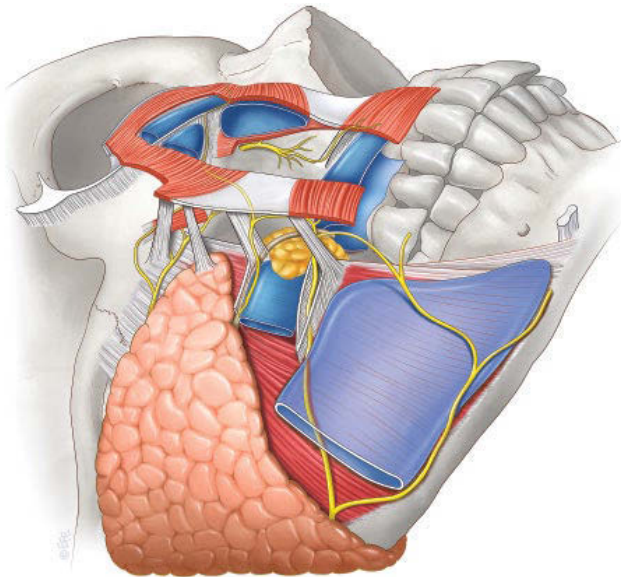


Figure 65.12 The rhomboid-shaped premasseter space overlies the lower half of the masseter. The roof of the space is formed by the platysma. The posterior border is defined by the anterior edge of the strong PAF and the anterior border is reinforced by the masseteric ligaments near the anterior edge of the masseter. The inferior boundary is mesenteric-like and does not contain any ligament. Weakness of attachment of the platysma roof at the inferior boundary leads to the formation of the jowl directly behind the strong mandibular ligament. The buccal space containing the buccal fat is anterior to the upper masseteric ligaments. All facial nerve branches course around and outside the space. The surgically important mandibular branch, after leaving the fixed PAF, courses under the inferior boundary of the space then rises onto the highly mobile outer surface of the mesenteric inferior border before reaching the mandibular ligament.

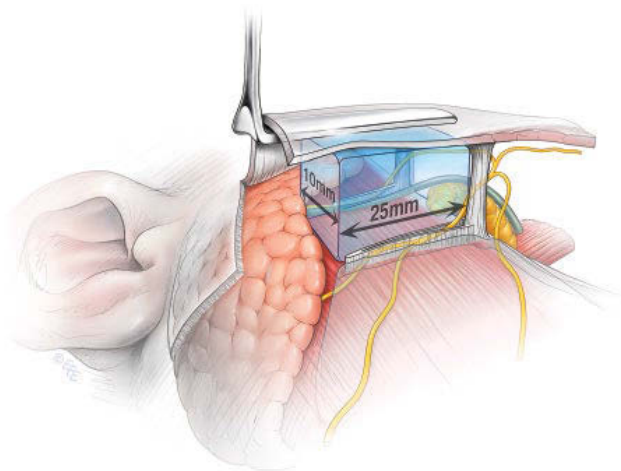


Figure 65.13 The middle premasseter space is located within the concavity of the parotid overlying the anterior masseter, above the lower space. The upper and lower buccal trunks of the facial nerve course outside the space within the upper and lower boundaries of the space. The masseteric process of the buccal fat pad is the only content and is variable.

Retaining ligaments of the face

To illustrate key concepts that retaining ligaments function to bind all five layers of the facial soft tissues together and that they are the anatomical basis for cutaneous grooves (seen between areas of laxity/bulges), let us examine the midcheek. The tear trough/orbicularis-retaining ligament complex is the key retaining ligament of the upper midcheek.¹⁵ It is located and closely follows the edges of the orbital rim. This retaining ligament system separates bony cavity (the orbital cavity) above from the bony platform (that of the midcheek skeleton) below, functioning to stabilize and secure the soft tissues of the lower eyelid before it transitions from the relative stability over the bone to the mobile area over the orbital cavity.

The *tear trough ligament* is a true osteocutaneous ligament originating from the maxilla and inserting into the dermis of the tear trough, passing through and binding all five soft tissue layers together.¹⁵ As previously shown, this is the retaining ligament that is the anatomical basis for the tear trough deformity (Figure 65.14). Laterally, the tear trough ligament continues as the orbicularis-retaining ligament. The orbicularis-retaining ligament has previously been demonstrated to be the anatomical cause of the palpebromalar groove. The continuity of the tear trough ligament with the orbicularis-retaining ligament then explains the observation that with more advance ageing, the tear trough becomes continuous with the palpebromalar groove, forming a prominent cutaneous groove sometimes called a prominent 'lid-cheek junction'. This demonstrates the key role facial soft tissue-retaining ligaments play in the cutaneous grooves seen with ageing.

Facial nerve branches

The danger zone for facial nerve injury has been well described in the literature, but is of limited value to the surgeon due to the two-dimensional perspective that gives the expected course of the nerve relative to surface landmarks.^{16,17} Confidence when approaching the nerve surgically comes from an understanding of the three-dimensional course of the nerve relative to the layered anatomy as described above and visually identifying the nerves in relation to defined landmarks (Figure 65.15).¹⁸ The facial nerve branches exit the parotid gland and remain deep to layer 5 in the lateral face. As they approach the anterior face, the branches traverse layer 4 to reach the underside of mimetic muscles of the face. It is at these transition points across layer 4 that the nerves are at greatest risk of injury. The transitions occur at predictable locations, in close association with retaining ligaments that provide stability and protection for the nerves. The surgical release of these ligaments to gain the needed mobility should be performed with extreme care on account of the proximity of the nerves.

The surface marking of the temporal branch of the facial nerve is along the Pitanguy line, from a point 0.5 cm below the

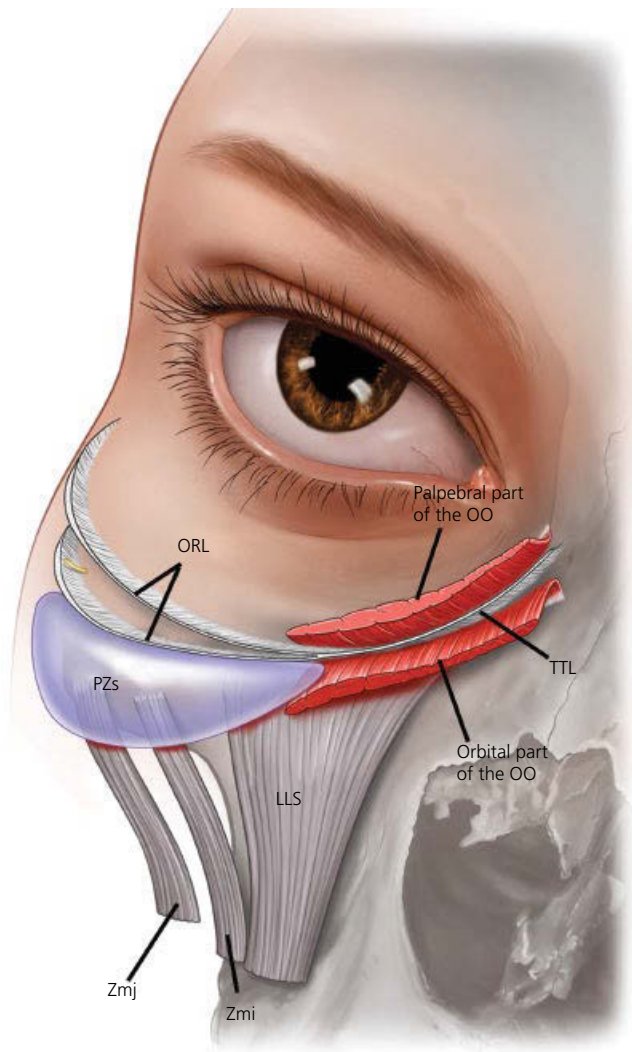


Figure 65.14 The tear trough ligament (TTL), the anatomical basis for the tear trough deformity. ORL, orbicularis-retaining ligament; OO, orbicularis oculi; PZs, prezygomatic space; LLS, levator labii superioris; Zmj, zygomaticus major; Zmi, zygomaticus minor.

tragus to a point 1.5 cm lateral to the supraorbital rim.¹⁹ It is traditional teaching that once the temporal branch exits the parotid, it immediately runs superficially from the deep fascia and comes to lie just deep to the SMAS as it crosses the arch of the zygoma.^{20,21} Because of its superficial location, surgical transection of the SMAS here, so-called high SMAS transection (i.e. at or above the arch) has been generally discouraged. It is now apparent that the nerve is deeper than was previously thought as it crosses the zygomatic arch. A histological study confirmed that the temporal branches are in transition from where they exit the parotid below the zygomatic arch, to where they enter the underside of the temporoparietal fascia some 2 cm above the arch.²² They course in a tissue layer (layer 4), just deep to the temporoparietal fascia (layer 3) and immediately superficial to the periosteum and above that, the temporalis fascia (layer 5), all along protected by a fascial, fatty layer, which is an upward

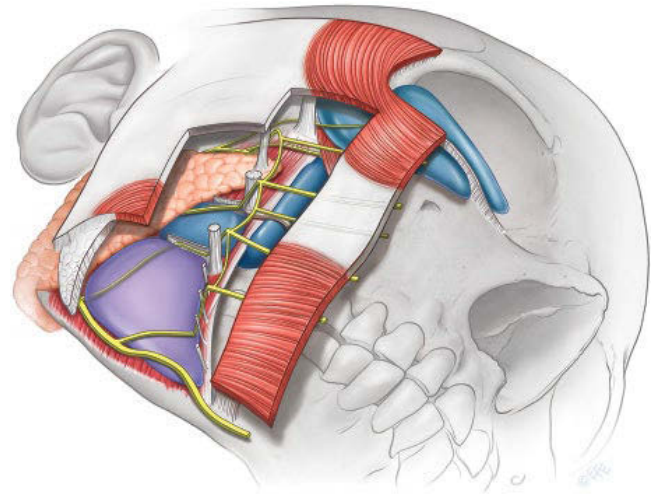


Figure 65.15 The relationship between facial nerve branches, spaces and retaining ligaments. The nerves stay deep to and outside the spaces at all times in the lateral face. In the boundary between the lateral and anterior face, the facial nerve branches transition from under layer 5 to enter layer 3, always in close association with the retaining ligaments of the face.

prolongation of the parotid-masseteric fascia and named the parotid-temporal fascia. In another study, the temporal branch was noted to transition to under the temporoparietal fascia (layer 3) at a distance of 1.5–3 cm above the zygomatic arch. The temporal branch would have transitioned to the underside of the temporoparietal fascia before the nerve crosses the sentinel vein more cephalically. Thus, once the sentinel vein is visualized from the temporal incision, the temporal branches would be located in the roof of the upper temporal space, in the wafer-like layer under the temporoparietal fascia.

The zygomatic branch exits the parotid gland deep to the deep fascia just below the zygoma and cephalad to the parotid duct. It travels horizontally on the masseter with the transverse facial artery. At approximately the origin of the zygomaticus major muscle, strong retaining ligaments (zygomatic ligaments) arise from the body of the zygoma. Here, the zygomatic nerve transition to the underside of the muscle it innervates. The zygomatic branch runs under zygomaticus major and minor and supplies them from the deep aspect in close association with the zygomatic ligaments. At the lateral border of zygomaticus major, a branch is usually given off to supply the orbicularis oculi, entering the muscle at its inferolateral corner. Careful dissection by vertical spreading of the scissors is crucial to avoid damaging this branch here.

The upper buccal trunk exits the parotid, about in line with, but superficial to the parotid duct and continues deep to the investing masseter fascia, close to but outside the upper boundary of the middle premasseter space. Approaching the anterior edge of the masseter, this branch leaves the floor under the masseter fascia in close association with upper key masseteric ligament. The lower buccal trunk leaves the parotid lower down, at about the level of the earlobe and remains under the

masseter fascia close to the lower boundary of the middle pre-masseter space. Similarly, upon approaching the anterior edge of the masseter, the lower buccal trunk transitions from deep to gain the underside of the SMAS in close association with the lower key masseteric ligament. After the nerves reach level 3, the zygomatic, upper and lower buccal trunks and mandibular branch connect with each other before continuing their course to innervate the mimetic muscles. This accounts for the overlap in muscles innervated by these nerves.

The temporal and mandibular branches are the most significant in terms of surgical risks because of the lack of cross-innervation of their target muscle. The marginal mandibular nerve is at risk where it is fixed by its close association with the retaining ligaments. Early in its course, around the angle of the mandible, this is within the PAF, and then well anteriorly by the mandibular ligament. Over most of its course, the mandibular branch is mobile, being in relation to the inferior boundary of the pre-masseter space. It is not necessary to dissect in the vicinity of the nerve because of the inherent mobility of the platysma where it overlies the jaw and submandibular area. The mobility of the nerve as it travels within the inferior membranous boundary of the lower pre-masseter space accounts for the reported variability of the location of this part of its course (occasionally below the mandible).^{23,24}

Ageing changes of the face

The youthful face has the general appearance of high rounded fullness, whereas the ageing process is characterized by a look of depletion and sagging, suggestive of tiredness. Changes with ageing occur at every level of the facial anatomy, starting with the facial skeleton. Current understanding of the ageing process remains largely empirical, given that it is based on the effectiveness of treatments designed to satisfy the requests of patients for a younger appearance. Historically, stretching of loose facial skin (traditional facelift), removal of apparent tissue excess (traditional blepharoplasty), tightening the dermis and evening the complexion (early phenol peels and CO₂ laser resurfacing) and, in recent years, soft tissue volume augmentation (lipofilling and soft tissue fillers) have all had a positive impact on rejuvenating appearance. The success of each is attributed to having reversed a cause of facial ageing. Yet, when each of these modalities is continued as the sole treatment, to further reverse the ageing appearance, the results tend to be bizarre, leading to the conclusion that multimodal therapy is required to further reverse, what must be, multiple components of the ageing process.

An understanding of the changes that occur in the layered anatomy forms the basis for logical treatment. Changes of the skin are readily observable and changes in the skeleton affecting layer 5 can also be observed radiologically. Because the changes within the superficial fascia (layers 2 and 3) are not directly measurable, empiricism has remained prevalent. A correlation

of the surface anatomy changes of ageing with the anatomy of layers 2, 3 and 4 indicates that bulging occurs over the roof of soft tissue spaces, which stand out in contrast to the absence of bulging of the adjacent cutaneous grooves. The grooves reflect the restriction imposed by the dermal insertions of the retaining ligaments at the boundaries of the spaces. The degree to which the bulging reflects true elongation from primary tissue degeneration and laxity and how much it is 'apparent' laxity secondary to loss of volume (skeletal and soft tissue) remains unanswered.

Skin

Skin ageing is influenced by genetics, environmental exposure, hormonal changes and metabolic processes. With ageing, the supple skin of youth becomes thinned and flattened, with loss of elasticity and architectural regularity.²⁵ Atrophy of the extracellular matrix is reflected by the decreased number of fibroblasts and decreased levels of collagen (especially types I and III) and elastin in the dermis. Although chronological skin ageing and photo-ageing can be readily distinguished and considered separate entities, both share important molecular features, that of altered signal transduction that promote matrix metalloproteinase (MMP) expression, decreased procollagen synthesis and connective tissue damage. Oxidative stress is considered of primary importance in driving the ageing process, resulting in increased hydrogen peroxide and other reactive oxygen species (ROS) and decreased antioxidant enzymes. These changes result in gene and protein structure alterations. Other environmental factors, notably smoking, accelerates skin ageing by between 10 and 20 years. Increased collagenase and decreased skin circulation have been suggested as possible mechanisms. The muscles of facial expression flex the skin in a specific pattern. As the underlying collagen weakens and the skin thins, the dermis loses its capacity to resist the constant force of the muscles and these lines become etched in the skin, ultimately even at rest.

Subcutaneous tissue

The fibrous and fat components in the subcutaneous tissue are not uniform but arranged in discrete compartments. Over specific sites, due to its prominence, the subcutaneous fat has been given specific names such as the malar fat pad and nasolabial fat. The boundary of these subcutaneous compartments corresponds to the location of the retaining ligaments, which pass superficially to insert into the dermis. In youth, transition between compartments is smooth and non-discernible. With ageing, a series of concavities and convexities develop, which separate these compartments. These changes have been attributed to a number of causes, including fat descent, selective atrophy and hypertrophy and attenuation of the retaining ligaments that causes the fat compartments to become malpositioned. It is apparent now, however, that fat descends minimally with ageing. As noted, the subcutaneous fat is not a confluent layer that can descend with ageing.

Distinct compartmentalization by the retaining ligaments holds the fat in its relative positions.

Muscle ageing

Skeletal muscles, in general, have been noted to atrophy up to 50% with age. This may be applicable to the muscle of mastication, such as the temporalis and masseter (compounded by the decreased demand and deterioration of the dentition with ageing), although no specific study on the effect of ageing on these muscles has been done to date. The mimetic muscles of the face, in contrast to skeletal muscles, may not undergo the same degree of degeneration with ageing because of their constant use with facial expression. The orbicularis oculi has been noted to remain histologically unchanged with no loss of muscle fibres, adherence to surrounding tissues or ptosis with ageing. The upper lip elevators, zygomaticus major and levator labii superioris were also noted to remained unchanged with ageing, based on MRI of their length, thickness and volume. In contrast, the upper lip orbicularis atrophies with ageing, with decreased muscle thickness, smaller muscle fascicles and increase in surrounding epimysium.

Facial spaces and retaining ligaments

The multilinked fibrous system attenuates with ageing, with decreasing strength of the ligaments and increasing laxity. The spaces expand with ageing as well, to a greater extent than the laxity that develops in the ligaments within their boundaries,

resulting in bulges between areas of relative fixations.¹³ Accordingly, the spaces dissect easily in older patients, and the boundaries widen as the ligaments weaken. In young people, the spaces are more potential than real and do not open quite so easily with blunt dissection (Figure 65.16).

Bone changes

The facial skeleton changes dramatically with ageing (Figure 65.17) and this has a profound impact on the appearance of the face with ageing (Figure 65.18).²⁶ At birth, the facial skeleton is underdeveloped and rudimentary. This explains why infants and toddlers often transiently have distinct midcheek segments (despite excellent tissue quality), which disappear as they grow older with the expansion of the midcheek skeleton. Peak skeletal projection is probably attained in early adulthood. Thereafter, although certain areas continue to expand, selective areas of the facial skeleton undergo significant resorption. Areas with strong predisposition to resorption include the superomedial and inferolateral aspects of the orbital rim, the midface skeleton, particularly that part contributed by the maxilla, including the pyriform area of the nose and also the prejowl area of the mandible. The resultant deficiencies in the skeletal foundation have a significant effect on the overlying soft tissues. In the midcheek in particular, retrusion of the maxilla causes increased prominence of the tear trough and the nasolabial folds. The retrusion of the facial skeleton causes the origin of the multilinked fibrous retaining ligaments to be displaced

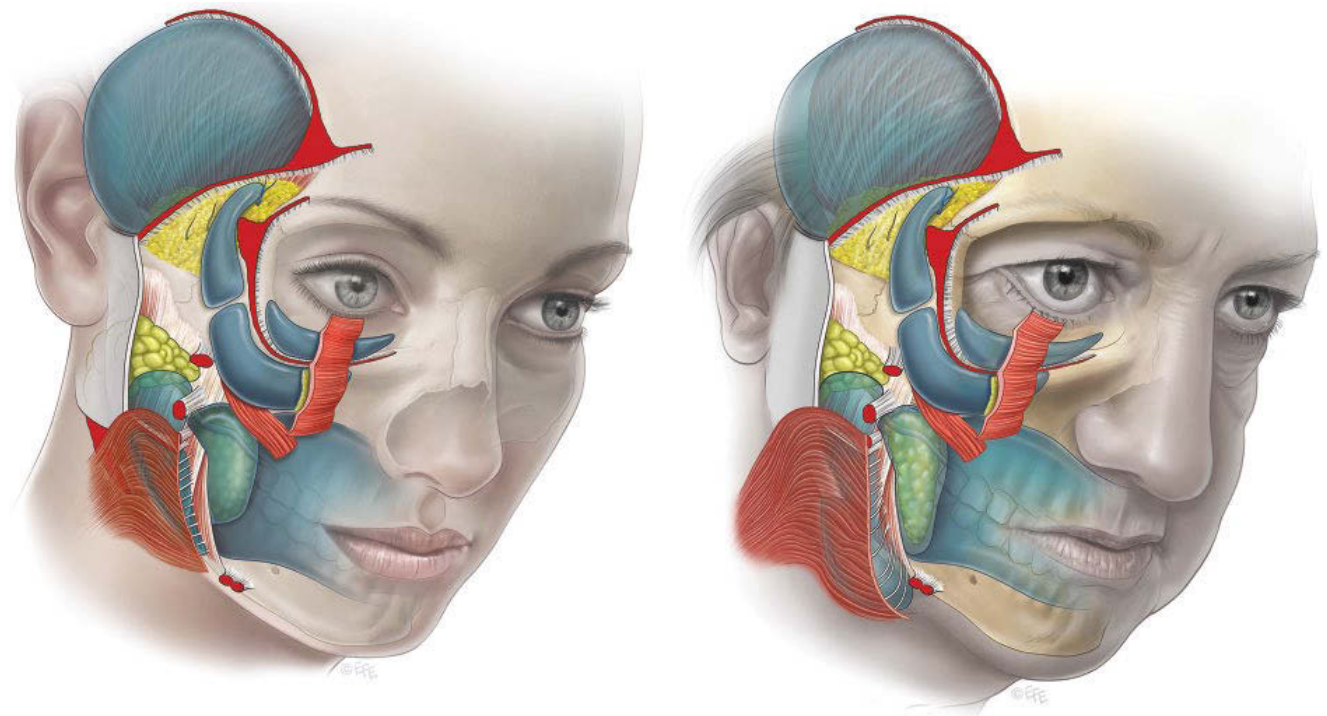


Figure 65.16 In youth, the spaces are tight and more potential than real. The retaining ligaments are stout and the transition between spaces and spaces are not discernible. With ageing, spaces expand to a greater extent than the laxity that develops in ligaments within their boundaries, resulting in bulges between areas of relative fixations. These spaces open with relative ease with blunt dissection.

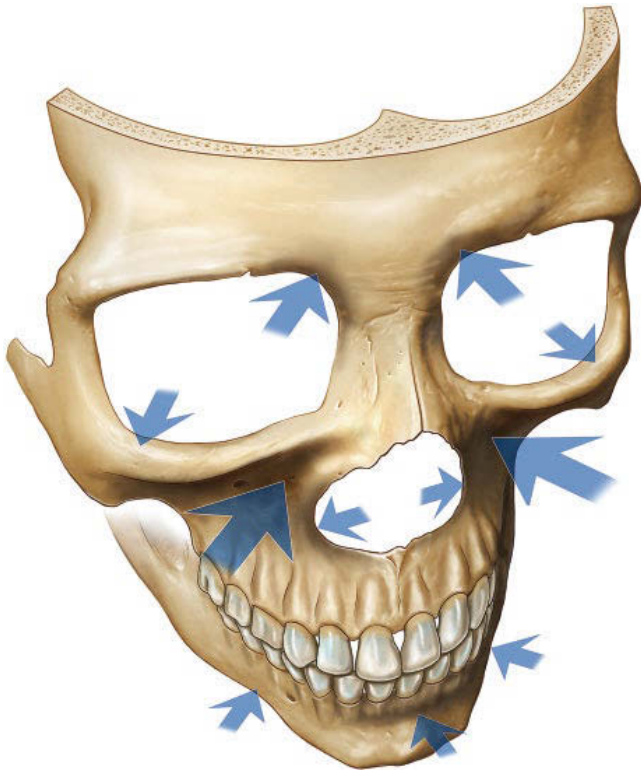


Figure 65.17 Arrows indicate the areas of the craniofacial skeleton that are susceptible to resorption with ageing.

posteriorly. This pulls the skin inwards, exaggerating the concavity between the areas of relative convexity that develop with ageing. Retrusion of the midcheek with loss of projection gives the visual impression of tissue descent with ageing. Some patients have a congenitally weak or inadequate skeletal structure. In such cases, the skeleton may be the primary cause of the manifestations of premature ageing. Accordingly, patients who suffer premature facial ageing, a weakness of the relevant part of the underlying skeleton is immediately suspect and should be addressed in order to obtain better aesthetic results.

Regional changes observed with the ageing face

Temple and forehead

The skin of the temple differs from that of the forehead, being thinner and less firmly supported to the underlying layers. The loose attachment reflects the underlying temporal space, which is extensive, and the nature of the surrounding temporal ligaments that are septal like and do not continue through the thin, loose subcutaneous layer 2 over the temple to fix to the dermis as do facial ligaments elsewhere. This explains why deep layer procedures in the temple are not as effective in toning the overlying skin as they are, for example, in the cheek.

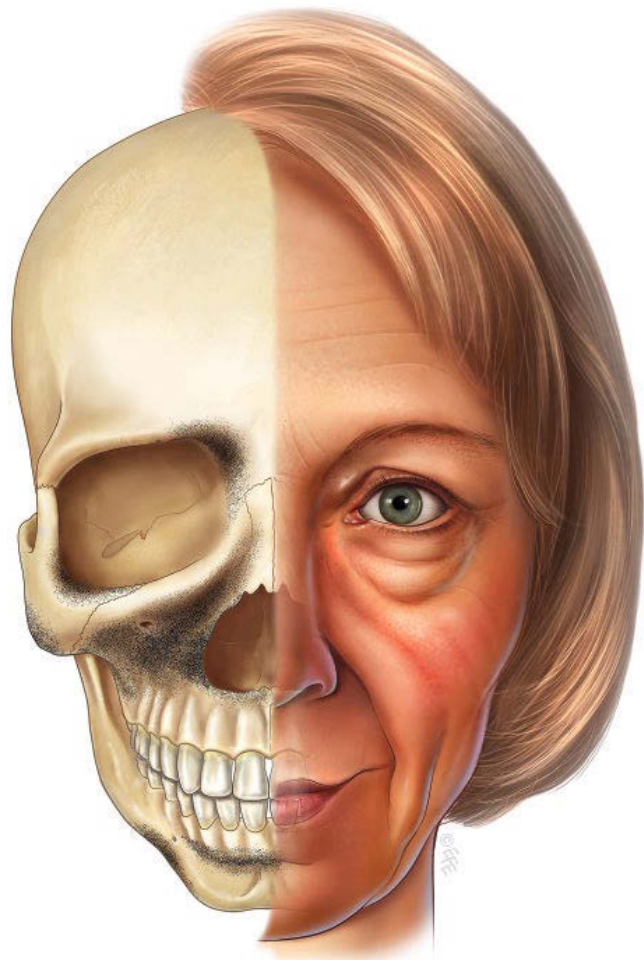


Figure 65.18 The darker areas denote areas of greatest bone loss. Note how the stigmata of ageing as manifested by the facial soft tissues correspond to the areas of weakened skeletal support due to bone loss.

Corrugator muscle contraction is associated with the emotional states of grief and sadness. The transverse head of corrugator supercilii moves the eyebrow medially and produce vertical glabella lines. The oblique head of the corrugator, the depressor supercilii and the medial fibres of the obicularis oculi act in concert to depress the medial brow and produce oblique glabella frown lines. The procerus, also a brow depressor, causes transverse nasal skin lines. Laterally, the action of the lateral fibres of the obicularis oculi with the transverse head of the corrugator supercilii promotes lateral brow ptosis. The ptosis of the lateral brow together and to a lesser extent the laxity of the skin with ageing produces a pseudoexcess of the upper eyelid skin. Frontalis muscle hypertonicity from lateral brow skin hooding and its reaction to the action of antagonistic muscles (corrugator supercilii, obicularis oculi and procerus) results in the development of transverse forehead skin lines.

The medial brow, in contrast, seldom descends with ageing and in fact may rise. The mechanism responsible for this includes the chronic activation of the frontalis muscle. This may

either be to elevate the brow/eyelid complex associated with clinical or subclinical levator system weakness or to relieve visual field obstruction due to excess lateral upper eyelid skin. Anatomically, the frontalis muscle ends at approximately the temporal fusion line (STS). Lateral to this, there is no upward vector to counteract the downward pull of brow depressors and gravity on the lateral brow. This may explain why descent preferentially occurs at the lateral brow.

The midcheek

The midcheek is the anterior part of the midface. It is triangular in shape and bounded superiorly by the pretarsal part of the lower eyelid, medially by the side of the nose and the nasolabial groove below, and laterally around the lateral cheek where the arch of the zygoma meets the body. A smooth rounded midcheek is a powerful image of youth and gives a certain freshness to the face. With ageing, the three midcheek segments become clearly discernible, as they become separated by the three cutaneous grooves of the midcheek: the nasojugal, palpebromalar and midcheek grooves. This 'segmentation' of the midcheek has a profound impact on appearance and is responsible for giving the 'tired' look we associate with ageing.

The soft tissues of the midcheek are structurally composed of three segments or 'modules', with each overlying a specific part of the midcheek skeleton (Figure 65.19). The lid–cheek

segment overlies the prominence of the inferior orbital rim, the malar segment overlies the body of the zygoma and the nasolabial segment overlies the anterior surface of the maxilla. The skeletal foundation of the midcheek borders the three bony cavities of the anterior face, the orbital, nasal and oral cavities. Because of the many spaces and limited bony support available, the midcheek has some intrinsic structural weaknesses. Three factors make the midcheek susceptible to ageing changes. These are (1) the wedge shape of the soft tissues of the midcheek, which are thin above and thicker below, (2) the natural posterior incline of the midcheek skeleton from the relative prominence of the infraorbital rim and (3) the significant retrusion as a result of resorption of the maxilla with ageing. This is not uniform, as the maxilla recedes more medially and inferiorly. With early ageing, the retrusion of the maxilla, along with a slight descent of the wedge-shaped cheek soft tissue results in an appreciable reduction of volume of the upper cheek. The result is that the small amount of orbital fat over the prominent edge of the infraorbital rim, (originally concealed by the volume of the upper cheek), now becomes revealed, especially the underside of the lid fat bulge over the middle part. The visual impression is of a 'lengthened' lower lid. At the same time, the increased thickness of the soft tissue mass over the lower cheek tends to conceal the degree of maxillary resorption and gives the profound visual impression that the soft tissue mass has descended into the lower part of the midcheek.²⁷

Of the three segments of the midcheek, the lower lid segment changes the most dynamically with ageing. It has two distinct grooves across its surface that varies in their expression during the ageing process, often coexisting. The upper is the infratarsal groove at the junction of the pretarsal and preseptal parts of the eyelid. It defines the lower boundary of the pretarsal bulge. The pretarsal bulge in youth is the visual separation of the lid above and the cheek below. This so-called 'high lid–cheek junction' is located well above the infraorbital rim and is a characteristic of youth. The infratarsal groove location does not change with ageing, although its contour usually fades. The lower groove is the lid–cheek junction that relates to the lower edge of the pre-septal part of the lid. It is not usually present in youth and appears with ageing and then progressively deepens and descends slightly over time. Its shape when it first appears is a gentle C contour but as it 'descends', particularly in its central portion, its shape changes to a progressively more angulated V shape with the medial side being formed by the developing nasojugal groove and the lateral side by the palpebromalar groove. The centre of the V, the lowest and deepest part has the nasojugal groove continuing down the cheek as the midcheek groove that separates the cheek into the malar and nasolabial segments.

The reason why this contour demarcation changes while the skin itself does not descend is explained by difference in the tissue layers. In level 4, the orbicularis-retaining ligament is not rigid where it is over the centre of the inferior orbital rim, so

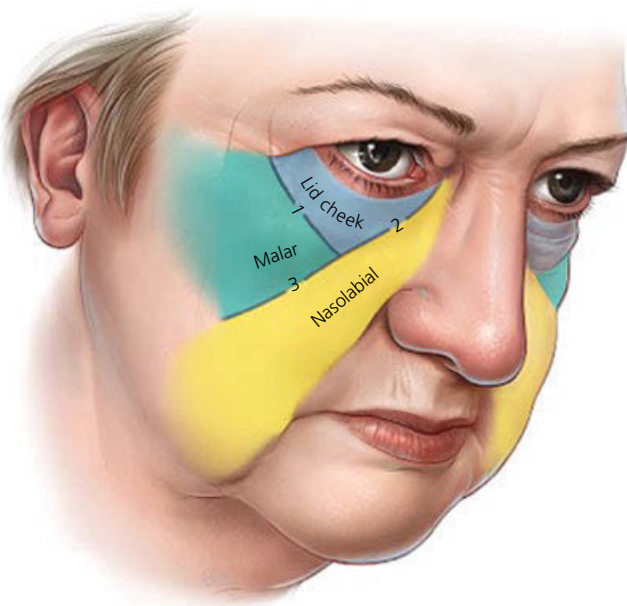


Figure 65.19 The midcheek has three segments, the lid–cheek segment (blue) and the malar segment (green) are within the periorbital part and are adjacent to the nasolabial segment (yellow) in the perioral part, which overlies the vestibule of the oral cavity. The three grooves define the boundaries of the three segments and interconnect like the italic letter Y. The palpebromalar groove (1) overlies the inferomedial orbital rim and the nasojugal groove (2) overlies the inferolateral orbital rim, then continues into the midcheek groove (3).

distension results in relative sliding between it and layer 3, the orbicularis oculi. As the lid–cheek junction becomes more prominent, it visually takes over from the infratarsal groove and becomes the new separation between the lower eyelid and the cheek. This is the basis for the commonly used but inaccurate phrase ‘lengthening of the lid cheek junction with ageing’ which is in fact the result of a visual shift from the prominence of the infratarsal groove in youth to the lid–cheek junction with age. Correction of the ageing of the lid–cheek segment of the mid-cheek, the visibly descended contour of the lid–cheek junction and long lower lid has gained the colloquial name of ‘blending the lid cheek junction’.

Lower face

The jowl and the labiomandibular fold in the lower face are not present in youth and develop with ageing. With the description of the concept of soft tissue spaces of the face, and specifically the premaseter space, the mechanism for the formation of the jowl can now be understood on an anatomical basis. With the onset of ageing, laxity develops in the roof of the premaseter space associated with attenuation of the anterior and inferior boundaries. The major retaining ligaments (the key masseteric and mandibular) in contrast remain relatively strong and at these locations the superficial fascia remains firmly fixed to the underlying deep fascia. Distension of the weaker masseteric ligaments at the anterior border of the lower premaseter space (below the key masseteric ligament) and inferior displacement of the buccal fat (within the buccal space) is the anatomical basis for the development of the labiomandibular fold. The mandibular ligament demarcates the transition from the labiomandibular fold above and the jowl below. The jowl develops as a result of distension of the roof of the lower premaseter space with resultant descent of the tissues below the body of the mandible. The more prominent the jowl, the more apparent will be the cutaneous tethering provided by the mandibular ligament. Accordingly, the anatomical solution to correcting these ageing changes is to reduce the inferiorly displaced buccal fat and to tighten the roof of the premaseter space.

Considerations for correcting ageing changes of the face based on the anatomy of the ageing face

Dissection planes

The subcutaneous plane of dissection (level 2) is the most commonly used plane for facelifts, either in isolation or more commonly with some form of SMAS management from the superficial aspect (Figure 65.20). A distinction should be drawn between subcutaneous dissection over the lateral face from that over the anterior face. This plane of dissection is perceived to be ‘safe’ as dissection remains superficial to the facial nerve branches at all times and is the main appeal of level 2 dissection. The subcutaneous dissection can be

performed either in the superficial subcutaneous or deep subcutaneous level. In the former, there is more density of the reticular cutis fibres as the multilinked ligaments branch out before inserting into the dermis. In the latter on the outer surface of the SMAS, there are fewer fibres, which tend to be thicker and stronger. The deep subcutaneous layer is not uniform in its tenacity: some areas, such as over the facial spaces, are inherently easier to dissect, whereas others that overlie the ligament are more adherent and require sharp release. For example, over the malar eminence at MrGregor’s patch, where the zygomatic ligaments are located, sharp release is often needed, as is required over the mandibular ligament. In contrast, in the lower face over the premaseter space, the subcutaneous layer separates quite readily, requiring only blunt finger dissection.

Sub-SMAS dissection (level 4)

In the scalp, this is the preferred tissue plane for dissection as the scalp readily separates from the underlying periosteum (level 5) through the avascular areolar tissue with ease and inherent safety. Bruising and swelling is kept to a minimum because of this anatomy. In the face proper, although the anatomical principles remain the same, level 4 is potentially the most risky plane to dissect because of the facial nerve branches which transition through this level, from level 5, to supply the facial muscles in level 3. However, it should be noted that similar to the situation in the scalp where raising the flap at level 4 gives a robust and structurally integrated composite flap that can be effectively tightened, sub-SMAS dissection in the face has the same advantages and potential benefits. Dissection can be performed safely in level 4 by applying the understanding of the three-dimensional anatomy of the face described earlier; the key being the facial spaces, which provide safe access through this layer. Because these spaces are ‘predissected’, access is quick, atraumatic and easy. An example of this is the lower premaseter space. Subcutaneous dissection is performed to approximately 30 mm anterior to the tragus through the zone of fixation, the PAF, where the SMAS is fused to the deep fascia including the parotid capsule. Because the objective of the surgery is to correct laxity in the mobile anterior face, the level of dissection used in the lateral face is of secondary importance.

A further benefit of leaving the PAF intact is that it is strong and can be used for suture fixation.²⁸ Once dissection has proceeded beyond the PAF (indicated by the posterior border of the platysma), the SMAS should then be incised to gain direct access into the lower premaseter space. The space can then be opened by gentle blunt dissection only, to define the boundaries of the space. The premaseter space below and the prezygomatic space above form a series of spaces in the anterior face (Figure 65.21). The boundaries of the spaces, reinforced by retaining ligaments, are where the important anatomy is located. These need to be precisely released to eliminate their tethering effect on the soft tissues, which is more difficult in younger patients as the

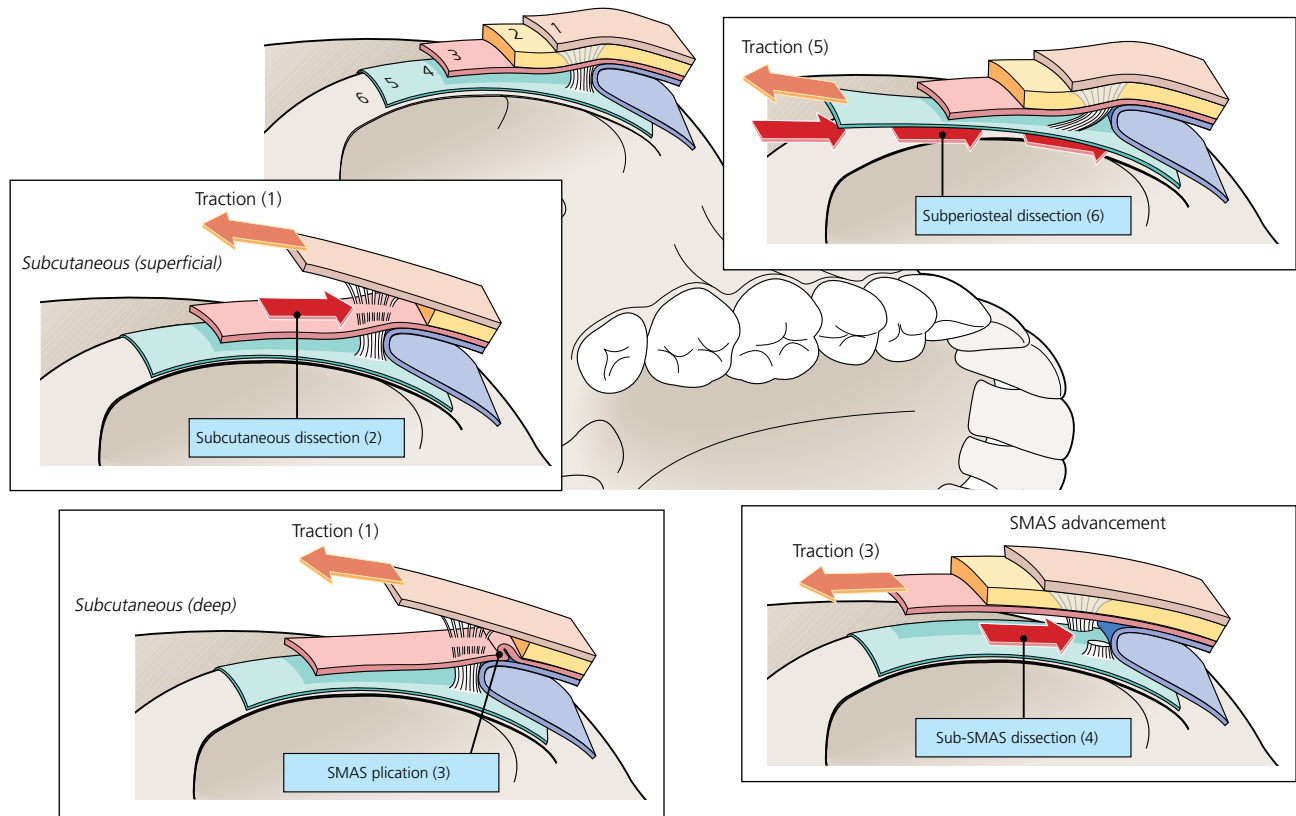


Figure 65.20 Alternative levels for dissection and redraping in facelifts. Dissection can be performed through any one of three alternative layers, namely subcutaneous (level 2), sub-SMAS (level 4) and subperiosteal for the upper two thirds of the face.

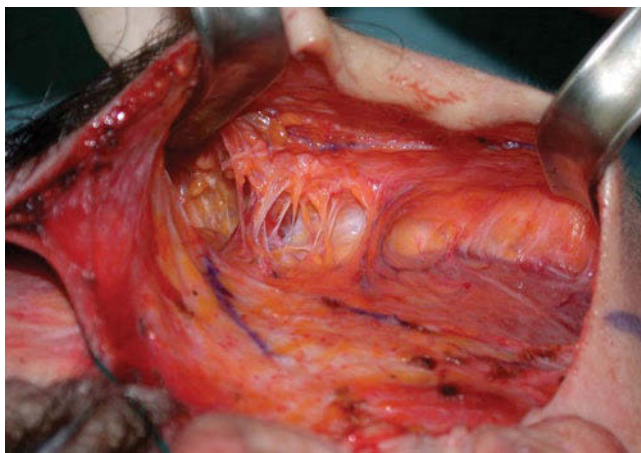


Figure 65.21 Using the facial spaces for safe and anatomical access to sub-SMAS dissection in facelifts.

ligaments are denser and stronger. Clear visualization, optimized by lifting the opened adjacent facial spaces, is beneficial. When blunt scissors are used with gentle vertical spreading of the blades the surrounding fat and areolar tissue separate to reveal the ligaments and the facial nerve branches in relation to them. With further lifting, the ligaments become more certainly

defined as they tighten further, at which time they can be safely released, while the nerves, being obliquely orientated, dislodge out of the way, unaffected by the controlled stretching.

The sub-SMAS spaces can be used to safely and atraumatically access various part of the face, the deep temporal space to the lateral brow, the preseptal space to the lower eyelid, and the prezygomatic space to the midcheek.

Level 5

Subperiosteal 'lifts' have the appeal of safety as far as the facial nerve risk is concerned as they are superficial and the remote nerves never cross this plane. However, there are inherent limitations to subperiosteal lifts. The accumulated ageing changes across all five layers are elevated as part of the subperiosteal lift. Overcorrection is required to effect the desired changes of soft tissue shape and skin tone, to compensate for the 'lift-lag' phenomenon, which is proportional to the soft tissue thickness and laxity. Accordingly, subperiosteal lifts work best in areas where the layers are more compacted as the lift-lag is minimized. An example of this is in the brow where subperiosteal lifts are popular and effective. Where the layers are thicker, such as the nasolabial segment of the midcheek, the lift-lag phenomenon significantly limits the improvement that can be achieved.

Because of the unyielding nature of the periosteum, extensive undermining beyond the target area is needed or alternatively a 'periosteal release' immediately beyond the area that requires lifting to isolate the area to a limited island of periosteum.

Placement of sutures

Although adequate surgical release is needed for mobility, it is the surgical fixation that achieves the desired effect by holding the mobilized soft tissue in its new position.²⁹ The strength and tenacity of the superficial fascia is not uniform. The areas where the retaining ligaments are located have an inherent ligamentous reinforcement, making them ideal for suture placement. It is also the location in which traction gives the most natural appearance, as these are the natural suspension sites of the face. Accordingly, the suture fixation should be placed where the retaining ligaments are located. Where fixation sutures are placed in the anterior face in sub-SMAS surgery, they function as replacements for the retaining ligaments that have weakened or have been divided in order to mobilize the composite flap. Accordingly, the replacement sutures should replicate the quality of support provided by the original ligaments as the 'mobile' spaces remain. In this respect braided permanent sutures are advantageous as they stimulate collagen and elastic deposition within the suture similar to a ligament. The PAF, a diffuse ligamentous area on the lateral face, provides an ideal area both anatomically and physically to fix the facelift flap, due to its inherent strength.

Summary

This chapter has been structured to assist the reader to develop a conceptual understanding of facial anatomy and how it changes with ageing. It is the framework that unifies the detailed anatomical information now available in the literature. Once understood, this knowledge will serve as the anatomical foundation for the logical and sound selection of surgical techniques for rejuvenation of the ageing face.

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Non-operative facial rejuvenation

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Introduction

In the ageing face, atrophy of subcutaneous tissues and changes to bony landmarks allow unsupported skin to fall into wrinkles and folds, and surface irregularities – such as coarse textural changes and alterations in pigmentation – to emerge. Traditionally, facial rejuvenation focused on invasive procedures, such as ablative laser resurfacing or cosmetic surgery, that were associated with considerable periods of recovery, patient discomfort and potential complications. A rise in demand for minimally invasive procedures with appreciable results and fewer side effects over the last two decades has led to a surge in popularity of ‘lunch-time’ treatments, from botulinum toxin (BoNT) and a plethora of injectable fillers, to a growing number of non-ablative light- or energy-based modalities intended to rejuvenate the skin. Used alone or in combination, these non-invasive therapies form the basis of an individualized approach to the modern ageing face.

Botulinum toxin

Botulinum toxin (BoNT) derives from *Clostridium botulinum*, the bacterium identified as the causative agent of botulism (from contaminated blood sausages) in nineteenth-century Germany. The symptoms of botulism include disturbances in vision, speech, and swallowing, with asphyxia and death sometimes occurring 18–36 h after ingestion.¹ The most potent serotype of *C. botulinum* – BoNT type A (BoNT-A) – was originally isolated, purified, concentrated and crystallized for use as a potential biological weapon in 1946, but the idea was abandoned when it became apparent that large doses were required to produce fatal results.²

Clinical experimentation with BoNT-A began in the late 1960s and early 1970s, when Dr Alan Scott (Smith-Kettlewell Eye Research Foundation, San Francisco, California, USA) used a second lot of BoNT-A to non-surgically align the eyes of primates with strabismus and published his findings in

1973.³ He followed his initial encouraging results with a landmark paper in 1980 demonstrating that intramuscular injections of BoNT-A could correct gaze misalignment in humans.⁴ By 1989, BoNT-A had been approved by the US Food and Drug Administration (FDA) for the non-surgical correction of strabismus, blepharospasm, hemifacial spasm and Meige’s syndrome in adults, and clinical use had widened to include the treatment of cervical dystonia and spasmodic torticollis.⁵ In 1992, Carruthers and Carruthers published the first report of the cosmetic use of BoNT-A for the treatment of glabellar frown lines.⁶ Other reports soon followed,^{7,8} along with the first double-blind, placebo-controlled study for the treatment of hyperkinetic facial lines.⁹

Mechanism of action and clinical effect

All serotypes block neuromuscular motor transmission by binding to receptor sites on motor nerve terminals. The light chain of BoNT-A cleaves to a 25 kDa synaptosomal associated protein (SNAP-25), whereas the light chain of BoNT type B cleaves to vesicle-associated membrane protein (VAMP or synaptobrevin).⁵ BoNT inhibits the release of acetylcholine and other neurotransmitters, including substance-P, glutamate and calcitonin-gene related peptide. When injected intramuscularly at therapeutic doses, BoNT produces temporary chemical denervation of the muscle, resulting in a localized reduction of muscle activity. The paralytic effect of the toxin is dose-related, with initial effects occurring within 2–3 days and peaking approximately 1–2 weeks after treatment. Effects generally last around three months; repeated injections can extend the clinical effect for up to 12 months.

Formulations

To date, the FDA has approved three distinct formulations of BoNT-A for both therapeutic and cosmetic purposes, and one formulation of BoNT-B for therapeutic use only. The original formulation of BoNT-A, onabotulinumtoxinA (BOTOX®/BOTOX Cosmetic®/Vistabel®/Vistabex®; Allergan, Inc., Irvine, CA, USA), has been joined by abobotulinumtoxinA (Dysport®;

Ipsen Ltd, UK/Medicis, Scottsdale, AZ, USA; and Azzalure® in 15 European countries; Galderma, France) and icobotulinumtoxinA (Xeomin®/NT-201; Merz Pharmaceuticals, Frankfurt, Germany). Only one formulation of BoNT-B – rimabotulinumtoxinB (Myobloc®/NeuroBloc®; Solstice Neurosciences Inc./Eisai Co., Ltd) – is available in North America. All products differ in terms of diffusion, potency and clinical effect. Likewise, clinical doses vary according to product and are not interchangeable.

Cosmetic use of BoNT-A

In 2002, after numerous open-label studies involving more than 800 subjects, and two large, double-blind, placebo-controlled randomized clinical trials, the FDA approved BoNT-A for the non-surgical reduction of glabellar furrows.¹⁰ Since then, off-label use of the toxin has exploded to include any number of hyperkinetic lines or deep grooves and folds in the face, neck and chest caused or exacerbated by repetitive muscular activity, with a high level of efficacy and patient satisfaction.^{11,12} Moreover, BoNT-A is often used to lift and shape the brow,¹³ widen the eyes,¹⁴ correct facial asymmetry¹⁵ and to reduce muscle thickness of the jaw in patients with masseteric hypertrophy.¹⁶

BoNT-A works particularly well in combination with other rejuvenating procedures, such as soft tissue augmentation and laser, light- or energy-based therapies, where it has been shown to prolong and enhance aesthetic results, especially for the treatment of deeper, more static rhytides and folds. In subjects who require surgical intervention, BoNT-A can be used during and after surgery to prolong surgical results and aid in wound healing; pre- and posttreatment injections stabilize the musculature surrounding the incision while the procedures heal, resulting in improved cosmesis of the scars.¹⁷

Adverse effects and complications

Most side effects of BoNT-A are injection-related and transient, such as swelling, bruising and pain. Complications occur when the toxin diffuses into non-targeted or glandular tissue surrounding the site of injection, due to overenthusiastic doses, inappropriate injection sites or poor technique.¹⁸ In the upper face, adverse events translate into a drooping eyelid or brow (ptosis), watery, dry or crossed eyes, and eyebrows that can appear askew or asymmetrical. Lower face complications impact muscle function, resulting in a flaccid cheek, lopsided smile, incompetent mouth, the inability to whistle, dysphagia and neck weakness. However, when used appropriately by experienced injectors, cosmetic doses of BoNT-A are well tolerated and safe, and over 20 years of clinical use has demonstrated a strong record of safety.¹⁹ Serious adverse events are rare and more likely to occur with the much higher doses used for therapeutic indications, particularly in individuals who may have underlying medical conditions that increase the risk of complications.²⁰

Fillers

While BoNT-A is first-line treatment for dynamic rhytides, deeper lines and folds require volume replacement for adequate correction. The volumizing effect of the first filler – autologous fat – was studied in 1893 when it was harvested and inserted into the face of a 20-year-old man to reconstruct a deeply retracted scar.²¹ Nearly a century later, autologous fat grafting was considered, by some, to be the ideal filling agent, allowing for soft tissue augmentation without any risk of allergy.²² However, although several grafting procedures were performed with variable success, results were often unpredictable and largely dependent on the method of harvesting, type of fat used and injector experience. The procedures themselves were complicated, with only a 25–30% rate of persistence when injected into areas of the face,²³ and rife with adverse effects that included donor site morbidity, calcification of the injected fat and unpredictable reabsorption.²⁴ By the 1980s, interest in using autologous fat had begun to wane, particularly after the introduction of the first ‘modern’ filling agent for human use: bovine collagen.

The most abundant protein in skin, human and animal-based collagen was first injected into volunteers in the late 1970s.²³ Bovine collagen was the first soft tissue augmenting agent approved in the United States,²⁵ but was characterized by a short duration of effect and required skin testing prior to injection. Human-derived collagen – developed to reduce the risk of antigenicity and hypersensitivity reactions – no longer required skin testing but did not last any longer than bovine collagen.²³ This lack of duration, particularly around areas of great animation, was collagen’s greatest disadvantage and was no doubt responsible for its gradual decline in use and eventual disappearance from the market when newer, more durable products arrived on the scene.

The development of synthetic fillers marked a new page in the history of facial rejuvenation. It was now possible to inject into different layers of the skin, and this discovery opened the door for three-dimensional volume replacement to achieve optimal aesthetic results, shifting from the treatment of lines and wrinkles to address the issue of subcutaneous atrophy and fat loss. Filling agents today generally fall under two categories: non-biodegradable and biodegradable.

Non-biodegradable filling agents

Ideal for individuals who desire permanent facial augmentation, non-biodegradable fillers can yield excellent results when used by skilled clinicians. However, because they are not readily broken down or absorbed by the body, they are associated with a higher risk of complications that can be more difficult to resolve.

Liquid injectable silicone

Liquid injectable silicone – purified polydimethylsiloxane – has been approved by the FDA only for intraocular use as a prolonged retinal tamponade during vitreoretinal surgery but has been used for subdermal volume restoration since 1997, when the US Modernization Act allowed physicians to use approved

medical devices and products for off-label indications.²⁶ Silicone oil induces neocollagenesis through fibroplasia in addition to direct volume restoration, but its use has been plagued by controversy thanks to associated complications, including migration and granulomatous reactions that may occur from weeks to decades after injection and can be severely debilitating.^{27,28} However, complications are often the result of improper technique, injection of large volumes and the use of industrial-grade products – frequently adulterated – injected by unlicensed or unskilled practitioners.^{29–31} Use of medical-grade, pure liquid injectable silicone and proper injection protocol – microdroplet serial puncture technique administered at monthly intervals to allow for product stabilization through adequate collagen deposition – can produce exceptional, permanent results,^{32,33} and liquid injectable silicone has been particularly beneficial over the long term in the treatment of HIV-associated facial lipoatrophy,^{33–36} acne scarring, and lip augmentation.^{37–40}

Polymethylmethacrylate

Approved by the FDA in 2006 for the correction of nasolabial folds, polymethylmethacrylate (PMMA; ArteFill®; Suneva Medical Inc., San Diego, CA, USA) is a third-generation filler comprising 20% PMMA microspheres (32–40 µm diameter) and 80% bovine collagen. After implantation, the carrier solution dissipates within 1–3 months, leaving behind the non-resorbable microspheres that induce fibroplasia and become encapsulated by collagen. Because of the animal component, skin testing is required prior to implantation. PMMA is used to treat well-defined, deeper folds and rhytides, as well as facial wasting and cheek depressions.^{41,42} Multiple treatments with small amounts of material are usually required to achieve optimal results and avoid overcorrection. Like silicone oil, the use of PMMA is controversial due to the history of side effects noted with first- and second-generation products (Arteplast® and Artecoll®, respectively),⁴² particularly nodules and beading in more mobile areas, hypertrophic scarring, ridges and granulomatous reactions that were reported to occur up to 6 years after implantation.^{43–48} Although severe complications are not as common with third-generation product,⁴⁹ PMMA is associated with a steep learning curve in terms of patient selection and injection technique and schedule, and should not be attempted by the inexperienced clinician.

Biodegradable fillers

Biodegradable filling agents require regular treatments to maintain the desired results but are less likely to provoke a severe complication and, in some cases, are entirely reversible.

Calcium hydroxylapatite

Calcium hydroxylapatite (CaHA) microspheres (Radiesse®; Merz Aesthetics, Inc., San Mateo, CA, USA) comprise biocompatible, smooth CaHA spherical particles (25–45 µm) that are identical in composition to bone material and suspended in an aqueous

carboxymethylcellulose gel carrier. The microspheres induce histiocytic and fibroblastic response, stimulating the production of collagen around the implant, while the gel carrier is absorbed. CaHA lasts for a year or more before the microspheres are broken down into calcium and phosphate ions and excreted.^{50,51} CaHA is used for its approved indications – the treatment of nasolabial folds and HIV-related facial lipoatrophy – as well as off-label for contouring and cheek augmentation, and to fill marionette lines and depressed scars, with a high level of patient satisfaction and results lasting up to 18 months.^{50–59} Although CaHA has a favourable long-term safety profile,⁶⁰ nodules and foreign body reactions have been reported,^{51–53,56,58,61} including one case of nodule formation from a distant injection site.⁶²

Poly-L-lactic acid

The FDA approved poly-L-lactic acid (PLLA; Sculptra®; Dermik Laboratories/sanofi-aventis, Bridgewater, NJ, USA), a biocompatible and biodegradable synthetic polymer from the alpha-hydroxy-acid (AHA) family, for the treatment of HIV-related lipoatrophy and moderate-to-deep rhytides in 2004 and 2009, respectively. When injected into the skin, PLLA stimulates fibroblasts to produce collagen, resulting in a gradual increase in dermal volume. The particles are eventually metabolized to lactic acid monomers that are further broken down by the body.⁶³ A number of studies have demonstrated the aesthetic efficacy of PLLA in the treatment of facial lipoatrophy,^{64–67} acne scars,^{68,69} and to fill deep rhytides and folds in the face.^{70–73}

Long-term trials have demonstrated the safety and tolerability of PLLA when used appropriately.^{74,75} Most side effects are due to the injection procedure, with complications sometimes attributed to poor injection technique,⁷⁶ although late-onset papules and nodules have been reported,^{71,73,77} as have cases of delayed granulomas.^{78–80} PLLA must be diluted 2–24 h prior to injection with sterile preserved water to allow adequate time for polymer hydration, and patients require a series of three injections over several months. These requirements, combined with an initial misunderstanding of proper injection protocol, have limited wider use of PLLA.²³

Hyaluronic acids

By far the most popular biodegradable filling agents are hyaluronic acid (HA) derivatives. An abundant key structural component of the skin and one of the chief components of the extracellular matrix, HA is a long, hydrophilic, cross-linked glycosaminoglycan that acts as a scaffold for collagen and elastin and contributes to cell proliferation and migration, as well as tissue repair.⁸¹ The HA molecule is stabilized through cross-linking with hydroxyl groups – allowing them to resist digestion by hyaluronidase – and it is the degree and type of cross-linking that determines longevity of the various HA products on the market. Although a large number of HA fillers are available for soft tissue augmentation (Table 66.1), they are all unique in terms of concentration of HA, cross-linking, particle size and manufacturing processes.

Table 66.1 Hyaluronic acid derivatives in clinical use in North America

Trade names	HA concentration (mg/mL)
Restylane®	20
Restylane® Fine Lines	
Perlane®	
Juvéderm®	24
Juvéderm® Ultra	
Juvéderm® Ultra Plus	
Juvéderm® Voluma®	20
Prévelle Silk®	5.5
Hydrelle®	28
Hylaform®	5.5
Hylaform® Plus	
Puragen®	20
Puragen® Plus	
Belotero®	22.5

When injected into the skin, HA combines with the body's own HA and binds to water, increasing skin turgor, elasticity and volume for a period of 4–12 months, depending on the product used. Most formulations are approved for the correction of moderate-to-severe facial rhytides, such as the nasolabial folds, but are also used off-label for volume replacement in the cheeks and elsewhere.⁸² The choice of product and viscosity depends on the depth of the defect and injector preference. The literature is replete with studies confirming the efficacy, safety and tolerability of HA fillers for the treatment of fine lines, deeper rhytides and folds, lip augmentation and facial contouring.^{82–84}

Side effects are generally mild and transient and include bruising and oedema. More serious adverse events are sometimes related to technique, such as the bluish Tyndall effect caused by injections placed too superficially.⁸⁵ Localized hypersensitivity reactions have been reported, particularly in products with higher amounts of protein.⁸⁶ More troublesome complications are rare but include granulomatous reactions^{87–89} and vascular ischaemic complications.^{90–94}

However, cross-linked HA is the only filler that can be fully reversed via the use of intracutaneous hyaluronidase, a soluble protein enzyme that breaks down and hydrolyses HA. Reports demonstrate that injection with hyaluronidase works within 24–48 h to relieve patient discomfort due to product misplacement or unwanted adverse effects.^{92,95–97}

Complications of biodegradable and non-biodegradable fillers

The most common adverse events for all filling agents are local injection site reactions and include transient pain on injection and intermittent bruising, oedema and erythema.⁸³ More serious complications can be classified as occurring early (up to 1 year after treatment) or delayed (1 year or more after implantation). Early complications usually result from poor injection technique and inappropriate placement of the filler, leading to palpable or visible implants, nodules and beading, changes to the texture and colour of the skin, and hypertrophic scarring.⁹⁷ Injections around

the mouth or in other areas of excessive movement or expression often cause lumpiness and migration of the implant. Hypersensitivity is rare but can occur with all fillers containing synthetic material; reactions range from persistent itching, burning and oedema, to granulomatous foreign body reactions. Vascular compromise leading to necrosis in the glabella or nasolabial folds – either by compression, injury or obstruction of the blood vessel – and retinal embolism after intravascular are rare but severe complications requiring immediate recognition and management.⁹⁸ Late-onset adverse events, such as excess fullness, persistent nodules, granulomatous or inflammatory reactions, chronic infections and filler migration, are almost always associated with non-biodegradable fillers, may be quiescent for over a year, and are often difficult to treat. Some complications can never be adequately reversed, particularly in the case of permanent filling particles; overcorrection, asymmetry and contour or textural irregularities require removal of the implant and may lead to scarring or other noticeable effects on the skin.⁹⁹

Cutaneous remodelling

Damage to the epidermis and dermis, which form a protective barrier against the environment, immediately initiates the tissue-repair cascade, in which inflammatory mediators induce fibroblasts to generate new collagen and elastin, ultimately leading to re-epithelialization and remodelling of the skin.

Microdermabrasion

Microdermabrasion is a simple and safe office procedure in which the skin surface is abraded with aluminium oxide crystals or other abrasive substances and removed with a hand-held vacuum device.¹⁰⁰ Aluminium oxide is the most commonly used abrasive, though less-effective crystals (sodium chloride, sodium bicarbonate and magnesium oxide) are also available.¹⁰¹ Crystal-free procedures involve diamond-tipped devices that are easier to use, neater and cause less pain.¹⁰² Depth of the treatment will depend on the rate and flow of the abrasive material, the amount of pressure applied with the hand-held device, and the number of passes over the skin. As with other cutaneous resurfacing techniques, surface trauma initiates the healing process, in which fibroblasts rush to produce new collagen, leading to smooth skin that looks fresh and robust. Weekly treatments can improve the appearance of fine lines, skin texture, pore size and acne scars, as well as postinflammatory pigmentation,¹⁰³ though posttreatment biopsies have shown no statistically significant improvement in rhytides.^{104,105}

Mild oedema and erythema are common immediately following treatment but usually subside within 48 h. Tiger stripes may appear on fair or sensitive skin. Adverse events are uncommon but include pain, burning, bruising (particularly around the lips), bleeding, infection and hyperpigmentation.¹⁰⁶ Uneven handling of the device can blemish the skin, and treatment that is too deep can permanently discolour the skin.¹⁰⁷

Chemical peels

A mainstay in the cosmetic clinician's armamentarium, chemical peels involve the topical application of chemical agents to the skin that in turn cause controlled destruction of part or all of epidermis, and sometimes the dermis.¹⁰⁸ Peels may be categorized as superficial, medium and deep; all stimulate collagen and glycosaminoglycan production, regardless of their depth of action.¹⁰⁹

Superficial peels affect the epidermis and upper dermal layers of skin and are used for rejuvenation and to treat mild dyschromias, acne, postinflammatory pigmentation and actinic keratoses. The most common agents for superficial peels include trichloroacetic acid (TCA, 10–20%), AHAs, such as glycolic acid (GA), and the beta-hydroxy-acids (BHAs), such as salicylic acid. Beta-lipohydroxy acid (LHA) is a derivative of salicylic acid that provides excellent exfoliation at lower concentrations with antibacterial, anti-inflammatory, antifungal and anticomedogenic properties.¹¹⁰ Epidermal regeneration occurs within 3–5 days after a superficial peel. Complications are rare, though transient burning, irritation and erythema are common.¹¹¹

Medium-depth peels use combination solutions containing TCA 35% as described by Brody (solid carbon dioxide + TCA),¹¹² Monheit (Jessner's solution + TCA)¹¹³ and Coleman (70% GA + TCA).¹¹⁴ Superficial peels are used for mild-to-moderate signs of photoageing, including pigmentary changes, lentigines, growths, dyschromas and rhytides.¹¹⁵ The healing process is longer, with full epithelialization within 7–10 days. Penetrating the reticular dermis, deep peels are usually performed with Baker–Gordon phenol in the surgical setting and are reserved for severe photoageing and deep rhytides, scars or lesions.¹¹⁰ Deep peels involve considerable risk of serious complications, such as scarring, changes in pigmentation and cardiotoxicity, and are considered invasive procedures that are frequently used as adjuncts to surgery. Adverse effects associated with medium-depth and deep peels include lines of demarcation, pigmentary changes, delayed healing and persistent erythema, bacterial infection, allergic reactions, scarring, acneiform eruptions, milia and rare instances of toxicity.^{110,115,116}

Conclusion

The ageing face undergoes many alterations, including changes to skin colour and texture, the emergence of wrinkles and furrows, and sagging caused by loss of underlying fat. The focus of rejuvenation today has shifted from surgical or ablative procedures associated with serious adverse events and long periods of recovery to those that are more convenient and less damaging. From injectables to ultrasound, significant advances and increasingly refined techniques have made it possible to subtly correct wrinkles, improve facial contours and irregularities, and restore volume and vitality with immediate aesthetic benefits, fewer complications, and little to no downtime.

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CHAPTER 67

Forehead lift

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Introduction

The first forehead lift procedures were performed using an 'open' technique in which long incisions on the scalp or along the frontal hairline were made, a forehead flap was turned inferiorly, and modifications were made to corrugator supercilii and other muscles under direct vision. These breakthrough procedures served several generations of patients well and, while still performed by many surgeons today, they have been rejected by some patients and abandoned by some surgeons who view them as too aggressive and time consuming. As endoscopic techniques made inroads into aesthetic surgery, one of the first procedures they were adapted to by plastic surgeons was the forehead lift. Although a decade or more of technical refinements had to pass before the 'closed' forehead lift procedure finally came of age, it has since come to be widely accepted and continues to be a viable alternative to open procedures for many patients. Experience has since shown that a 'closed' technique without an endoscope can be used to mobilize and release the forehead and modify the corrugator supercilii muscles if the anatomy is understood and the operation is appropriately planned.^{1,2} In addition, transpalpebral corrugator myectomy, when used in conjunction with closed mobilization and re-suspension of the forehead, not only provides a scheme for the performance of closed foreheadplasty without the need for an endoscope, but also give a method by which medial brow elevation can be minimized or avoided. This may, indeed, be one of the procedure's most important merits.

Patient options in forehead rejuvenation

Patients seeking improvement of their forehead appearance have three general categories of procedures available to them:

- open procedures using a coronal incision,
- open 'hairline lowering' and 'forehead shortening' procedures using an incision or partial incision along the hairline and
- closed 'limited-incision' or 'short-scar' endoscopic procedures.

Selection of the 'best' procedure depends on the patient's specific characteristics, including hairline position, the quality of tissues, the condition of the scalp, the amount of improvement sought, and the overall degree of ageing changes that are present.³

Coronal incision techniques

Foreheadplasty techniques using a coronal incision offer certain advantages over closed procedures and should result in an inconspicuous scar and few complications if skilfully performed.⁴⁻⁶ These advantages include that they provide an actual reduction in forehead scalp and/or skin redundancy, and many surgeons feel that this translates into better brow elevation and a more durable and sustained improvement. Procedures performed through a coronal incision also provide optimal access for modification of the frontalis muscles, if necessary, and numbness, dysaesthesias and related problems can be minimized if the anatomy of the sensory nerves is understood and nerve branches are protected during the procedure.⁷ In practice, a procedure using a coronal incision may be the procedure of choice for patients with abnormally low hairlines who wish their hairline to be raised and placed in better proportion with the rest of their face. This is the most common circumstance in which the procedure is performed in the authors' practice (Figure 67.1).

Procedures using coronal incisions have certain well-known possible problems and complications, however, including hair loss, poor scars, hairline elevation, numbness and dysaesthesias, and patients generally have the notion that these procedures will result in a 'surprised' or 'startled' appearance. Although these problems should be uncommon if the procedures are skilfully performed by an artistic surgeon, and can occur when 'limited-incision' and endoscopic techniques are used, patients are nonetheless understandably attracted to 'short-scar' techniques, and perceive them to be less likely to precipitate problems or result in unnatural appearances. In addition, because of the influence of the media, the Internet and other physicians, many patients arrive in the plastic surgeon's office already convinced that they only wish to undergo an 'endoscopic' forehead lift, and thus

Figure 67.1 Coronal forehead lift procedure. Patients with low hairlines and advanced forehead ageing are ideal candidates for coronal incision plans because these procedures provide for maximum improvement in eyebrow position and configuration and produce the most elevation of the hairline. (a) Patient, age 49, preoperatively. Note that suboptimal eyebrow position and configuration impart a sad and melancholic appearance. Note also the low frontal hairline. (b) Same patient seen 14 months following coronal forehead lift. Note that suboptimal eyebrow position and configuration seen preoperatively have been corrected and that hairline retro-displacement has expanded the forehead and placed it in better overall proportion with the rest of the face. (The patient has also undergone upper and lower blepharoplasty, trichloroacetic acid peel of the lower eyelids, and secondary rhinoplasty.) Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.



most surgeons will have to offer their patients some kind of limited-incision or endoscopic procedure as an option.

Hairline and partial hairline incision techniques

Many patients seeking rejuvenation of the forehead are troubled by high hairlines and the prospect that surgery will make this problem worse. Hairline elevation occurs in both coronal and closed endoscopic techniques, and hairstyles cannot practically conceal an overly high forehead. A high forehead also appears unnatural, masculine and detracts from an otherwise attractive and agreeable appearance.^{8–11} Hairline elevation can be particularly objectionable after surgery in the older patient with a receded hairline who has marked forehead and scalp redundancy (Figure 67.2).

Unlike procedures using a coronal or endoscopic technique, procedures using an incision or partial incisions along the hairline allow hairline position to be maintained or lowered and hairline shape to be optimized¹² and the improvement thus gained may outweigh the drawback of the resulting scar.

Patients with high hairlines generally are well aware of their own problem and are more than willing to accept the associated trade-offs, including the scar, when informed of this option. In such situations, hairline lowering is arguably of more overall benefit to the patient than is eyebrow elevation or frown-line reduction.

Closed and short-scar techniques

'Closed' foreheadplasty techniques, including endoscopic foreheadplasty, 'limited-incision' forehead lift, and short-scar foreheadplasty employing transpalpebral corrugator myectomy, have found their earliest and widest applications in younger

patients with low or low-normal hairlines who had minimal or modest frontalis hyperfunction and transverse forehead wrinkling and who were undergoing isolated forehead or forehead and eyelid surgery. In these situations, a full coronal incision and scalp excision is indeed not necessary if the surgeon can improve eyebrow position and configuration and perform muscle modification with a near equal degree of precision as in an 'open' technique. Over time, however, 'closed' foreheadplasty techniques have also proven to be useful and effective in older patients troubled by more advanced ageing deformities and in those patients undergoing simultaneous facelifts. Indeed, experience has shown that it is not necessary to remove scalp in many patients to produce an attractive and sustained improvement in upper facial appearance (Figure 67.3).

Experience has also shown that an endoscope is not required to mobilize and release the forehead and modify the corrugator supercilii muscles if forehead anatomy is known, the operation is correctly planned and a transpalpebral approach to treat the corrugators is used. Although an endoscope can be used for these purposes if desired, designing a procedure in which it is not necessary saves time, avoids problems related to the device itself (fogging, mechanical failure, etc.) and avoids the costs of purchasing, leasing and maintaining the expensive equipment needed.

A number of strong arguments can be made against the use of an endoscope for corrugator muscle modification, however, even though this is considered by many to be a breakthrough application of the technology. First, it is exceedingly easy to over-elevate the medial brow in forehead lift procedures but deceptively difficult to raise the lateral eyebrow. Because the corrugator muscles act as partial depressors of the medial brow, simple



(a)



(b)

Figure 67.2 Hairline forehead lift. (a) Patient seen preoperatively. She has previously undergone face lift, upper and lower blepharoplasties and genioplasty performed by an unknown surgeon. Note suboptimal eyebrow position and shape and high forehead. A coronal or closed forehead lift would make her high forehead worse. (b) Same patient following hairline-lowering forehead lift using an incision along the hairline. The poor eyebrow position and shape seen before surgery have been corrected and the hairline has been lowered. Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.



(a)



(b)

Figure 67.3 Closed, small-incision, non-endoscopic forehead lift. (a) Patient, age 42, seen preoperatively. Note suboptimal eyebrow position and shape. Some would claim that this degree of brow abnormality would be best treated with an 'open' foreheadplasty technique. (b) Same patient, 13 months following closed, small-incision, non-endoscopic forehead lift. The poor eyebrow position and shape seen before surgery has been corrected with minimal elevation of hairline. The patient has also undergone facelift, neck lift, upper and lower blepharoplasty, partial facial fat injections and an upper lip lift. Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.

muscle excision, regardless of technique, can result in some medial brow elevation. The obligatory undermining of the glabella and dividing the periosteum that is necessary to gain access to the corrugator muscles endoscopically also disrupts the ligaments and attachments that hold the medial brow in an aesthetically optimal low position¹³ and predictably results in additional undesirable central brow elevation. Modifying the corrugators transpalpebrally through an upper eyelid incision preserves ligaments and periosteal attachments in the glabellar area, however, and helps keep the medial brow in a more natural appearing and attractive lower position. As a practical matter,

endoscopic access to the corrugators can also often be difficult in the patient with a high hairline or curved, sloping forehead, because of the endoscope's straight design.

Transpalpebral corrugator myectomy, when used in conjunction with closed mobilization and re-suspension of the forehead, not only provides a method for performing a closed, short-scar foreheadplasty without the need for an endoscope, but also provides a means by which medial brow elevation can be minimized or avoided.

The method of corrugator modification also has an impact on the procedure's outcome and can reduce the frequency of

complaints patients have after surgery. In endoscopic procedures, a near-total corrugator myectomy is generally performed in which the majority of the corrugator muscle is removed and the supratrochlear nerves are skeletonized. This, however, can result in paraesthesias and dysaesthesias, which can be disturbing to patients. When a transpalpebral modification of the corrugator is performed, the muscle can simply be divided (myotomy) between the supraorbital and supratrochlear nerves and an interposition fat graft taken from elsewhere on the face can be placed between the cut ends of the muscle. When this is done, muscle function is usually significantly reduced, but the nerves need not be formally dissected. Typically, this results in fewer disturbing sensations and dysaesthesias in the forehead area. (It should be noted that a simple corrugator myotomy (dividing the corrugator) rarely results in a sustained reduction in corrugator supercilii muscle function in and of itself. In most such cases, the cut ends of the muscle heal together and the muscle regains function.)

A final and initially unexpected benefit of closed techniques, including the non-endoscopic one described here, is that hairline retro-displacement is reduced in comparison to coronal procedures. This is because the majority of the forehead flap elevated in a closed dissection is elevated in a subperiosteal plane, and the flap is thus relatively inelastic as compared with a flap dissected through a coronal incision and elevated in a subgaleal plane. In all, the closed, short-scar non-endoscopic forehead lift procedure provides an overall reduction in incision length, less hairline elevation, decreased operating time, better improvement eyebrow shape and possible reduction in forehead and scalp neurosensory disturbance.

Anaesthesia

Most foreheadplasties performed in the authors' clinic are performed under local anaesthesia with heavy sedation using a laryngeal mask airway, but any skilfully administered anaesthetic is adequate.¹⁴ After sedation has been administered or anaesthesia established, supraorbital, supratrochlear and lacrimal nerve blocks are performed with 0.25% bupivacaine (Marcaine) with 1:200 000 adrenaline (epinephrine). Proposed sites for incisions are subsequently infiltrated with the same solution. 'Hyperinflating' incision sites with additional (and typically more dilute) local anaesthetic solution (0.1% lidocaine (lignocaine) with 1:1 000 000 adrenaline) immediately before incisions are made helps reduce bleeding by establishing a partial hydrostatic tamponade. Local anaesthesia should be administered even if general anaesthesia is used to reduce bleeding and lower the overall amount of narcotics and anaesthetics required. Upon completion of the procedure but before the patient is awake, nerve blocks should be re-administered to minimize patient discomfort in the postoperative period.

Operative technique for closed, non-endoscopic forehead lift

The closed, short-scar, non-endoscopic forehead lift technique is performed through four (or occasionally five) small scalp incisions made just posterior to the hairline, and consists of a blind subperiosteal mobilization of a forehead flap centrally and a subgaleal dissection laterally, a release of the temporal line and periosteum over the lateral brow, flap suspension with permanent suture to converging unicortical bony tunnels (or other method of direct cranial suspension of choice) and transpalpebral modification of the corrugator supercilii muscles (if indicated and desired) (Box 67.1).

Marking incisions

Incisions should be marked after anaesthesia has been administered but before prepping and draping is performed. Four incisions approximately 2 cm in length just within existing hairlines are required. In patients with large foreheads and higher hairlines, a central incision is occasionally made to facilitate subperiosteal undermining in the upper central forehead, though this is seldom needed if proper instruments are available.

The two most superior incisions (see 1 in Figure 67.4) are marked radially near the junction of the frontal and fronto-temporal hairlines (Figures 67.4 and 67.5).

In bald or balding men, the superior most incisions can be placed a bit more laterally along the anterior margin of the temporal tuft of hair in the same approximate location. In either case, the incisions should lie just medial to the temporal line and are used to provide access for subperiosteal undermining of the ipsilateral forehead (Figure 67.6). They also are used as points for flap fixation. Making these incisions radially facilitates closure and results in a scar that is less conspicuous than when incisions are made in transverse fashion or placed along the hairline.

The two temporal incisions are marked just within the temporal tuft of hair behind the fronto-temporal hairline (see Figure 67.4). These incisions lie lateral to the temporal line and are used to provide access for subgaleal undermining of the ipsilateral temple and lateral orbital regions (see Figure 67.6). These incisions also provide access for dividing the bridge of tissue along the temporal line between the central subperiosteal dissection of the forehead and the subgaleal dissection of the

Box 67.1 Overview of closed, non-endoscopic foreheadplasty technique

- 1 Four small scalp incisions
- 2 Blind mobilization of forehead in a subgaleal plane laterally and a subperiosteal plane centrally
- 3 Release of the temporal line
- 4 Release of periosteum beneath the lateral brow
- 5 Suture suspension to unicortical bony tunnels
- 6 Transpalpebral (through eyelid incision) corrugator excision (if indicated)

temple and lateral forehead (Figure 67.7), and for dividing the periosteum beneath the lateral two thirds of the ipsilateral eyebrow under direct vision (Figure 67.8). If a facelift is performed at the same time as closed foreheadplasty and the temporal part of the facelift incisions are in a traditional position on the temporal scalp, the upper portion of the facelift incisions can be used to undermine the temporal forehead, divide the bridge of tissue between the subgaleal and subperiosteal planes, and to release the periosteum beneath the lateral two thirds of the eyebrows. In such cases, the separate temporal incisions described above are not necessary.

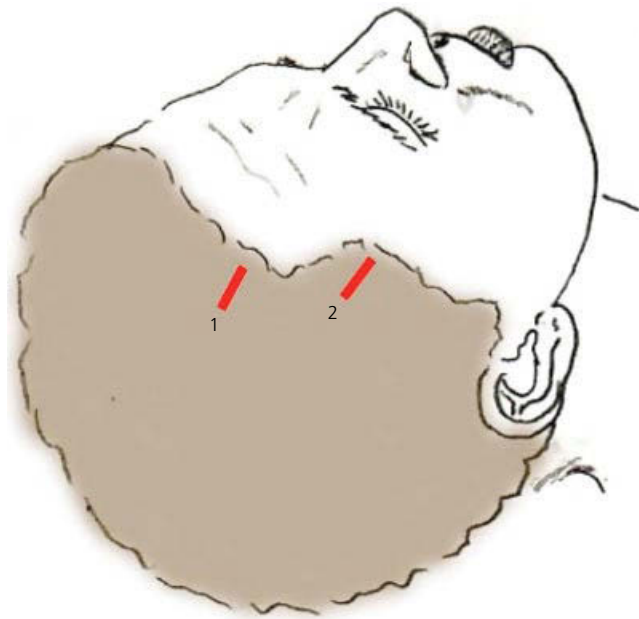


Figure 67.4 Incision plan for closed, non-endoscopic forehead lift. The two most superior incisions (1) are marked radially near the junction of the frontal and fronto-temporal hairlines. The two temporal incisions (2) are marked just within the temporal tuft of hair behind the fronto-temporal hairline. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

Making incisions

Incising the scalp is a key step in forehead lift surgery in which the surgeon exercises direct influence over the quality of the resulting scar. All incisions on the scalp, including the small 'closed' ports used in these procedures, must be made precisely parallel to hair follicles to avoid injury to them. An incision made carelessly or other than in this manner will injure hair follicles and result in peri-incisional alopecia.

After each incision is made, the wound edges are retracted and vessels traversing the wound in the deep subcutaneous plane are grasped and cauterized. Care is taken to avoid thermal injury to the more superficially situated hair follicles, and no cauterization is performed in the plane in which the hair follicles are present. Superiorly, incisions are carried down through the galea and the periosteum is incised. Laterally, dissection is deepened through the galea and the temporalis muscle fascia exposed.

Forehead flap elevation

The plane beneath which the forehead flap is raised in 'closed' foreheadplasty procedures has been the subject of debate and surgeons have advocated both subgaleal and subperiosteal dissections. Most surgeons now seem to prefer the subperiosteal plane, owing to its safety, ease of dissection and the protection it affords the deep branch of the supraorbital nerve. It should be noted that when a 'subperiosteal' technique is used, the entire flap dissection is not in a subperiosteal plane. Medial to the temporal line, the dissection is subperiosteal. Lateral to the temporal line, the dissection is in a subgaleal plane.

Forehead undermining and release is usually begun in the temple regions (Figure 67.9). Wound edges are retracted, the galea incised and the subgaleal plane entered. Typically this is a loose areolar plane that is dissected bluntly with closed Metzenbaum scissors, Boise nasal elevator, or similar blunt instrument, and dissection generally proceeds easily up to and over the lateral orbital rim in subgaleal/pre-periosteal plane in this manner. The dissection is then blindly and bluntly extended



(a)



(b)

Figure 67.5 Incision plan for closed, non-endoscopic forehead lift. (a) Markings for incisions for closed, non-endoscopic forehead lift in a male patient. (b) Markings for incisions for closed, non-endoscopic forehead lift in a female patient. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

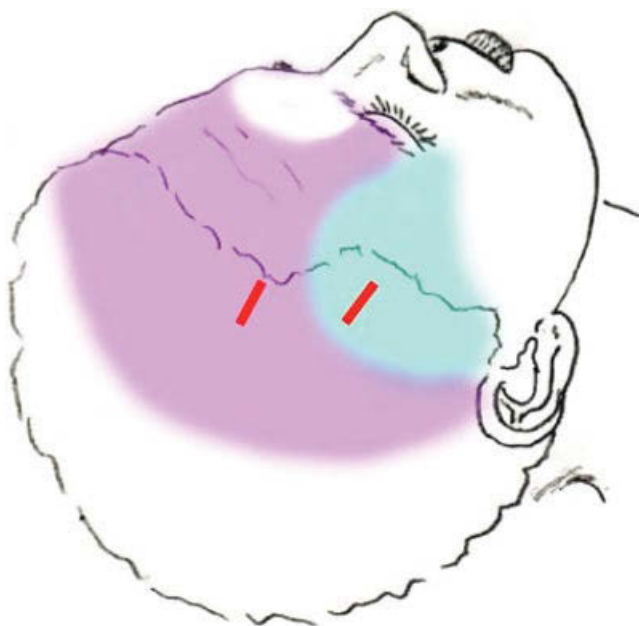


Figure 67.6 Flap undermining in closed, non-endoscopic forehead lift. The incision on the temporal scalp (see 2 in Figure 67.4) is used to undermine the lateral forehead in a subgaleal plane (light green shaded area). The superior incision at the junction of the frontal and fronto-temporal hairlines (see 1 in Figure 67.3) is used to undermine the central forehead in a subperiosteal plane (violet shaded area). Note that subperiosteal undermining is carried posteriorly to accommodate flap advancement, but that the inferior central forehead and glabellar region are not released. The interface between the lateral subgaleal and the medial subperiosteal planes is the temporal line of fusion or 'temporal line'. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

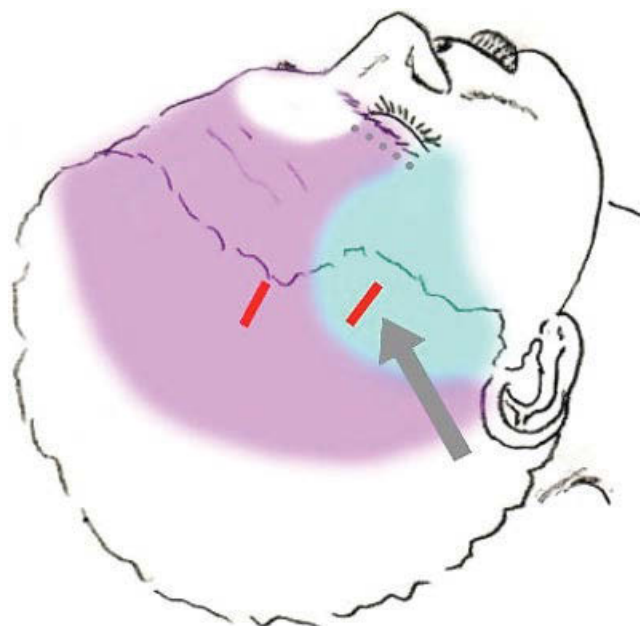


Figure 67.8 Releasing the periosteum over the lateral brow. The lateral incision on the temporal scalp is used to divide the periosteum beneath the lateral eyebrow (dotted line) to mobilize and release the forehead flap. This can be done without an endoscope under direct vision with pistol grip endoscopic scissors (or even Metzenbaum scissors) and small retractor if the patient's head is turned, the table is elevated to eye level, and a fiber-optic headlight is worn. Red lines indicate sites of incisions. Light-green shaded area is area of sub-galeal dissection. Violet shaded area is area of subperiosteal dissection. (Courtesy of T. J. Marten, MD, San Francisco, CA.)

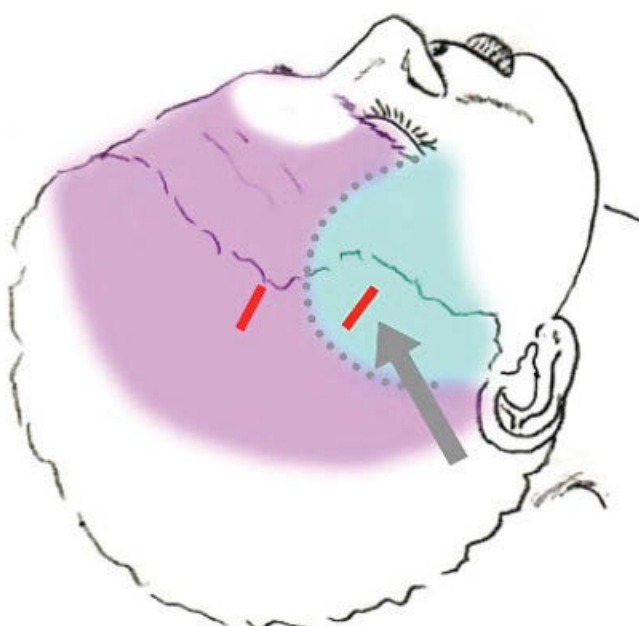


Figure 67.7 Releasing the temporal line. The lateral incision on the temporal scalp is used to divide the bridge of tissue along the temporal line (dotted line) between the central subperiosteal dissection of the forehead and the lateral subgaleal dissection. This can be done without an endoscope under direct vision with Metzenbaum scissors and small retractor if the patient's head is turned, the table is elevated to eye level, and a fibreoptic headlight is worn. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.



Figure 67.9 Subgaleal dissection of temple. Forehead undermining and release is usually begun in the temple regions. Wound edges are retracted, the galea incised and the subgaleal plane entered. Typically this is a loose areolar plane that is dissected bluntly with closed Metzenbaum scissors (as shown), and dissection proceeds up to and over the lateral orbital rim in subgaleal/pre-periosteal plane. The dissection is then blindly and bluntly extended superiorly to the temporal line, and inferiorly over the lateral orbit through somewhat thicker attachments onto the upper zygoma. Due to the location of the access incision and the fact that dissection is made bluntly with a blunt instrument, this can be accomplished without injuring the sentinel vein. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.



Figure 67.10 Subperiosteal dissection of forehead. Wound edges are retracted, the galea and periosteum are incised, and the central-lateral forehead undermined in a subperiosteal plane. This dissection can be quickly made without the aid of an endoscope from temporal line extending posteriorly using an Obwegeser or similar periosteal elevator. Some posterior dissection is required to allow the flap to redistribute itself after it is advanced, and to avoid puckering and gathering posterior to the suspension points when the flap is anchored. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

superiorly to the temporal line, and inferiorly over the lateral orbit through somewhat thicker attachments onto the upper zygoma. Due to the location of the access incision and the fact that dissection is made bluntly with a blunt instrument, this can be accomplished without injuring the sentinel vein.

Once the lateral forehead has been dissected, attention shifts to the most superiorly situated incision located above the junction of the frontal and fronto-temporal hairlines (see 1 in Figure 67.4). Wound edges are retracted, the galea and periosteum are incised, and the central-lateral forehead undermined in a sub-periosteal plane. This dissection can be quickly made without the aid of an endoscope from temporal line to temporal line, extending posteriorly to the approximate location of the coronal suture using an Obwegeser or similar periosteal elevator. Some posterior dissection is required to allow the flap to redistribute itself after it is advanced, and to avoid puckering and gathering posterior to the suspension points when the flap is anchored (Figure 67.10).

As the mid-inferior brow is approached, the inferior aspect of the supraorbital rims should be palpated through infrabrow skin and the location of the foramen or notch from which the supraorbital nerves exits identified. This manoeuvre helps avoid dissection of these areas, which can vary somewhat anatomically from patient to patient. In the majority of cases, a notch ('frontal notch') is palpable on the inferior aspect of the supraorbital rim along a vertical line passing through the medial limbus. In patients with no palpable notch, the nerve usually exits through a foramen, which often is not palpable, more superiorly. Additional care in dissection is taken under these circumstances or an endoscope can be used.



Figure 67.11 Releasing the adherence along the temporal line. Mobilization of the forehead flap continues by dividing the bridge of tissue along the temporal line of fusion between the subperiosteal dissection in the lateral forehead and the lateral subgaleal dissection in the temple, beginning superiorly and extending inferiorly to the lateral orbital rim (see Figure 67.5). As experience is gained, this can be done without an endoscope through the incision on the temporal scalp under direct vision using a fibreoptic headlight and Metzenbaum scissors by raising the table to eye level, turning the patient's head and retracting the flap with a small malleable ribbon retractor or similar instrument as shown. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

When corrugator resection is planned through an eyelid incision ('transpalpebral corrugator myectomy'), the centro-inferior forehead and glabella should not be undermined. Indeed, this is a significant advantage of this technique because it limits central forehead dissection and minimizes the possibility that the medial brow will be elevated.

Mobilization of the forehead flap continues by dividing the bridge of tissue along the temporal line of fusion between the central subperiosteal dissection and the lateral subgaleal dissection, beginning superiorly and extending inferiorly to the lateral orbital rim (see Figure 67.7). As experience is gained, this can be done without an endoscope through the incision on the temporal scalp under direct vision using a fibreoptic headlight and Metzenbaum scissors by raising the table to eye level, turning the patient's head and retracting the flap with a small malleable ribbon retractor or similar instrument. If facelift is being performed concurrently and the temporal portion of the facelift incision is in the temporal scalp this manoeuvre will be even easier (Figure 67.11).

Releasing and mobilizing the lateral brow

To fully mobilize the lateral forehead and obtain optimal elevation of the lateral brow, the periosteum beneath the lateral two thirds of the eyebrow must be divided (see Figure 67.8) (Box 67.2). As experience is gained, this can be done without the aid of the endoscope through the incision on the temporal scalp under direct vision using a fibreoptic headlight and Metzenbaum or a pistol grip endoscopic scissors by raising the table to eye level, turning the patient's head, and retracting the flap with a small malleable ribbon retractor or similar instrument (Figure 67.12).

Box 67.2 Overview of flap mobilization in closed forehead lift

- 1 Blind mobilization of temple in a subgaleal plane laterally
- 2 Blind mobilization in sub-periosteal plane in lateral and upper central forehead
- 3 Release of the temporal line (interface of subgaleal and subperiosteal dissections at temporal line of fusion)
- 4 Release of periosteum beneath the lateral brow
- 5 Blunt mobilization of lateral brow



Figure 67.12 Releasing the lateral brow. To fully mobilize the lateral forehead and obtain optimal elevation of the lateral brow, the periosteum beneath the lateral two thirds of the eyebrow must be divided (see Figure 67.6). As experience is gained, this can be done without the aid of the endoscope through the incision on the temporal scalp under direct vision using a fiberoptic headlight and Metzenbaum or a pistol grip endoscopic scissors by raising the table to eye level, turning the patient's head and retracting the flap with a small malleable ribbon retractor or similar instrument. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

When the flap is retracted, the periosteum is visible as a dense white fascial layer, and its lateral margin corresponds to the release made previously along the temporal line. Division of the periosteum beneath the lateral brow begins by confirming one's position beneath the flap by pushing up upon it with the closed scissors and visually confirming on the skin surface that one is starting in the proper location. The periosteum only is divided in small increments, beginning laterally and working medially to avoid injury to the deep branch or the supraorbital nerve that lies several centimetres medially and more superficially, but in near proximity to the periosteum. Division of the periosteum continues medially over the lateral two thirds of the brow just at the level of the eyebrow. The superficial (main) branch of the supraorbital nerve should remain well medial to the dissection and should not be seen. If facelift is being performed concurrently and the temporal portion of the facelift incision is in the temporal scalp this manoeuvre will be even easier.

After the periosteum of the lateral orbit has been divided, a Boise (or similar) blunt elevator is used to complete the release of the lateral forehead by applying gentle upward and outward

pressure to the upper lateral orbital area. Typically this manoeuvre provides additional flap mobilization that would not be obtained if the periosteum was only divided. Care must be taken, however, even when blunt elevation is made in the lateral orbital and temporal areas, because delicate terminal fibres of the frontal branch of the facial nerve travel through them. Dissection in this manner continues until adequate and symmetric release of the right and left lateral forehead is obtained. This is determined by returning the flap to the forehead, advancing it superiorly by placing gentle traction on the two most superior scalp incisions, and noting the effect on the eyebrows and brows.

Although it is deceptively easy to elevate the lateral eyebrow preoperatively by pulling superiorly on the suprabrow skin, it is in reality much more difficult to do so intraoperatively by pulling from a point more superiorly on a composite forehead flap dissected in a subgaleal or subperiosteal plane. This is particularly true of thick-skinned individuals with advanced brow ptosis in whom standard flap undermining and release often proves inadequate in releasing the lateral brow and providing adequate elevation of the lateral eyebrow. In these cases, additional lateral brow elevation can be obtained by extended undermining of the temple and lateral face.

Drain placement

Experience has shown that a drain, although not absolutely necessary, helps close down deadspace beneath the parietal scalp behind forehead flap suspension points, reduce fluid collections in this area, and limits the amount of postoperative periorbital oedema and ecchymosis experienced by patients. Because of this, drains are used routinely in most procedures.

Drain placement must be performed properly, however, if a beneficial effect is expected and problems are to be avoided. Careless drain placement can result in alopecia, slough, pain, paraesthesias and other problems.

A 7-F round multi-perforated soft Silastic Jackson-Pratt closed suction drain is placed through a central-lateral site of insertion approximately 1–2 finger-breadths posterior to the hairline and 1–2 finger-breadths medial to the right superior incision (Figure 67.13). The stab incision for drain insertion should be made carefully parallel to hair follicles to avoid injury to them. The drain should then be pulled through in a retrograde inside to outside fashion to avoid pulling hair into the wound and anchored with a 4–0 nylon suture tied loosely to the scalp but securely to the drain itself. Loosely tying the suture to the scalp allows for scalp swelling without hair follicle injury and facilitates drain removal. Overtightening the drain suture to the scalp can result in postoperative discomfort, difficulty in suture removal and peri-incisional alopecia.

After the drain has been properly secured at its exit site, its perforated portion is cut to appropriate length and inserted subperiosteally and directed inferomedially across the mid forehead to the opposite side. After incisions have been closed, the drain tube is attached to a small bulb suction reservoir. This

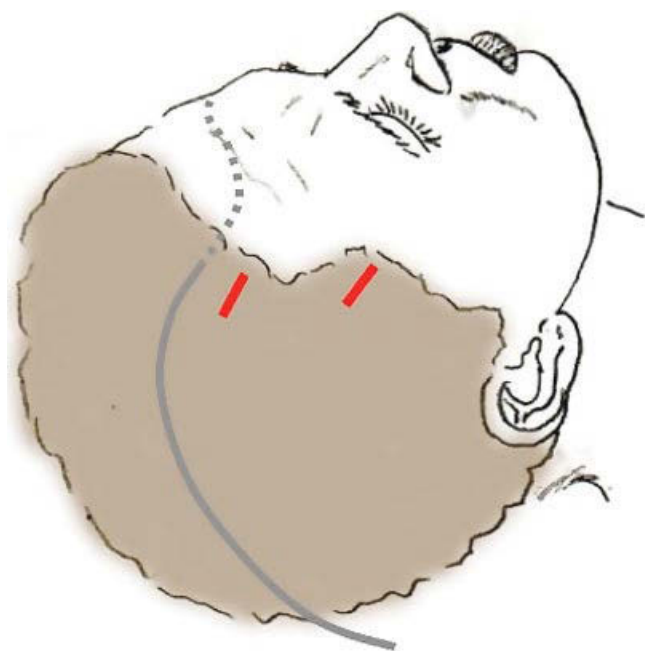


Figure 67.13 Drain placement. A 7-F round multiperforated soft Silastic Jackson-Pratt closed suction drain is placed through a central-lateral site of insertion approximately 1–2 finger-breadths posterior to the hairline and 1–2 finger-breadths medial to the right superior incision. The drain is then directed across the forehead to the opposite side. Red lines indicate sites of incisions. Solid line indicates external part of drain. Dotted line shows approximate path of internal portion of the drain. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

provides continuous low vacuum and is easily managed by most patients while in place.

Flap repositioning

Once the drain has been inserted and secured, the forehead flap is elevated by pulling superiorly and slightly medially on the two most superior incisions and proper eyebrow position and configuration confirmed. For most women, this should be in a soft arch with the lateral portion of the eyebrow lying several millimetres above its medial most portion. For men, the eyebrow should be horizontal or near horizontal, with the medial and lateral portions lying at the same level. In thick-skinned individuals with heavy forehead tissues and brows, some overcorrection laterally is usually required.

In 'closed' procedures, the amount of eyebrow elevation obtained is determined by the extent of flap release, and there is no contribution obtained from scalp excision as is the case in 'open' approaches.

If inadequate brow elevation is seen to be present, or if asymmetry is noted, additional undermining and release are indicated and should be continued until the desired effect is obtained. Tying anchoring sutures tightly cannot compensate for incomplete flap release and will predictably result in hair loss at the anchoring point.

There should be no lateral component of shift when repositioning the forehead flap in closed foreheadplasty procedures,

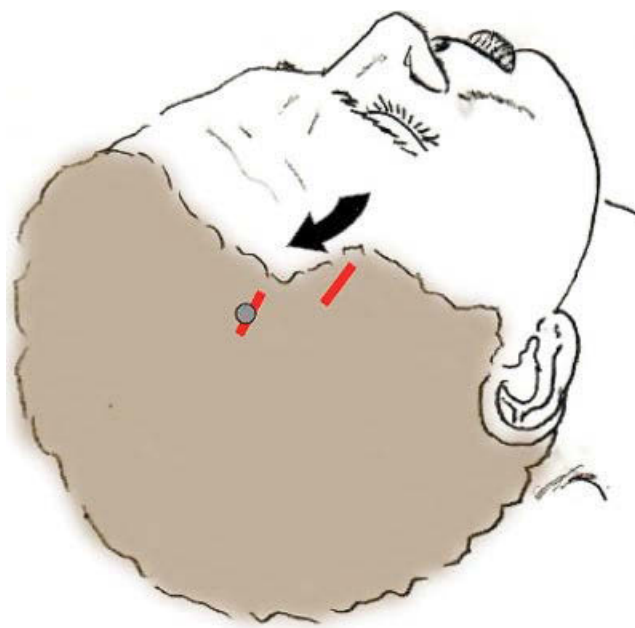


Figure 67.14 Flap anchoring. The periosteal component of the 'closed' forehead flap situated just inferior to the inferior most extent of the superior incision (blue dot) is anchored to converging unicortical bony tunnels in the outer calvarial cortex (grey dot) with a 3–0 braided polyester (Mersylene) suture. Arrow shows proper (superiorly directed or slightly superomedial) direction of flap shift. Red lines indicate sites of incisions. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

and the best biomechanical effect on the brow is obtained when the forehead flap is elevated along a vertical or slightly vertical–medial vector (see arrow in Figure 67.14). Many surgeons mistakenly add a lateral component of shift in a conceptually and technically flawed attempt to smooth glabellar frown lines. Pulling laterally on the forehead will not correct glabellar frown lines, as proper and effective treatment of these requires attenuation or elimination of corrugator supercilii muscle action.

Flap anchoring

Various methods of forehead flap anchoring have been advocated for use in closed foreheadplasty procedures but it should be recognized that the means by which the forehead flap is anchored is not the definitive step in the procedure and that there is no special 'magic' in any one particular method. How the flap is anchored is unimportant as long as it achieves the intended purpose, and success or failure lies largely in the adequate mobilization of the flap – not the particular method of anchoring used.

Methods of forehead flap anchoring that have been described and reported include cranial flap fixation using an internal microscrews, flap fixation using a microcrew and pledget of Gortex (polytetrafluoroethylene), flap fixation using prethreaded metallic anchors (Mitec anchor), flap fixation using an absorbable polyglycolic acid (PGA) cranial peg (Endotine) or PGA (Lactosorb) screw, and suture fixation to

converging unicortical bony tunnels. Other flap-anchoring strategies include suspension using a removable transcutaneous cranial post (large, removable, externalized screw), an external bolster and tissue glues. The use of small 'T' to 'V' excisions of scalp at endoscopic entry ports has also been reported. Each of these methods has advantages and disadvantages that should be considered, including, but not limited to, poor patient acceptance (external bolsters, external removable screws, internal metallic screws), palpability (PGA pegs and screws), expense (Endotine, Mitec), poor healing and hair loss ('T' to 'V' excisions and other attempts to excise scalp) and concerns regarding disease transmission (pooled plasma-derived tissue glues).

Experience has shown that the effectiveness and longevity of closed forehead lift procedures are significantly improved when some sort of secure and permanent direct cranial fixation of the flap is performed, and suturing the periosteum at the leading edge of the superiorly situated incisions (1 in Figure 67.4) to converging unicortical bony tunnels made in the outer cortex of the calvarium with a 3-0 braided polyester (Mersylene) suture has proven to be a convenient and reliable way to accomplish this. This method, unlike many of the others noted above, is easy to perform, reliably results in excellent healing and is readily acceptable to most patients (see Figure 67.14) owing to the fact that no metal screws are used and the fixation is low profile, non-palpable, non-inflammatory, long-lasting and inexpensive.

Once balanced and symmetric release of the forehead flap has been confirmed and the flap advanced, the edge of each superior incision is retracted and a mark made in the centre of each on the cranium in water-soluble surgical ink (methylene blue). These spots mark the sites at which the flap will be anchored. A Desmarres retractor is then used to protect the wound edge on the side being drilled and converging unicortical drill holes made at approximately 30–45° angles. Care must be taken to ensure that minimal pressure on the drill handpiece is applied while holes are being drilled, and that only the outer cortex is penetrated (Figure 67.15).

After converging holes have been drilled in the outer calvarial cortex at marked sites, a 3-0 braided polyester (Mersylene) suture is passed through each drill hole by bending the needle and passing it in a retrograde fashion through each, or by drawing the back end of the suture through the hole by applying a Frazier-tipped suction to one side. At first, this step can prove to be a bit cumbersome, but, as experience is acquired, it can be performed quickly and easily. The suture is then passed through the periosteum just at the inferior aspect of each incision and tied under modest tension only.

Anchoring the flap superiorly provides the needed mechanical effect on the brow and it is typically not necessary or productive to anchor the flap at the temporal incision sites. It is also not necessary, and will make any secondary procedure more difficult, if temporalis muscle fascia is excised in an effort to create suspensory adhesions in the lateral temporal areas.



Figure 67.15 Flap anchoring to converging unicortical drill holes. A Desmarres retractor is then used to protect the wound edge on the side being drilled and converging unicortical drill holes made at approximately 30–45° angles. Care must be taken to ensure that minimal pressure is applied to the drill handpiece while holes are being drilled, and that only the outer cortex is penetrated. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

Incision closure

After flap-anchoring sutures have been placed, irrigation is performed with antibiotic solution and scalp incisions are closed in one layer with simple interrupted sutures of 4-0 nylon. No deep suture is required. If closure is performed meticulously and in a way that provides good wound edge alignment without overly tight sutures, an inconspicuous scar will be obtained (Figure 67.16).

Staples are generally inadequate for closing scalp incisions in open and closed foreheadplasty procedures in that a properly made incision on the scalp is typically bevelled to some extent so as to remain parallel to hair follicles and avoid injury to them, and staples are designed to close wounds with perpendicular edges. Attempting to staple an incision with bevel edges often results in overriding of wound edges and poor wound edge alignment. The use of staples is also frequently uncomfortable and disturbing to patients. A ratcheting staple and external screw technique should not be used to suspend the flap in closed foreheadplasty procedures. Such a plan places excessive tension on the scalp tissue in the plane of the hair follicles and commonly results in hair loss.

Corrugator myoplasty

Once flap mobilization, repositioning, anchoring and incision closure is complete, attention turns to the corrugator myoplasty, if indicated and/or desired by the patient. Attenuating corrugator hyperfunction forms the basis of the treatment of glabellar frown lines and is adequate for many patients with mild to moderate problems. The expectations of patients with marked muscle hyperfunction and deep lines must be properly set, however, and all patients must be made aware that the goal of



Figure 67.16 Incision closure. After flap-anchoring sutures have been placed, scalp incisions are closed in one layer with simple interrupted sutures of 4–0 nylon. (Note that the incision on temple scalp had already been closed.) Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

surgical treatment of corrugator hyperfunction and glabellar frown lines is a reduction in frown-line intensity, and not complete elimination of all glabellar movement. Patients should also be made aware that they may want or need to undergo neurotoxin (botulinum toxin (Botox)) injections, even after properly performed procedures.

Proper management of the corrugator supercilii muscles requires a detailed understanding of their anatomy, and their relationship with adjacent agonist and antagonist muscles. The corrugators are obliquely oriented muscles that originate on the medial most portion of the supraorbital ridge. The muscles traverse superolaterally to and insert on the skin overlying the medial and mid brow. The corrugators are intimately associated with a group of important agonists and antagonists that together are responsible for the expression of a wide range of human emotions.¹⁵ These include the procerus, frontalis, orbicularis oculi and the depressor supercilii muscles. The limits of these neighbouring muscles are not always easily defined, and as the corrugators are followed laterally toward their insertion they are seen to overlap, override, intermingle and merge with them, and these relationships allow for complex interactions and are important when contemplating surgical modification of muscle action.

A careful consideration of the anatomic arrangement of the corrugator supercilii muscles makes clear their effect on the eyebrow and the bulges and lines that develop upon their contraction. Each muscle is arranged so that its fibres exert action in several directions, and as such, they exert different effects at various locations along the brow. Over the medial third of the eyebrow, the corrugator acts as an antagonist to the frontalis and an agonist to the procerus and superior medial portion of the orbicularis oculi. There is no specific antagonism to medially directed contraction of the corrugator, however, and thus no opposing force to its medially directed pull on the

brow. This explains why, upon cursory consideration, vertical glabellar wrinkling seems to be the overriding effect of corrugator action. It also explains why resecting the corrugator, in and of itself, can result in medial brow elevation, even if no undermining and advancement of a forehead flap is made.

The corrugator muscle action results in a complex action on the medial aspect of the eyebrow and the inferior central forehead, resulting in vertical glabellar wrinkling and an angry or disapproving appearance. Traditionally, near-total corrugator myectomy was performed to diminish these expressions using an open or endoscopic technique, and when properly performed was effective in doing so. Traditional myectomy, performed using either of these techniques has several disadvantages, however, including disruption of attachments and ligaments that hold the medial brow and eyebrow inferiorly. Traditional open or endoscopic myectomy also typically produces some temporary and occasionally permanent paraesthesia and dysaesthesia due to dissection of the supratrochlear nerves. These problems can often be diminished or avoided if a modified technique and transpalpebral approach is used, in which the corrugator muscle is divided or divided and partially resected lateral to the supratrochlear nerve branches and a fat graft is interposed to defunctionalize the muscle by preventing the divided segments from reattaching upon healing. This approach will henceforth be referred to as 'corrugator myoplasty' and the involved steps are described in the following paragraphs.

Dissection of the corrugator supercilii muscles is facilitated and bleeding is diminished if the medial brow is hyperinflated by injecting a dilute local anaesthetic-adrenaline solution and an adequate time is allowed for a proper anaesthetic and haemostatic effect. The surgeon should also wear a headlight, and fine-tipped cautery, Ragnell retractor and a Frazier-tipped suction should be available.

Upper blepharoplasty incisions are made according to the preoperative plan and need not be modified when transpalpebral corrugator myoplasty is being performed. If a patient does not need upper blepharoplasty but desires frown line reduction, consent is obtained to make an incision along the upper lid tarsal crease to provide access to the corrugator muscle and to allow corrugator muscle myoplasty to be performed. When the advantages of the technique are explained to patients, most will allow an eyelid incision to be made in such situations and, if it is closed properly, the scar will usually be difficult to find once healed. If patients do not wish to have an upper eyelid incision, the muscles can be treated in a traditional endoscopic fashion, or by other means.

The superior margin of the blepharoplasty incision is retracted by the assistant using two 10 mm double-pronged skin hook retractors and dissection is carried down to the orbital septum using needle-tipped cautery and low electrocautery current (Figure 67.17). Dissection is then continued superiorly superficial to the septum up to the supraorbital rim in a preseptal plane. Typically, this dissection is made in a loose areolar



Figure 67.17 Transpalpebral approach to the corrugator supercilii muscle. The superior margin of the blepharoplasty incision is retracted by the assistant and dissection made superiorly superficial to the septum up to the supraorbital rim using needle-tipped cautery and low cautery current in a preseptal plane. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

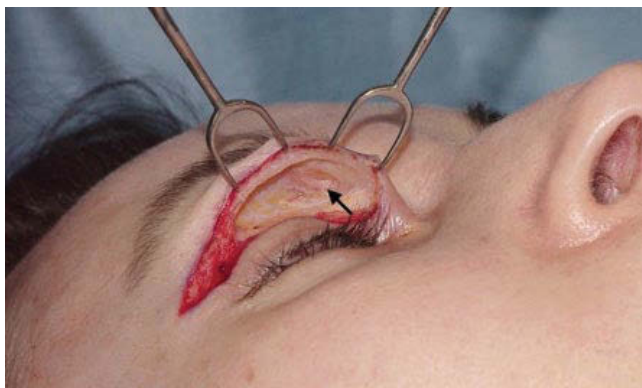


Figure 67.18 Transpalpebral exposure of the corrugator supercilii muscle. As dissection is made superiorly on top of the orbital septum to the supraorbital rim, the inferior part of the corrugator supercilii muscle (arrow) becomes visible. Source: Courtesy of T. J. Marten, MD, San Francisco, CA.

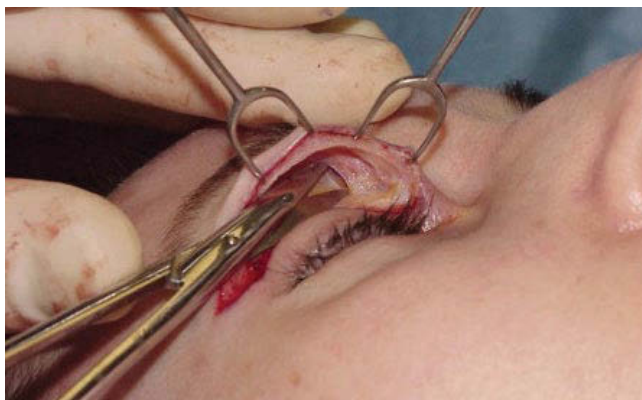


Figure 67.19 Transpalpebral dissection of the corrugator supercilii muscle. Once the corrugator is identified, the tips of a pair of tenotomy scissors are introduced between the underside of the muscle and the brow, and they are used to bluntly dissect the plane between the muscle and the bony periosteum. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

plane and as the medial aspect of the supraorbital rim is approached, the outline of the inferior portion of the corrugator supercilii muscle will be seen (Figure 67.18).

Once the corrugator is identified in this fashion, the tips of tenotomy scissors are introduced between the underside of the muscle and the brow bone, and they are used to bluntly dissect the plane between the muscle and the bony periosteum (Figure 67.19). This dissection is performed well medial to the supraorbital nerve, which is typically not seen and need not be specifically identified during the procedure. If the glabella has been hyperinflated with local anaesthetic-adrenaline solution before muscle dissection, minimal bleeding is usually encountered. If bleeding is encountered, however, it is best controlled by placing firm direct pressure on the skin overlying the region against the bony brow for several minutes or until it stops.

Next, the tips of the tenotomy scissors are introduced between the anterior surface of the muscle and the brow skin, and they are used to bluntly dissect the plane between them (Figure 67.20). If the brow has been hyperinflated with local anaesthetic-adrenaline solution before its dissection, little bleeding will typically be encountered during this part of the dissection as well. If bleeding is encountered, it is best controlled as previously described by placing direct pressure on the skin overlying the region until it stops. Once this manoeuvre is complete, the corrugator is isolated and can be grasped with a forceps or a similar instrument (Figure 67.21).

Once the corrugator has been isolated and separated from adjacent structures, it is divided or a small segment is resected (Figure 67.22). This is accomplished by introducing one blade of the tenotomy scissors between the corrugator muscle and the brow bone, and the other between the muscle and the skin. The scissors are then turned parallel to the supraorbital and supratrochlear nerves and the muscle divided between them. If the brow has been hyperinflated with local anaesthetic-adrenaline solution before its dissection, little bleeding will typically be encountered when the muscle is divided. If bleeding is encountered following this manoeuvre, it is best controlled by packing the wound with the corner of a sponge and placing direct pressure on the skin overlying the region until it stops. Alternatively, bleeding can often be controlled directly in some cases by placing a Ragnelle-type retractor in the wound, applying suction with a small Frazier suction, and cauterizing oozing areas with a fine-tipped cautery under direct vision. This is most readily accomplished, when necessary, while the surgeon wears a fiberoptic headlight.

Once the corrugator has been divided and haemostasis obtained (if necessary), the cut edges of the muscle retract and a gap between the cut edges will be seen (Figure 67.23). If fat is available from elsewhere in the face (e.g. from a blepharoplasty, facelift or neck lift procedure), it is used as an interposition graft to defunctionalize the muscle and to ensure that smooth brow contours will be present once healing is complete (Figure 67.24). If this is not done, the muscles will

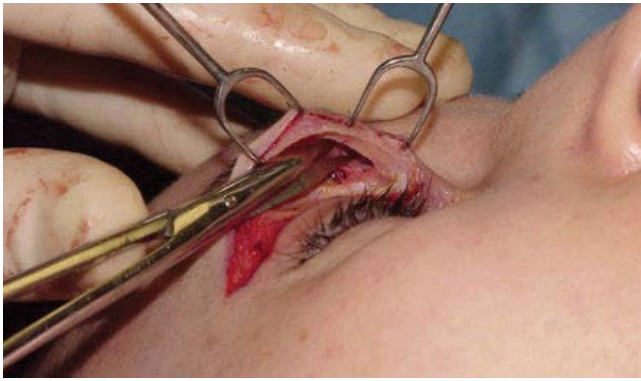


Figure 67.20 Transpalpebral dissection of the corrugator supercilii muscle. After the deep side of the muscle has been dissected, the tips of the tenotomy scissors are introduced between the anterior surface of the muscle and the brow skin. They are then used to dissect the plane between them. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

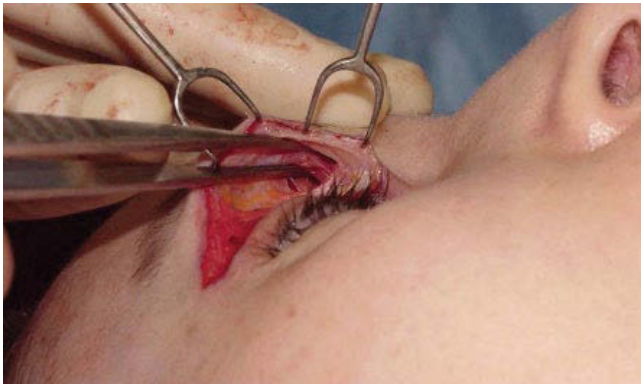


Figure 67.21 Transpalpebral dissection of the corrugator supercilii muscle. Once the superficial and deep surface of the corrugator has been dissected, its central portion will be separated from adjacent structures and can be grasped with a DeBakey forceps or a similar instrument. It can then be divided (see Figure 67.14) or subtotally resected as desired. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

predictably heal back together and minimal improvement will typically be obtained. If fat is not available from the face, the small amount needed can be harvested through a small incision in the suprapubic or other concealed area where adequate fat is present. Alternatively, the head of the muscle can be subtotally resected by carefully picking it out from between the supratrochlear nerves with a fine-tipped haemostat (Figure 67.25). When this is done, more forehead dysaesthesia and upper eyelid ecchymosis will be more likely to occur and should be expected.

Fat grafts are trimmed to fit the defect and typically measure approximately 10×10×10 mm. Fat grafts are most easily secured using a transcutaneous pullout suture of 6-0 nylon (see Figures 67.24 and 67.26). This is placed while the wound is held open with a Ragnell retractor and 10 mm double-pronged skin hook, and is introduced through the brow skin just at the superomedial aspect of the eyebrow and brought out



Figure 67.22 Dividing the corrugator supercilii muscle. Once the corrugator has been isolated, one blade of the tenotomy scissors is inserted between the corrugator muscle and the brow bone, and the other between the muscle and the skin. The scissors are then turned parallel to the supraorbital and supratrochlear nerves and the muscle is divided between them. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.



Figure 67.23 Completed transpalpebral division of the corrugator supercilii muscle. Once the corrugator has been divided and the cut edges of the muscle retract and a gap appears. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

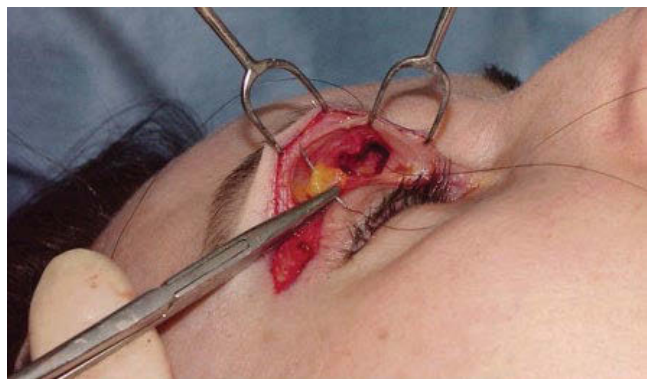


Figure 67.24 Transpalpebral placement of interposition fat graft. Fat obtained from elsewhere in the face (e.g. from a blepharoplasty, facelift or neck lift procedure) is used as an interposition graft to prevent the cut muscle edges from re-adhering to each other and to defunctionalize the muscle. The fat graft also ensures that brow contours will be smooth once healing is complete. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.



Figure 67.25 Transpalpebral myectomy. If a fat graft is not available, the corrugator muscle can be subtotally resected by excising a portion of it between the supratrochlear and supraorbital nerves with a fine-tipped haemostat. When this is done, more forehead dysaesthesias and upper eyelid ecchymosis should typically be expected. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.



Figure 67.26 Securing the interposition fat graft. Fat grafts are most easily secured using a transcutaneous 6-0 nylon pull-out suture. Placed while the wound is held open with a Ragnell retractor as shown, the suture is introduced through the brow skin just at the superomedial aspect of the eyebrow and brought out through the eyelid incision between the divided muscle segments. The needle is then passed through the fat graft and then back through the eyelid incision between the muscle segments and out of the skin near the spot it entered. As the suture is pulled up and tied, the fat graft is drawn up into the incision and held between the divided muscle segments. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

through the eyelid incision between the divided segments of corrugator muscle. The needle is then passed through the fat graft, and then back through the eyelid incision between the muscle segments and out of the skin near the spot it entered. As the suture is pulled up and tied, the fat graft is drawn up into the incision and held between the divided muscle segments (see Figure 67.26).

Because the knot is small and sits at the superior edge of the eyebrow, it is often not noticed by patients during the recovery process (Figure 67.27). The eyelid incision is then closed with a simple running suture of 6-0 polypropylene (Prolene) or another suture of choice.

Not all patients today will need or want corrugator muscle modification surgery that was in one form or another more or less a routine part of foreheadplasty procedures performed in the past. Many patients will now be encountered who are using neurotoxins (botulinum toxin, Botox) to control corrugator hyperfunction who are happy with the improvement they obtain from it (albeit a temporary one) and who wish to continue to use it. Indeed, in many instances it could be argued that neurotoxins produce a more predictable, complete and trouble-free improvement than surgical muscle modification does, and that its use has largely supplanted muscle surgery for treatment of glabellar frown lines. In these cases transpalpebral myoplasty (or other forms for corrugator muscle modification) need not be performed and the foreheadplasty procedure is undertaken to optimize eyebrow position and configuration only (tilt the lateral eyebrow up), and it is referred to as a 'temple lift' to distinguish it from more comprehensive 'forehead lift' and/or 'brow lift' procedures. It is important that the patient has a clear understanding of whether or not corrugator muscle surgery will be performed, however, and when corrugator muscle modification is planned in the authors' clinic 'corrugator muscle modification' is typically listed on the patient's informed consent document and procedure price quote, and



Figure 67.27 Completed transcutaneous anchoring of the interposition fat graft. Because the suture knot is small and sits at the superior edge of the eyebrow (arrow), it is typically not noticed by patients during the recovery process. Source: Courtesy of T.J. Marten, MD, San Francisco, CA.

additional charges are applied for the additional time and materials involved in carrying out the procedure.

Dressings

After all planned procedures have been completed and all incisions have been closed, the patient's hair is washed with shampoo and conditioner and, if long, placed in a soft braid. The hair is then allowed to dry or is blown dry in the recovery room. No dressing is required or applied. Patients are typically discharged with a hat, scarf and sunglasses following the procedure.

Postoperative care

Proper postoperative care of the foreheadplasty patient ensures the best result with the fewest problems and complications. Although most patients are treated as outpatients unless a face-lift or related procedures are simultaneously performed, all are discharged to the care of a responsible adult third party with a specific set of written aftercare instructions.

Cool compresses

Patients are asked to rest quietly and apply cold compresses to their forehead and eyes for 15–20 min of every hour they are awake for the first 3 days after surgery. For most patients, oedema peaks at about this time. It is not necessary or productive to apply ice compresses continually throughout the day or at night.

Medications

All patients are provided oral (usually non-narcotic) analgesics, sleeping pills, oral dissolving antiemetic tablets, preservative-free ophthalmic ointment and preservative-free 'artificial tears' solution with instructions for their use. Patients are required to use ophthalmic ointment each night for the first three weeks after surgery or until all signs of eye irritation are gone. Artificial tears are used throughout the day on an as-needed basis.

Drain removal

The forehead drain is removed the morning after surgery if the patient stays overnight in the aftercare facility, or at the first postoperative visit.

Wound care

Patients are instructed to begin a daily routine of showering and shampooing no later than 3 days after surgery even if their drains are still in place. This helps remove crusting about the suture lines, keeps incisions clean and usually improves the patient's general outlook and wellbeing. Patients are also informed that they need not be as thorough as usual when washing their hair and assured that shower water, shampoo and conditioners are not harmful and will not interfere with healing or cause infection.

Hair care

Patients are allowed to use their usual hair-care products but must be warned that their scalp may be partially numb after surgery and that they must be careful to ensure that shower water is not too hot and that hair dryers are not used on high heat settings. Patients are also instructed not to 'perm', tint, dye, highlight, colour or otherwise chemically treat their hair for two weeks after surgery to limit the chance of hair breakage or hair loss. 'Hot curlers' and 'curling irons' must be used with care for several months following forehead lift surgery as patients might unknowingly burn their foreheads and scalps. Patients are advised to apply hair gel, hair cream, hair spray and similar products sparsely or preferably not at all, until all sutures have been removed.

Suture removal

Sutures are typically removed over a period of 7–9 days after surgery. When incisions have been made along the hairline (hairline-lowering foreheadplasty and partial hairline forehead lifts) the simple interrupted sutures of 6–0 nylon are removed 5 days after surgery and half-buried vertical mattress of 4–0 nylon are removed 7–9 days after surgery. When incisions have been made within the scalp (small incision or coronal procedures) the simple interrupted sutures of 4–0 nylon are removed 7–9 days after surgery. Patients are then checked 2–3 weeks after surgery to ensure that no stray sutures are present and that all have been removed.

Return to work after surgery

How long it is before patients return to work and social life depends upon their tolerance for surgery and anaesthesia, their capacity for healing, the type of work they do, the activities they engage in and how they feel overall about their appearance. Typically, they are advised to set aside 7–9 days, if possible, to recover from forehead lift surgery. Additional time off is sometimes required or desired before an important outing, vacation or similar event.

Patients making uneventful recoveries may return to work and limited social activity when they feel up to it, and many patients have been able to attend social functions a few days after surgery. It is often wise for patients to begin with a limited workday, however, and to adjust their schedules thereafter as tolerated. If a patient's work requires more strenuous activity or physical labour, a longer period of convalescence may be needed. Patients are advised not to drive until they are feeling well, their vision is clear and they are off pain medications and sleeping pills.

After-surgery diet

Patients are allowed to take their usual diet as tolerated after surgery but are advised to restrict their intake of salt. Patients are asked to abstain from the intake of alcohol for two weeks after surgery and until they are no longer taking narcotics, paracetamol (acetaminophen) or sleeping medication. Patients are asked to abstain from smoking and avoid secondhand smoke in the immediate postoperative period.

After-surgery activity

In the first 10–14 days after surgery, patients are advised to avoid bending, stooping and all strenuous activity, exercise and sports. Thereafter, they may begin to exercise, gradually working up to their presurgical level of activity.

Case examples

Figures 67.28, 67.29 and 67.30 show patients before and after the procedure.



Figure 67.28 (a) Patient, age 55, preoperatively. Note that she has a somewhat tired, stern and angry appearance even though her face is in repose. She has had no prior plastic surgery. (b) Same patient, 1 year and 6 months following small-incision non-endoscopic forehead lift. Note that her eyebrow position and configuration has been improved and that she has a more rested and agreeable appearance. A facelift, neck lift, upper and lower eye lifts, chin augmentation and fat transfer to the cheeks and lips have also been performed. (c) Preoperative view of same patient frowning. Note that she has strong frown muscles that produce deep creases and an intense angry appearance. (d) Same patient, 1 year 6 months following small-incision non-endoscopic forehead lift with transpalpebral corrugator myoplasty. Note that she is trying to frown but frown intensity is markedly reduced. Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.

Summary

Each patient presenting for improvement of their forehead appearance will present with different problems and concerns and no one forehead lift procedure will be best for all patients. Foreheadplasty techniques using a coronal incision offer certain

advantages over closed procedures and should result in an inconspicuous scar and few complications if skilfully performed. These advantages include that they provide an actual reduction in forehead scalp and/or skin redundancy, and as such may provide better brow elevation and a more durable and sustained improvement. In practice, a procedure using a coronal incision



(a)



(b)



(c)



(d)

Figure 67.29 (a) Patient, age 49, preoperatively. Note that she has suboptimal eyebrow position and configuration that imparts a somewhat angry and disapproving appearance even though her face is in repose. She has had prior upper and lower blepharoplasty performed by another surgeon. (b) Same patient, 14 months following small-incision non-endoscopic forehead lift. Note that she has a more rested and agreeable appearance. A facelift, neck lift, upper and lower blepharoplasties and fat transfer to the cheeks, lips, jawline and infraorbital areas have also been performed. (c) Preoperative view of the same patient frowning. Note that she is able to produce deep creases and has an angry appearance. (d) Same patient, 14 months following small-incision non-endoscopic forehead lift and transpalpebral corrugator myoplasty. Note that frown intensity is markedly reduced. Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.)

may be the procedure of choice for patients with abnormally low hairlines who wish their hairline to be raised and placed in better proportion with the rest of their face.

Many patients seeking rejuvenation of their foreheads are troubled by high hairlines and the prospect that surgery will make their problem worse. Hairline elevation occurs in both coronal and closed endoscopic techniques, and hairstyles cannot always conceal an overly high forehead. A high forehead also appears unnatural,

masculine and detracts from an otherwise attractive and agreeable appearance. Unlike procedures using a coronal or endoscopic technique, procedures using an incision or partial incisions along the hairline allow hairline position to be maintained or lowered and hairline shape to be optimized, and the improvement thus gained may outweigh the drawback of the resulting scar.

Experience has shown that it is not necessary to use an endoscope to mobilize and release the forehead and modify the corrugator



(a)



(b)



(c)



(d)

Figure 67.30 (a) Patient, age 47, preoperatively. Note that she has a somewhat tired, angry and disapproving appearance, even though her face is in repose. She has had no prior surgery. (b) Same patient, 1 year and 7 months following small-incision non-endoscopic forehead lift. Note that eyebrow position and configuration have been improved and that the patient has a more relaxed and agreeable appearance. A facelift, neck lift, upper and lower blepharoplasties and fat transfer to the cheeks, lips, jawline and infraorbital areas have also been performed. (c) Preoperative view of same patient frowning. Note that she has strong frown muscles that produce deep creases and an intense angry appearance. (d) Same patient, 1 year and 7 months following small-incision non-endoscopic forehead lift and transpalpebral corrugator myoplasty. Note that frown intensity is markedly reduced. Source: Courtesy of the Marten Clinic of Plastic Surgery, San Francisco, CA, with permission.

supercilii muscles if forehead anatomy is understood, the operation is appropriately planned and a transpalpebral approach to the corrugators is used. Although an endoscope can be used for these purposes if desired, a procedure in which an endoscope is not necessary saves time, avoids problems related to the device itself (e.g. fogging, mechanical failure) and avoids the costs of purchasing, leasing and maintaining expensive related equipment. In addition, transpalpebral corrugator myoplasty, when used in conjunction with closed mobilization and resuspension of the fore-

head, not only provides a means to perform a closed foreheadplasty without an endoscope, but also gives a method by which medial brow elevation can be minimized or avoided.

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CHAPTER 68

Upper eyelid rejuvenation

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Introduction

Upper eyelid surgery is one of the oldest and most common surgical procedures. Descriptions of eyelid surgery were recorded in Indian *Sanskrit* texts more than 2000 years ago.¹ Such eyelid surgery is consistently one of the most common reconstructive and appearance-related procedures and is performed throughout the world. But many people doing this operation are mistaken about its simplicity, as well as the common complications that may be expected when it is done without proper knowledge, training and understanding of the operation and its potential outcomes.

Upper blepharoplasty can indeed be performed under local anaesthesia, and in an office setting. Yet surgeons should not underestimate this detail-oriented, complication-prone procedure as quick and easy because it looks and seems simple. It can, in selected cases, be done in a short period of time, and is widely considered a relatively quick procedure, with a reliable source of insurance income generation in some countries. But we, as surgeons must not fail to choose the needed and indicated (and perhaps more complicated – and longer) periorbital operation(s), in favour of the one that seems quicker, simpler and predictably paid for by third parties.

Indications for surgery

Upper eyelid surgery is done for both functional and appearance reasons. Visual fields are profoundly affected by the upper eyelids, either by excess tissue weighing down on the lids and lashes, or by the attenuation of eyelid – and brow – muscles causing droopy eyelids. Commonly the brow and forehead play a major role in functional and appearance, in lid deformity, lid asymmetry and eyelid ptosis, both unilaterally and bilaterally, often making the corrective operation more complicated functionally, aesthetically (and financially).

Cosmetic and functional correction of baggy eyes is sought by addressing the amount of eyelid, brow and forehead skin

excess, the position of the lid crease, the tilt of the eye, any bulging puffiness, hollowness and other potential asymmetries. This is usually done by carefully examining the patient who is seated vertically, directly in front of the examining surgeon.

Diagnosing lid ptosis preoperatively is important. In the senior author's (RSF) huge number of courses on lid surgery taught around the world, he would traditionally start by asking the class for the most common cause of postoperative lid ptosis. The answer was so simple, but never answered correctly – except by an occasional former student. The most common cause of postoperative ptosis is ... undiagnosed preoperative ptosis. Simple but true.

Another way to separate true ptosis from the weight of the brow causing one or both lids to sag, is to simply elevate the brow slightly with a finger, and see if the lid ptosis disappears. Doing this bilaterally can also make the surgeon aware of unrecognized lid retraction, hidden by brow ptosis, which could result in a most unattractive outcome from lifting the brow.

Preoperative evaluation

Good preoperative evaluation is the key to achieving great results with blepharoplasty. The periorbital region should be evaluated as a whole, including the brow position and lower eyelid characteristics. The effects of adjacent structures such as eyebrows (and sometimes lower eyelids) on the upper eyelids should be pointed out to each patient. Commonly a person's right brow is lower than their left (more than 80% of the time in senior author's personal experience) with the patient's left being lower in only 10% of the time, and the brows proving equal in approximately 7%. These statistics do vary some degree between ethnicities. Know, for sure, however, that upper blepharoplasty does not correct eyebrow ptosis, nor can it overcome any imperfection of the lower eyelid and the orbital bone structures. The senior author pointed out many years ago the importance of combining frontal lift with upper eyelid surgery for the best eyelid results in many, if not most, eyelid patients, while occasionally choosing browlift alone to clean up the brow, lids and upper orbit.²

Often we use that same brow access to restore youthful characteristics of the lower lids (via canthopexy into the bone) for canthal restoration and lower lid correction.^{2,3}

Careful and thorough preoperative evaluation identifies the problems applying to each selected patient, and a surgical plan is crafted accordingly. General physiology of the eye, such as tearing, dry eyes, increased globe tension (diagnosed with a simple tenometer test) and careful diagnosis of globe protrusion is essential. Consultation assuring appropriate candidacy for surgery, confirmed by a friendly ophthalmologist, should be sought when deemed appropriate. General health conditions are also important considerations to ensure the patient is a good surgical candidate for elective surgery, with minimal risks of bleeding, infection, abnormal scarring and adverse medical complications.

Few eye surgeons understand how and why women in particular *always* raise their eyebrows, smile slightly and tilt their heads posteriorly every time they look into a mirror, or have someone look directly at their face, as well as each time a camera is pointed toward their face (especially in the doctor's surgery). Because of this, we rarely have accurate preoperative and postoperative pictures by which to gauge the way our patients look before we do their surgery. As well as hindering our assessment of their postoperative appearance, this behaviour leaves us uncertain of the quality of our patients' surgical results. A large number of patients seek out plastic surgical repair because of a coincidental snapshot looking so different from their expressions and looks in professionally prepared photographs, or their own glances into the many mirrors before which they have posed each day.

Eyelid surgery is commonly referred to in fashion magazines as 'eye lifts'. In general, 'eye surgery' is anything but a 'lift'! Brows are continually and commonly raised to the patient-learned attractive level, even in youthful people. This is done not only to raise the eyebrows and keep them from interfering with vision, but also to convey a more attractive, more youthful 'look'. This is especially significant when being looked at and scrutinized when looking into a mirror, or being 'captured' by a casual camera.³ As mentioned earlier, excision of eyelid tissue encourages the brows to drop, exaggerating that look the person has tried so hard to prevent – looking older, more tired and angry as well. This detrimental appearance is caused by a relaxation-drop in the lateral-upward pull of the frontalis muscle (relaxation of which is responsible for creating a frown), which inserts primarily into the central third of the brow musculature. This lessens the upward pull of the frontalis muscle, allowing a greater prominence of the now less opposed frown activity of the corrugator musculature, causing exaggeration of the frown, while the brows (especially in their medial aspect), and often the upper lids come plummeting down too.

From time to time we see elderly patients in whom maximal brow elevation is still insufficient to have comfortable forward vision, even with the head tilted backwards. In these patients significantly large amounts of upper lid-juxta-brow skin can

often be sacrificed, allowing the patient's profound brow elevation to now have a sufficient effect to have good forward vision. In these patients simple upper blepharoplasty, often exaggerated, is of immense value, even when the brow eventually drops a bit. These patients will be 'blepharoplasty happy', and some even experience significantly improved facial appearance!

In less elderly people the more lid skin removed, the more the brow will drop (making the person look older and more tired) as well as allowing the relaxation of brow, making that same person look more angry and unhappy. The one positive feature is that transverse forehead wrinkles generally diminish to some degree, often enough to significantly benefit a person's looks, and that alone does a great deal to counteract the old, tired and often angry appearance.

One can observe this phenomenon by having the patient sit relaxed in front of you, with eyes closed. Gently stroke the brow and you will see the frown quickly develop as the brow relaxes. When the patient is totally relaxed, note (and record) the symmetry of the eyebrows, and then press the brow skin firmly down against the skull. Holding tight, ask the patient to open his or her eyes, and carefully notice how far caudal the brow is capable of descending by blocking elective brow and lid elevation. This type of examination shows the surgeon where the greater problem commonly lies – in correcting brow position rather than in excising seemingly excess lid skin. This is true for patients as young as in their twenties, thirties and forties, or even in or before their teens. Performed well, browlifting is often far more important than correction of the upper lids, but generally a lot more expensive! Realize, however, that in many patients, especially older ones, lid tissue removal is an important part of the repair, but more so when combined with brow and/or frontal lifts. Remember, if you do nothing to correct the brow, your result is likely to be disappointing to both you and your patient.

Anatomy

The upper eyelid has eight layers that are relevant to surgery, from superficial to deep: skin, orbicularis muscle, preseptal or retro-orbicularis oculi fat, orbital septum, orbital fat, levator palpebrae muscle/aponeurosis, Müller's muscle/tarsal plate, and conjunctiva (Figure 68.1).⁴ The lacrimal gland lies at the same depth as orbital fat, and it is considered as contained in the lateral compartment, whereas orbital fat is organized into medial and middle compartments. The pretarsal portion of skin adheres to underlying layers through a series of septa, forming the eyelid crease and fold, which may be absent in East Asian patients; creating or adjusting the eyelid crease is performed with this adhesion in consideration. The palpebral muscle and Müller's muscle determine the degree of eyelid opening, with somatic nervous control for palpebral muscle and autonomic control for Müller's muscle; correction of ptosis involves these muscles. Orbital fat and preseptal fat contribute to the bulging and puffiness of the eyelid. The line where the orbital septum

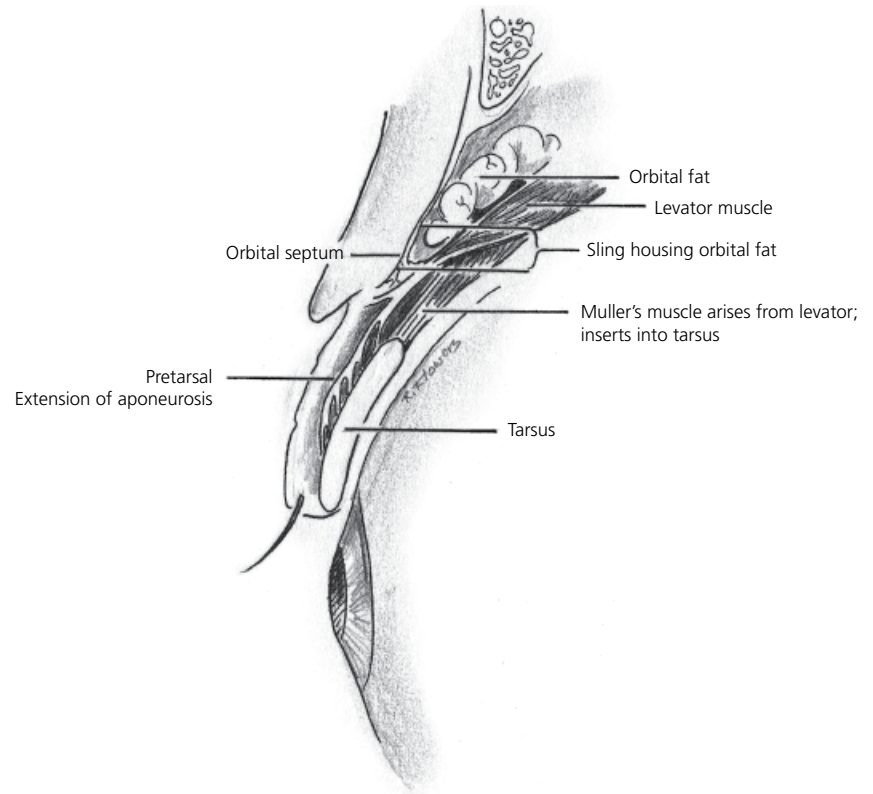


Figure 68.1 Essential surgical elements of upper eyelid anatomy.

fuses with levator muscle creates the depth of the upper eyelid, which can appear hollow if this line is retracted. The surgeon should keep in mind the correlation between these anatomical structures and the eyelid appearance and function while performing the art of upper eyelid surgery.

Operative techniques

The operative techniques should address the concerns detected in the preoperative evaluation, layer by layer of the eyelid.⁵

Skin incision and excision

Mark the incisions while the patient is sitting upright. The amount of skin excision will depend on whether a browlift accompanies the upper eyelid surgery or not. Try a combination of a frontal lift with upper blepharoplasties, when possible, to achieve better outcomes. Know that only about 1 out of 10 patients have brows that do not drop precipitously as a result of eyelid skin excision.

Marking is done prior to local anaesthetic infiltration, first with eyes open and the patient sitting up for determination of the level of eyelid and brow ptosis (as described earlier), then with eyes closed while the patient is lying down in relaxed position. The skin incision line (eyelid crease line) is marked along the natural line of the eyelid; positioned at the desired level of the eyelid crease, usually 7–9 mm above the lash line in women

and 5–7 mm above the lash line in men. It is curved superiorly at the lateral ends to provide the pleasing upward slant and to avoid puckering or ‘dog-ear’ formation distally (Figure 68.2a,b). The amount of excess skin to be removed is commonly estimated by pinching with a fine forceps above this incision line at mid-pupil vertical axis and tapered toward both ends (Figure 68.2c). Symmetry of the pretarsal lid segments (lash margin to lid crease distance) is much more important than symmetry of lid crease to brow distances.

One should always keep in mind the position of eyebrows. If a browlift does not accompany the upper eyelid surgery, then more skin and orbicularis muscle should be removed on the side where the brow is lower to compensate for the asymmetry caused by brow location (Figure 68.3).⁵ But the more lid skin excised, except in the elderly, the greater will be the brow drop, and the more lid and brow excision necessary to visualize the juxta-brow covered eyelid and pretarsal lid skin.

Lidocaine (lignocaine) 1% with 1:100 000 adrenaline (epinephrine) is the usual local anaesthetic, injected subcutaneously at the beginning, with additional injection as needed when dissection is carried to deeper layers.

Orbicularis muscle layer

A strip of muscle along the incision can be excised to reinforce the new position of the eyelid crease, or to create a crease for those who do not have it. The sliver of orbicularis muscle excision should occur separate from the skin excision (Figure 68.2d).

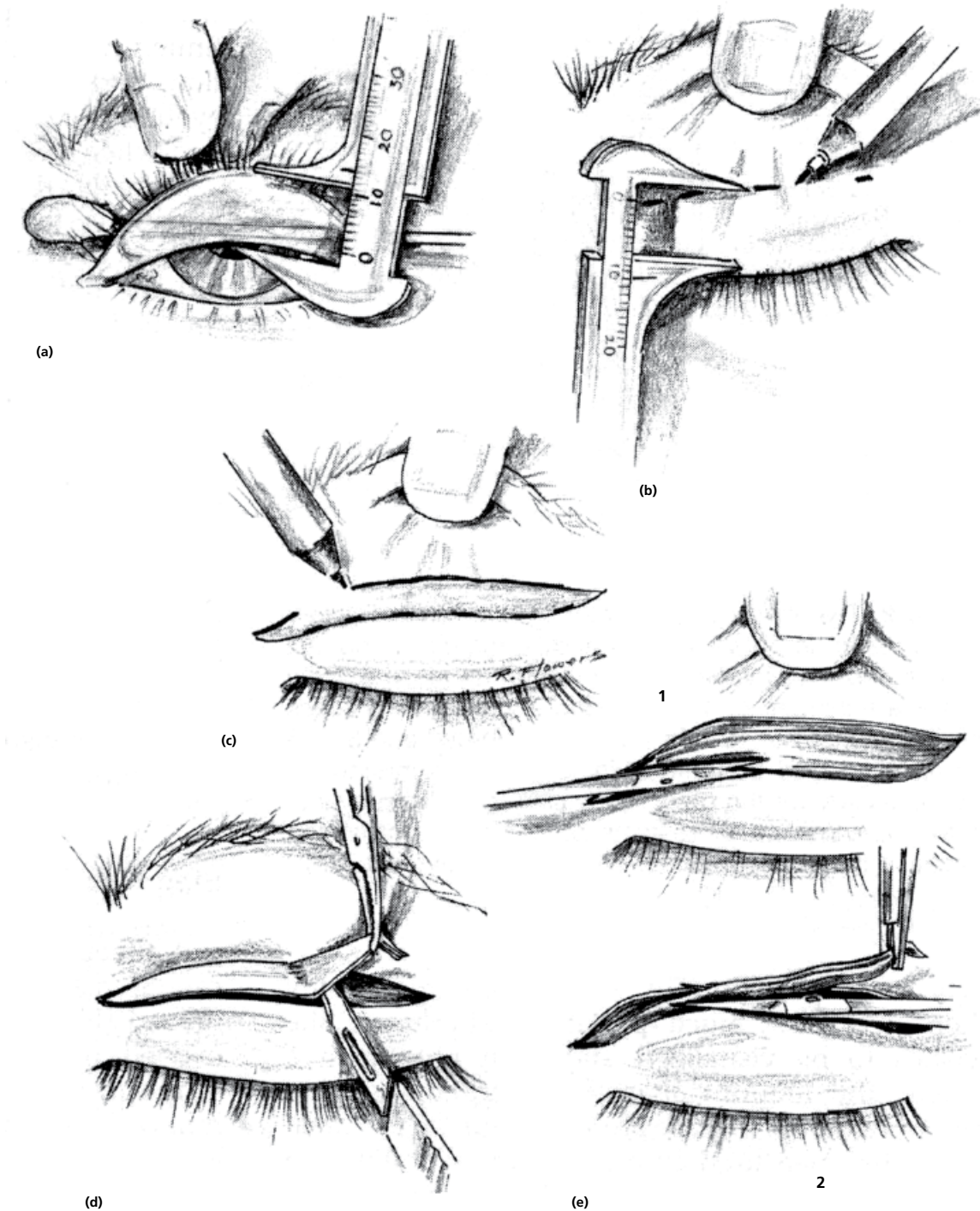


Figure 68.2 (a) At the beginning of procedure, turn the lid over and measure the tarsus. (b) With the skin under uniform tension, mark the lower skin incision with a Jameson caliper. Usually the height of pretarsal skin incision mark is less than the height of the tarsus. (c) Mark the upper skin incision. (d) Skin excision occurs separate from the orbicularis muscle excision. (e) Remove sliver of orbicularis muscle carefully. Never tent the muscle up during excision for fear of inadvertently transecting the aponeurosis.

Make sure you do not injure the aponeurosis when trimming the muscle. One must never tent the muscle up during excision for fear of inadvertently transecting the aponeurosis (Figures 68.2e and 68.4). A larger strip of muscle can also be removed to reduce

any existing looseness or redundancy. This step can be bypassed if there is no need to adjust the eyelid crease or muscle excess, and access to orbital fat is achieved by splitting the muscle layer at respective compartments in that case.

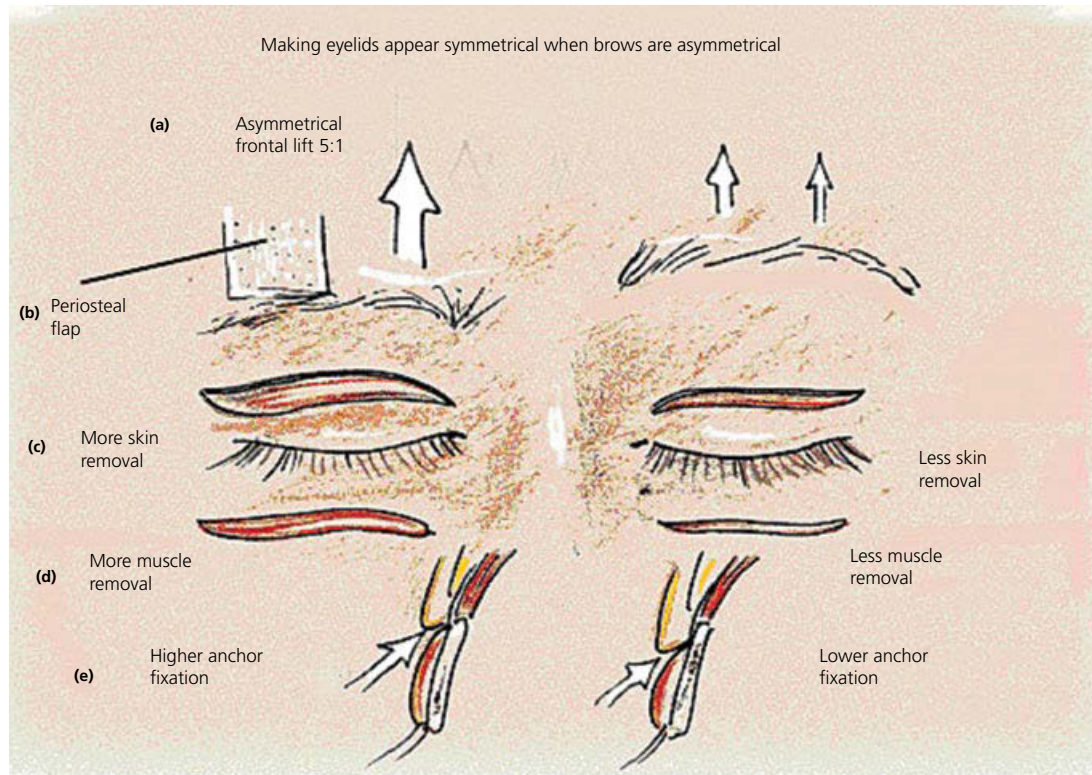


Figure 68.3 Different ways to correct eyelid asymmetry due to asymmetrical brow position. (a) Asymmetrical frontal lift. (b) Insert a superiorly based periosteal flap into the junction of the middle and lateral thirds of the brow which is lower. (c) Excise more lid skin on the side of the lower brow. (d) Excise more muscle on the side of the lower brow. (e) Make the pretarsal skin segment taller on the side of the lower brow, and anchor it at the higher level into the tarsus or the aponeurosis or both.

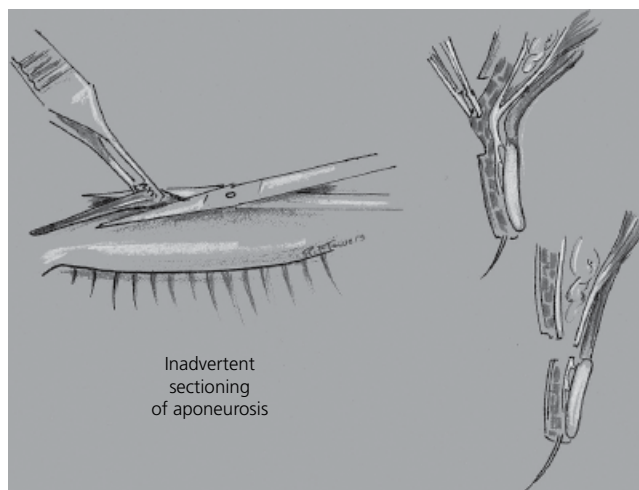


Figure 68.4 Inadvertent injury to levator aponeurosis during orbicularis muscle excision, when the muscle is mistakenly tented during excision.

Preseptal and orbital fat layers

Orbital fat is approached through openings on the orbital septum, although care should always be taken to open the septum in a way that preserves maximum length of the aponeurosis (Figure 68.5), or at least of the septo-aponeurotic extension

thereof, to assure a proper connection without creating lid retraction. The orbital septum should be entered laterally to prevent damage to the aponeurosis, which may cause further lid ptosis. The septo-aponeurotic sling that houses the orbital fat descends lower laterally, and migrates cephalad nasally. If this direction of sling is not appreciated, an attempt to open the septum across the lid is likely to sever the aponeurosis instead. Once the orbital septum is open across the lid, then the fat excision is quite simple. Be conservative! The baggy and puffy appearance of upper eyelid can be softened with conservative removal of excess preseptal and orbital fat. (Figure 68.6).

Lacrimal gland

The lateral bulging of upper eyelid can be a result of a sagging lacrimal gland. The orbital lobe of the lacrimal gland can be sutured to the periosteum lining the inner side of the superior orbital rim to correct this appearance.

Levator muscle and orbital septum

Preoperative diagnosis of lid ptosis is essential. Ptosis caused by attenuation or laxity of levator muscle can be corrected with imbrication of the muscle to the superior edge of tarsal plate, using interrupted absorbable suture such as 6-0 Vicryl or PDS. This manoeuvre also brings the orbital septum–levator muscle

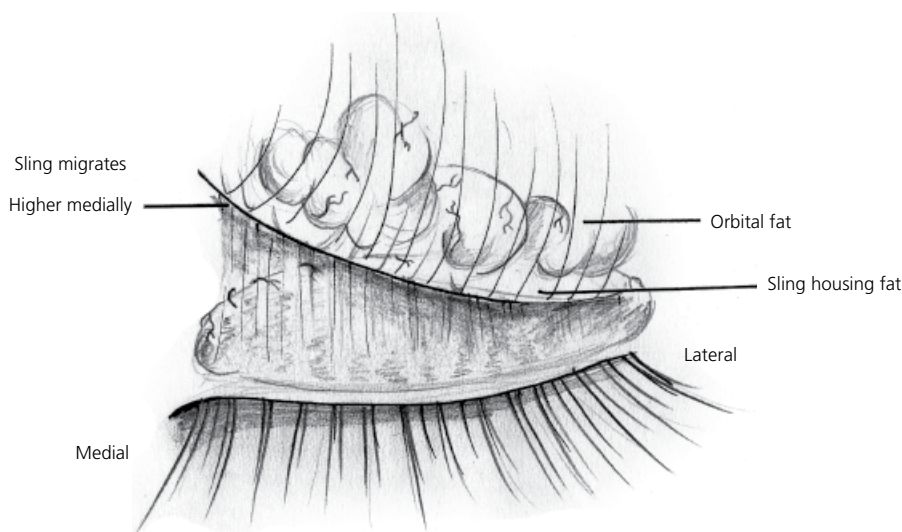


Figure 68.5 The septo-aponeurosis sling housing the orbital fat descends lower laterally and migrates cephalad nasally. If this direction of sling is not appreciated, an attempt to open the septum across the lid may sever the aponeurosis instead.

fusion line forward, softening the hollowness of the upper eyelid. In case of severe upper lid hollowness, tighten the suspensory ligament of the globe with the same orbital rim suture used in the canthopexy, commonly described in the senior author's publications and many lectures and courses. This is the best treatment when the thickening on the orbital septum between medial and middle compartments is weakened by ageing or previous surgery. Tightening the orbital septum with the same canthopexy suture gives additional tightness to the lower lid, which lifts the globe and tightens the lower lid and orbital contents significantly, pushing on and lifting the upper lid fat so it fills out the hollowing of the upper lid that frequently accompanies ageing, especially when accompanied with weight loss.

Closure of incision

The incision is closed simply with a running suture. Another advantage of simple running closure is the ability to recreate or further define the lid crease by incorporating the edges of the aponeurotic tabs into the skin closure across the lid (Figure 68.7).

Examples

Examples of upper blepharoplasties and coronal browlifts are shown in Figures 68.8 and 68.9.

Postoperative care

Patients should be advised to sleep at 45° head elevation post operatively. This diminishes the swelling, which can be significant for the first three weeks. To minimize the early excessive swelling, compressive bandage can be used overnight for periorbital protection, although this will blindfold the patient, and may not be well tolerated by some. It does, however, speed up the recovery and minimize haematoma. Cold compressions should be used for the first few days after the

bandages are removed to further control the swelling. They need not be used for longer than 2 days. Usually the swelling has diminished by around two weeks, however rarely it may last longer than several weeks.

Patients should avoid any vigorous activity for several weeks, with no sudden increase in heart rate and blood pressure, thus minimizing the risk of postoperative bleeding.

Pain is usually minimal, and well tolerated with paracetamol.

The sutures are usually removed within 5–7 days, and the patient should avoid rubbing the eyes, so the fine sutures are not loosened or broken within the early stage.

During the first week or two, ophthalmic ointment should be used, particularly at night, since most patients are unable to close the lid fully (postoperative temporary lagophthalmus) due to early tightness on the lid as well as swelling.

Complications

Complications of eyelid surgery are usually due to bleeding, transient disturbances in tear mechanism and excess skin excision. Good compressive overnight bandaging diminishes bruising, swelling and damage, as does avoidance of rubbing of the eyes.

Haematoma

Superficial haematoma usually occurs when a patient experiences a surge in blood pressure, such as during vomiting, coughing, straining, pain or anxiety. Some over-the-counter medications, such as aspirin or ibuprofen, taken inadvertently, could contribute to this complication. Haematoma that appear to extend posterior to the orbital septum must be addressed urgently, with exploration under good light for irrigation and haemostasis. Ice compresses, head elevation and pain medication are some routine measures that might prevent this kind of complication.

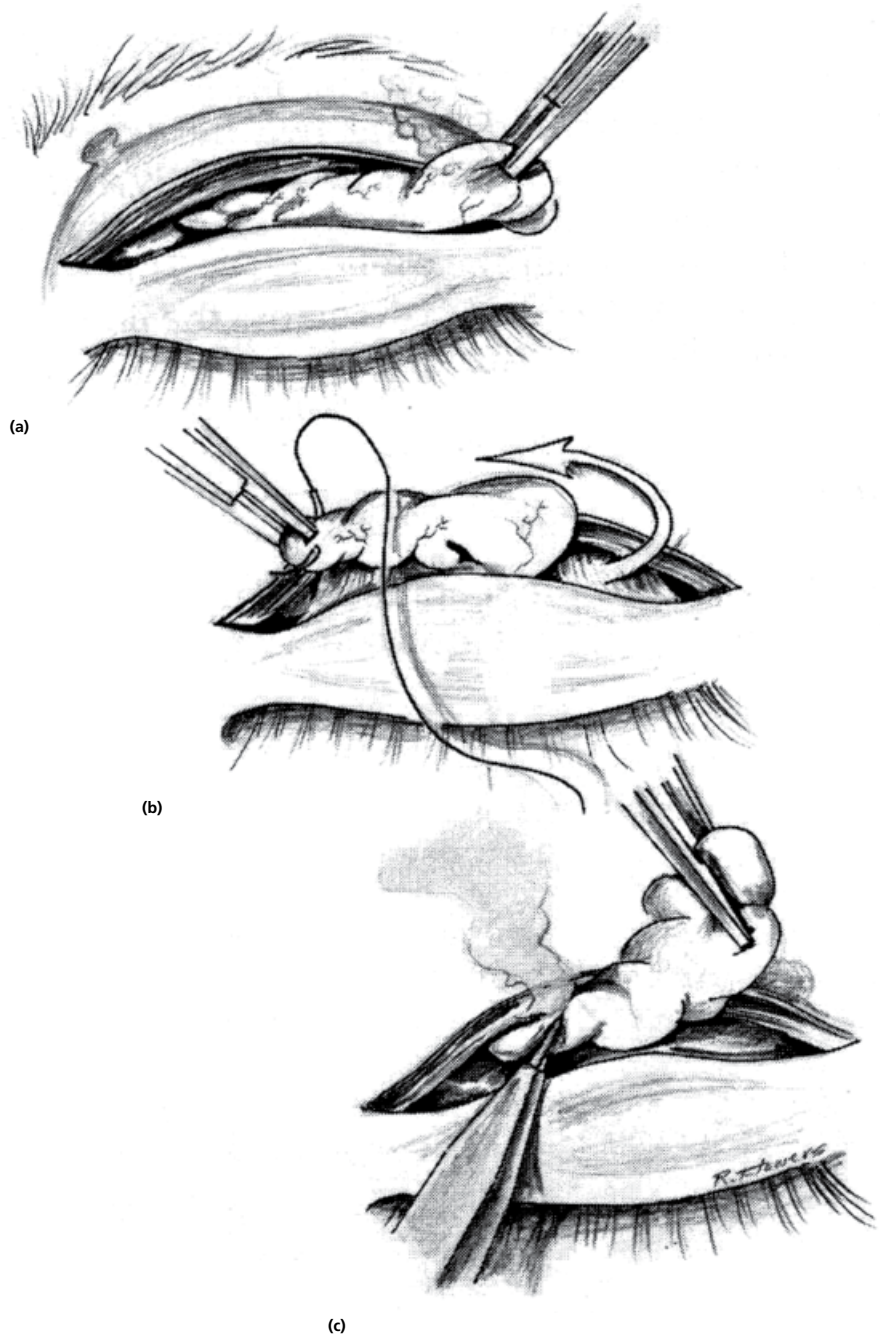


Figure 68.6 (a) Fat excision is quite simple with the orbital septum open across the lid. (b) There is often a prominent tuft of lateral orbital fat tucked behind the orbital rim, next to the lacrimal gland. This fat may be excised, or it may be transposed into the hollow defect. (c) Careful haemostasis is performed with bipolar cautery.

Subconjunctival haemorrhage

This is a collection of blood under the conjunctiva; it is usually absorbed in 10–14 days. Irritation and tearing can result from this condition. Ice saline compresses, artificial tears or ocular lubricant can keep the patient comfortable until resolution.

Conjunctivitis

Inflammation of the conjunctiva can be from sutures, blood clots, pieces of tissue, corneal abrasion or infection. A severe reaction to light suggests corneal abrasion, and examination with

fluorescein should be done. Obvious pus should be cultured. Artificial tears and ocular lubricant are used for inflammation without infection. Antibiotic drops such as Sulfamyl or antibiotic ointment such as bacitracin can be used for infection. An eye patch provides relief for corneal abrasion or severe irritation.

Abnormal tearing

The tearing mechanism may need some time to return to normal functioning after surgery. During the first couple weeks post-op, patient may experience excessive tearing (epiphora),

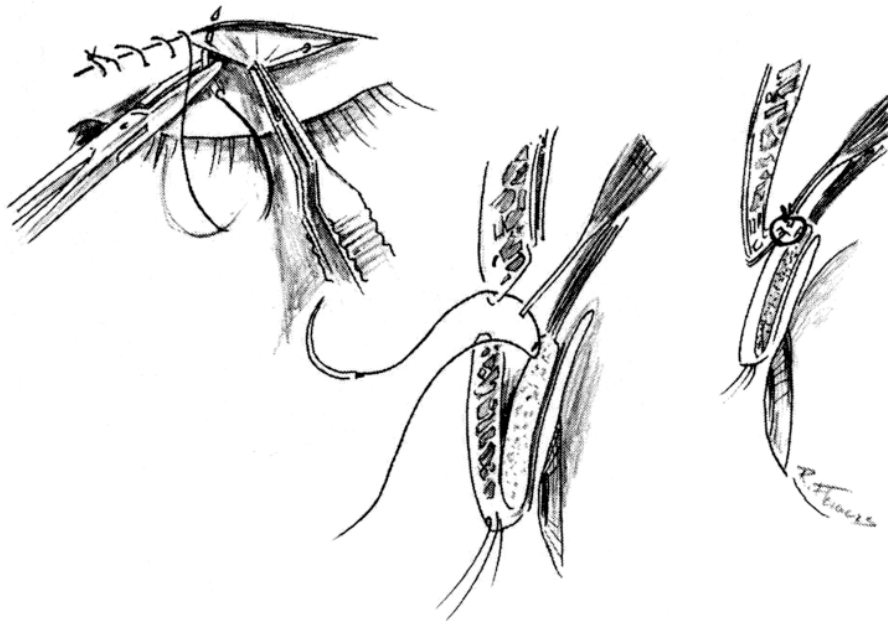


Figure 68.7 Skin closure and recreating or further defining the lid crease by incorporating the edge of the aponeurosis into the skin during closure.



Figure 68.8 Isolated upper blepharoplasty. Before and after pictures. (Performed by RSF.)

dry eye syndrome and visual blurring. Symptomatic treatments with artificial tears, lubricant and patching usually resolve the condition over time.

Lagophthalmos and ectropion

Lagophthalmos is a condition where an eyelid cannot close completely. If this occurs post operatively, it may mean that too much skin has been removed. Often patients can tolerate 1–2 mm lagophthalmus, but 2–3 mm can lead to desiccation of the ocular surface. Factors causing lagophthalmos are excess skin excision, weakness of orbicularis muscle, paralysis of the temporal branch of the facial nerve, tethering of the eyelid to deeper structures or bad scar formation. Initial treatments include the usual supportive measures of artificial tears, ocular lubricant and eye patch; and stretching eyelid massage.

Ectropion is the turning outward of the eyelid, exposing palpebral conjunctiva. This condition is the result of excessive removal of skin, especially in patient with pre-existing laxity of eyelid. Treatments are similar to those for lagophthalmos.

Profound drop of brows and accentuation of corrugator frown

This commonly occurs after upper blepharoplasty when compensated brow ptosis is not recognized before surgery, and simultaneous or planned browlift is not incorporated into the plan.

Asymmetry

Failure to recognize brow ptosis preoperatively, failure to accurately measure and mark the skin for the lid incision and skin excision, failure to recognize and compensate for asymmetrical eyelid ptosis are all causes of postoperative eyelid asymmetry. The most important manoeuvre to minimize this happening is a superb preoperative exam, and careful marking and planning.

Lid ptosis

Varying degrees of lid ptosis are common in the early postoperative period due to swelling, usually subsiding without intervention. The commonest cause of persisting lid ptosis after



Figure 68.9 Upper blepharoplasty and coronal browlift. Before and after pictures. (Performed by AC.)

surgery is undiagnosed preoperative ptosis, which is often exacerbated in the early postoperative period by stiffness and increased weight of lid oedema. If the preoperative ptosis is not well documented and pointed out to the patient, the postoperative ptosis will inevitably be blamed on the surgeon. Another common cause of ptosis is iatrogenic injury to levator muscle or aponeurosis, commonly caused by careless lifting of the tissue while excising a strip of orbicularis muscle. Bleeding to Müller muscle may also cause prolonged ptosis.

Conclusion

Upper blepharoplasty can be a splendid operation when done by skilled surgeons on the right selection of patients, under the correct conditions. The operation usually occurs under local anaesthesia, assisted by light oral or intravenous sedation.

Good outcomes soar after upper blepharoplasties, especially when combined with skilled browlifts, and even more so when globe-lifting lower lid-tightening procedures are added.

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CHAPTER 69

Lower eyelid–cheek junction rejuvenation

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Introduction

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Background

The appearance of tired eyes, saggy skin or bulging lower lid fat are the most commonly sighted reasons for seeking remedy from a plastic surgeon. Traditionally, lower lid ageing is considered in isolation by textbooks and surgeons but is a more complex phenomenon. Anatomical nuances and support structures of the lower lid profoundly influence what we perceive as blemishes of age. Moreover, ageing of the surrounding terrain profoundly influences the lower lid appearance. A complete lower lid assessment should not be carried out in isolation but at the very least should include the midface.

As operative techniques have evolved over the years, our anatomical knowledge and understanding of the ageing process has grown. Arguably, many traditional operative techniques are not a restoration of anatomy to its youthful appearance, but camouflage techniques addressing part of outward appearance. This chapter provides a basic background to lower lid blepharoplasty, before following the evolution of thought and techniques of Dr Hamra over the last 40 years, allowing the reader to benefit from his enormous experience, innovations and philosophical approach.

Anatomy

The lower eyelid and the cheek must be considered together in aesthetic surgery. At the level of the inferior orbital rim the lower eyelids transition to cheek. The lower lid crease, extending 3–4 mm inferior to the lid margin medially, extends to 5–6 mm below the lid margin laterally. This corresponds to the fibrous connections of the pretarsal orbicularis oculi to the skin. The papebral aperture is approximately 30 mm wide and 15 mm high. The lateral canthus is approximately 2 mm superior to the medial canthus. Normally, in neutral gaze there is no scleral show, with the eyelid touching the lower limbus. The thin lower eyelid skin covers the orbicularis oculi muscle, which is divided into pretarsal, prepetal and orbital components. The lower

orbicularis oculi muscle covers the origins of nose and lip elevators. At the medial canthus a deep slip inserts onto the posterior lacrimal crest, a tripartition of the canthus inserts anteriorly, superiorly and inferiorly. The lateral canthal tendon is made up of four components:

- lateral horn of levator palpabrae superioris;
- Lockwood's ligament;
- check rein ligaments of the lateral rectus muscle;
- preseptal and pretarsal orbicularis oculi muscle

The lateral raphe is a complex structure, which consists of a thickening of the pretarsal orbicularis oculi, lying superficial to the lateral canthal tendon, medial to the orbital rim. The lower eyelid is divided into three components: the anterior lamella formed by skin and orbicularis muscle, the middle lamella by the septum and the tarsus, and the inner lamella formed by the conjunctival lining. Beneath the orbicularis oculi muscle and superficial to the septum lies the suborbicularis oculi fat (SOOF) (Figure 69.1). The septum extends from the inferior border of the tarsal plate to a periosteal thickening at the inferior orbital rim, the acrus marginalis. The tarsal plate is 4–5 mm wide and the capsulopalpebral fascia, the lower lid retractor, originating from the inferior oblique, inserts onto its lower border. Orbital fat is divided into three compartments: medial, central and lateral. The inferior oblique muscle separates the medial and central compartments (Figure 69.2). Orbital fat is physiologically different to the rest of the body in that it:

- has smaller cells,
- has less lipoprotein lipase,
- is less metabolically active and therefore unaffected by diet, and
- is more saturated.

An arcade formed by the inferior medial and lateral palpebral arteries supplies the lower lid. Aesthetically, the cheek supports the lower lid, with the orbimalar ligament serving as a retaining structure preventing descent. The nasojugal groove or tear trough extends from the medial canthus to mid-pupillary level. Lateral extension of this groove as the palpabromalar groove forms the lid–cheek junction.² More recent anatomical studies have elucidated an osteocutaneous ligament, the tear trough

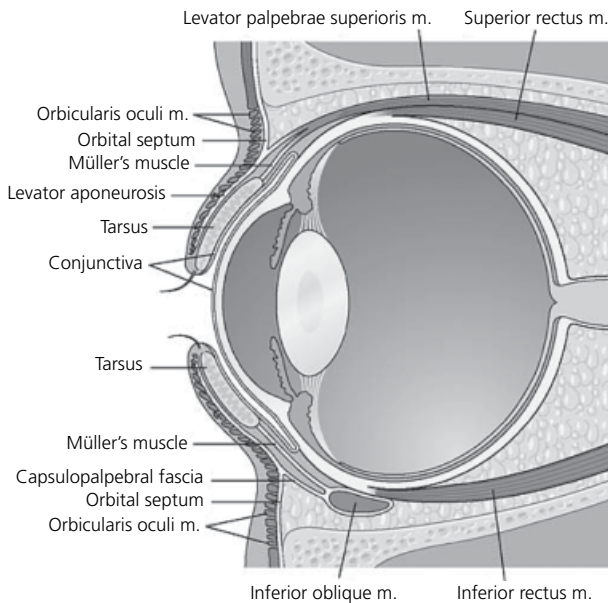


Figure 69.1 Cross section of the globe and eyelids showing the schematic of anatomical structures. Source: Lee & Thomas, 2005.¹ Reproduced with permission of Elsevier.

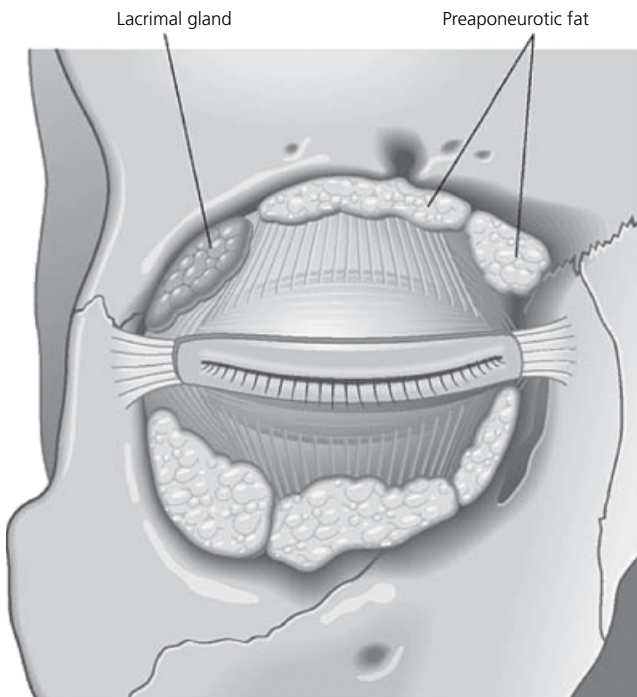


Figure 69.2 Post-septal fat compartments of the upper and lower lids. The lower lid has three compartments, the medial, lateral and central. Source: Lee & Thomas, 2005.¹ Reproduced with permission of Elsevier.

ligament, extending from the maxilla through the septal and orbital orbicularis oculi components.³ More details about the process of lower lid ageing can be found in earlier sections of this compendium and more specialized texts.

Examination

After a thorough medical and ocular history, a focused physical examination is undertaken. A general observation of the face, its subunits, presence of scars, asymmetry, globe position and general skin condition from multiple views is performed. Medications including antiplatelet and blood thinners are noted.

In neutral gaze there should be no scleral show. Lid retraction and snap test are used to assess lower lid laxity. A superomedial and superolateral gaze cause protrusion of the medial, central and lateral fat pads respectively. Gentle palpation of the globe with the eyes closed also enhances their appearance. Where the globe is more anterior to the most anterior part of the zygoma, a negative vector position is noted. The normal intercanthal tilt is 10–15°, with the lateral canthus lying more superior. Both a negative vector and/or canthal tilt have unfavourable aesthetic implications for the lower eyelid.

Operative approach

Although the lower eyelid should be considered as part of a larger lid–cheek subunit, traditional division in approach persists. Here we will discuss the basics of isolated lower lid blepharoplasty as a background, to Dr Hamra's more comprehensive approach to lower lid rejuvenation. Operative surgery can be thought of as addressing the three major components in the lower eyelid in different measures:

- skin only or skin/muscle,
- orbital septum, and
- orbital fat.

These are undertaken through either transcutaneous or transconjunctival approaches.

Where a transcutaneous approach is taken, a standard subciliary incision is made, elevating the marked redundant skin or a skin and muscle flap. A preseptal approach is taken to the level of the infraorbital rim. Where the septum is not addressed, stab incisions and gentle pressure on the globe allow excision of the fat compartments before closure of the cutaneous incision. Resection of the fat compartments however, leaves the eye with a hollow and ultimately aged appearance. Moreover, it does not adequately address the tear trough deformity. Although use of soft tissue fillers to address this deformity has become common place it was Loeb who first suggested release of the medial fat compartment, its transposition and fixation against the more medially placed nasal and lip elevator muscles to address the nasojugal groove.⁴ Septal release at the level of arcus marginalis allowing free draping of the fat compartments over the orbital rim followed by the septal reset procedure was conceived by Dr Hamra and is discussed in more detail in the next section.^{5,6}

Where a transconjunctival approach is taken, the skin is either in very good condition with no redundancy or is expected to be amenable to surface laser rejuvenation. In this approach an incision is made below the tarsal and either a direct approach through the capsulopalpebral fascia or a

preseptal approach to the fat compartments is taken.⁷ More recently the nuanced nature of septal support has been appreciated. Plication of the most inferior part of the septum, thought to have become redundant with age and contributing to appearance of fat herniation, is undertaken through this approach. The proponents of this technique cite preservation of the anterior lamellar and subsequently reduced risk of scleral show as a major advantage.⁸

There are many other permutations and combinations of the surgical approach to lower eyelid rejuvenation. The interested reader is referred to more specialized texts in this field.

Complications

The most catastrophic complication is a retrobulbar haematoma related to fat excision, which may cause blindness. Fortunately, the rates of this complication are estimated around 0.05%. The surgeon must be vigilant for its signs and expeditious in its treatment. Disproportionate pain, proptosis, lid ecchymosis, dilated pupils, decreased vision and reduced extraocular movements are signs of an evolving retrobulbar haematoma. If untreated, central retinal artery occlusion and subsequent blindness will ensue. This is considered irreversible after 100 min. Its treatment is a matter of surgical emergency, the patient's head is elevated, surgical incisions are released, mannitol (12.5 g IV bolus over 3–5 min), acetazolamide (carbonic anhydrase inhibitor 500 mg IV bolus), topical beta blockers, large dose of steroids (Solu-Medrol 100 mg IV) are administered, surgical cantholysis and re-exploration of the surgical field is undertaken urgently.

Corneal injuries, dry eyes, scleral show, ectropion and lid position asymmetry are among other complications. Where an early postoperative scleral show and evolving ectropion is noted during follow-up, increased massage and consideration of administration of steroid or 5-fluorouracil could be given to reduce this risk. Persistent scleral show and established ectropion will ultimately have to be thought of as cicatrization or shortness in one of the eyelid components, anterior or middle lamella. In extreme circumstances revisional surgery must be undertaken to address the tissue deficit. Dr Hamra's philosophy and evolution of personal techniques over the last few decades follows and entails many lessons in approaching lower lid rejuvenation.

Lower lid rejuvenation: evolution of approach and technique

Sam T. Hamra

Lower eyelid rejuvenation has been a part of facial surgery since the early part of the twentieth century. As early as 1928, removal of the lower eyelid fat was a consistent manoeuvre in most eyelid surgery in an attempt to remove the stigmata of fatigue and age.

My experience with lower blepharoplasty began in 1970 with my residency training. Initially a skin flap was elevated, followed

by three stab wounds to retrieve the three pockets of fat always described under the lower eyelid orbicularis. About that time, British surgeons began elevating a skin muscle flap, which seemed to be an advance by producing less ecchymosis, although many surgeons at that time were still excising a pre-marked pattern of skin without muscle by doing a 'skin pinch' manoeuvre with small forceps. Even without undermining, this was a satisfactory approach, as the width of the pattern was rarely 2–3 mm so that primary closure could be done without muscle excision. As a resident, I was not in a position to follow these patients any length of time, therefore I have no idea how many eventually developed some degree of lower lid malposition. When I entered private practice in 1973, my procedure choice was a skin muscle flap, with fat removal when indicated, which of course was very often, in my opinion.

I continued this approach throughout the 1970s and 1980s in spite of the fact that frequently neither the patient nor I were happy with the results. In general, all facelifts of that era were skin lifts with superficial musculoaponeurotic system (SMAS)⁹ elevation. I was fortunate to have learned the Skoog rhytidectomy several years earlier. Forehead lifts were not commonly done. In the majority of blepharoplasties that I performed, results were acceptable, at least by the criteria known to me at that time. There were occasional problems with scleral show, which would become manifest often after several months, as oedema from both the facelift and eyelid procedure subsided. It was during this period also that the transconjunctival lower eyelid fat technique became popular. I used it frequently for young patients, depending on any loose skin to shrink as it did with liposuction and rhinoplasty. As with standard blepharoplasty results, I still felt that the lower eyelid anatomy and contour were not in many cases sufficiently rejuvenated.

From this point going forward my experience and philosophy of orbital rejuvenation became intimately integrated with facial rejuvenation. Although this chapter emphasizes blepharoplasty procedures, my experience, as opposed to many surgeons around the world, must be described in terms of the impact of facelift operations on the orbital zone. In order to fully describe my personal steps on the way to the technique that I utilize today for the lower eyelid I must give the reader a background of my facelift experience, and how these milestones in my personal journey impacted the results I can achieve today.

In 1985 I began exploring the possibility of improving the nasolabial fold, which at the time seemed the most unimproved with facelift surgery. I discovered that the fat of the cheek overlying the zygomaticus major and minor muscles could be elevated with the skin. Since 1973 I had routinely performed a Skoog rhytidectomy.^{10,11} When adding the cheek fat to the Skoog flap that contains the platysma muscle of the lower face, my facelift results seemed much better. I called this technique a 'deep plane rhytidectomy', as published in 1990.¹² The results of this technique caused me to look more carefully at the lower eyelid. Moving the fat of the cheek, or malar fat as some surgeons labelled it, in a superior lateral vector seemed to impact

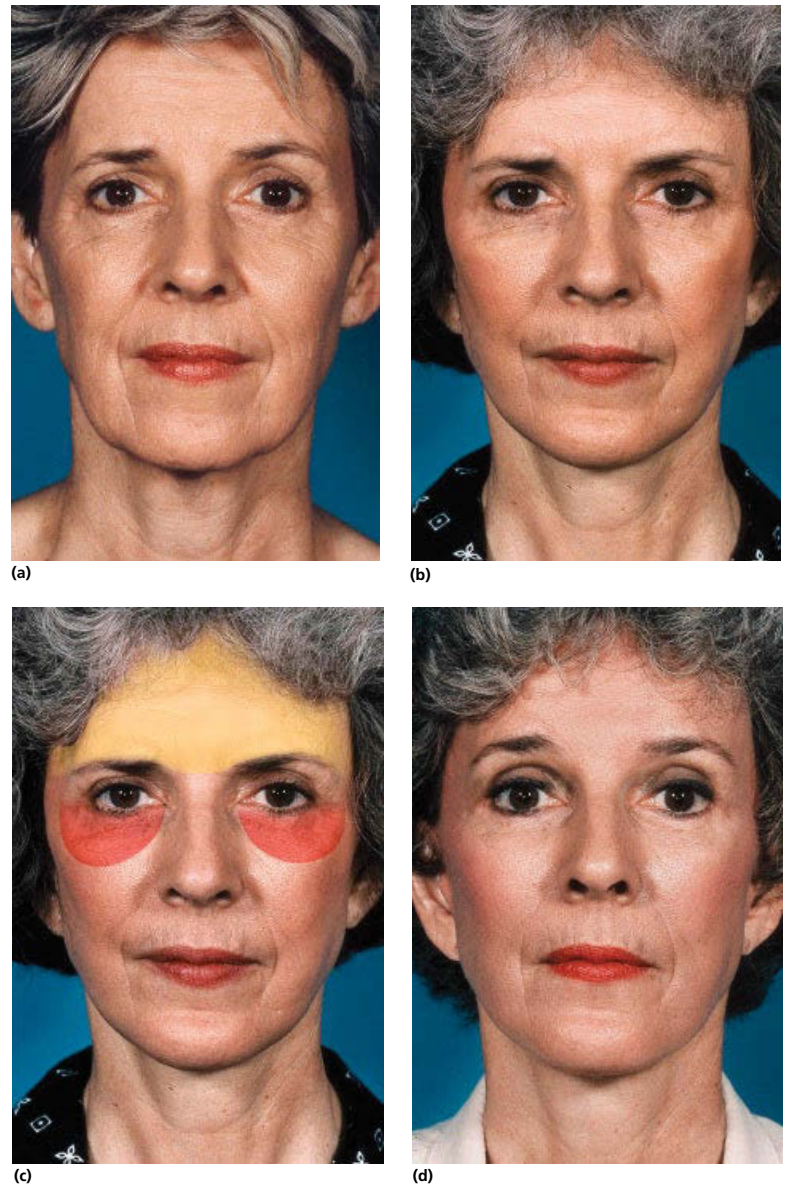


Figure 69.3 (a) Postoperative standard blepharoplasty with orbital fat removal. (b) Following a deep plane rhytidectomy. (c) This patient then underwent an isolated orbicularis repositioning with a coronal brow lift. (d) The brow lift and orbicularis repositioning then gave the patient a harmonious facial rejuvenation.

the orbicularis oculi, which at the time was not elevated. This is to say that the juxtaposition of the elevated malar fat next to the unelevated orbicularis created an even deeper appearing lower eyelid following blepharoplasty than I had seen before. This appearance was made more severe with the removal of lower eyelid fat. This was the first time that I realized the balance between the lower eyelid and cheek anatomy historically following cosmetic surgery was suboptimal, since frequently the patients appeared less attractive and less youthful than they had before surgery was done.

It was in 1990 that I decided to reposition the lower orbicularis oculi along with an isolated forehead lift. The patient in Figure 69.3 was the first to undergo this combination. I had done a conventional upper and lower blepharoplasty on her in 1984, with what I thought to be an acceptable result

(Figure 69.3a). In 1989, however, I performed a deep plane face-lift (Figure 69.3b). As described in the deep plane article, the cheek fat had been moved in a superior lateral vector. Rather than creating an attractive person, I had in fact disrupted the harmony of the ageing face with the combination of procedures that I thought were innovative and acceptable. Faced with a dilemma of what to do, but having no previous experience that would direct my next move, I decided to return to the operating room to perform the isolated orbicularis repositioning with a coronal forehead (Figures 69.3c,d). I then witnessed for the first time in my career what I thought to be the closest I had seen to a harmonious facial rejuvenation. It was for me a 'Eureka' moment.

I then decided that if I had in fact included the cheek fat with the platysma muscle in the facelift flap, perhaps I could simply

add the orbicularis muscle to the same flap. Searching for a new description of this technique, I decided on the term 'composite rhytidectomy'^{13,14} since the orbicularis, cheek fat and platysma muscle maintain their intimate relationship with each other in the flap, very much like the 'composite' grafts from the ear that contain skin, fat and cartilage, which have been used for many years for nasal reconstruction. The word 'composite' is defined in the dictionary as 'made of many parts', which reaffirmed my feeling that this was an excellent name for a surgical procedure, rather than the commonly used acronyms in plastic surgery that lend themselves to marketing for the lay public. Unfortunately, because of my earlier use of 'deep plane', some confusion naturally arose. However today, as then, a composite rhytidectomy absolutely must include the elevation and repositioning of the orbicularis oculi muscle of the lower eyelid.¹⁵

A deep plane or high SMAS procedure does not impact the lower eyelid at all in spite of claims of vertical movement. This composite facelift technique was presented at the American Society for Aesthetic Plastic Surgery annual meeting in New York City in 1991. Part of the explanation of the advantage of the composite technique was demonstrated with the patient in

Figure 69.4, who achieved a very obvious shortening of the height of the lower eyelid, with the pre- and postoperative results marked to demonstrate this result. To my knowledge this was the first presentation that clearly demonstrated the shortening of the height of the lower eyelid.

Unfortunately, the concept was so new that few understood what I was trying to accomplish. Mainline plastic surgery thinking, which included the surgeons who had been doing SMAS techniques, allowed little room for what appeared to be an approach that was too new for many to accept. For many, it appeared to be a technique needing a generous amount of time for the steep learning curve.

From 1990 to 1992 as I performed the orbicularis repositioning with a composite facelift, I continued to remove lower eyelid fat, as tradition dictated. In spite of what I thought was a marked improvement in the harmony of the face, I still noticed a certain hollowness of the lower eyelids (Figure 69.5) when comparing my results to daughters of my patients who bore a strong resemblance to their mothers. In fact I published a series of mothers and daughters in my 1993 book entitled *Composite Rhytidectomy* in an attempt to draw attention to my target of a youthful eyelid-cheek junction. I received one day



(a)



(b)



(c)



(d)

Figure 69.4 In 1990 this patient underwent a composite facelift with orbicularis repositioning, plus removal of lower eyelid fat. This was the first demonstration of the shortening of the lower eyelid, which was presented in 1991.

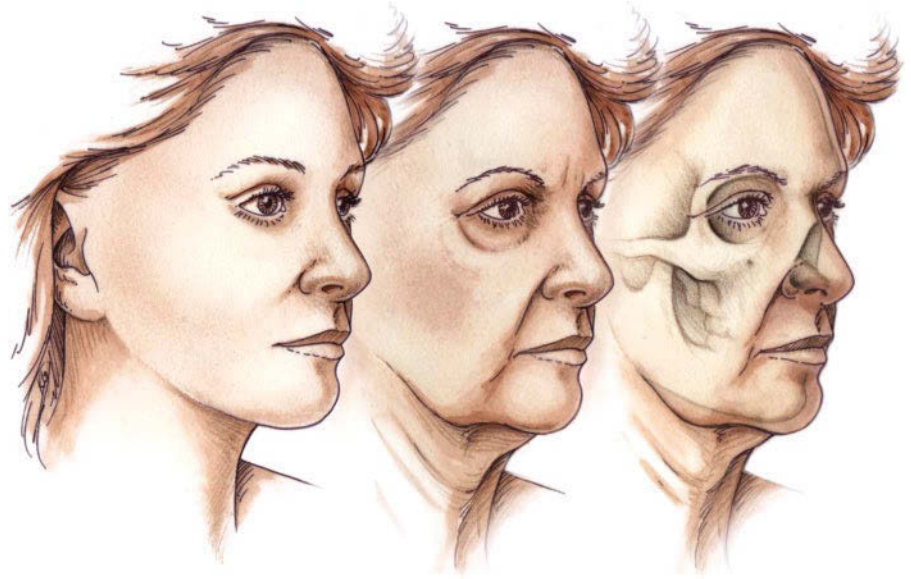


Figure 69.5 In the latter half of the third decade, the soft tissue covering of the bony framework of the human face loses tone exposing the underlying anatomy. It is at this point that a 'eye socket' seems to become manifest.

in the mail a list of new plastic surgery books. One was a book on eyelid surgery from Raul Loeb, a Brazilian surgeon. He described a 'fat sliding procedure' in which he took the medial fat pocket of the lower eyelid and put it under the orbicularis medial attachment to improve the 'nasal-jugal groove'.¹⁶ However, he continued to remove the middle and lateral fat pockets and did not reposition the orbicularis muscle. The problem I saw with my results was a generalized hollowness of the complete lower eyelid. Since I had the orbicularis elevated, I then attempted to use all the fat of the lower eyelids and to suture it over the orbital rim under the repositioned orbicularis oculi muscle. Because I had been trained that the septum orbitale should not be manipulated, else an ectropion may occur, I felt it best to excise the septum. I published this paper in 1995 and called the procedure an 'arcus marginalis release' with preservation of all of the lower eyelid fat.^{17,18} Up to that point I had never heard the term *arcus marginalis* used when surgeons described periorbital rejuvenation, and I had never seen an operation where this manoeuvre was utilized. However, when I used the bovie to free the lower eyelid fat at the orbital rim, I was in fact releasing the anatomical barrier that was keeping the fat within the orbit. In this paper I attempted to describe the advantage of not removing the lower eyelid fat, which was commonly done throughout the world since it was described in 1928. By comparing the youthful contour of an eyelid–cheek junction to the ageing contour, it appeared to me that removal of the lower eyelid fat quickly created an even deeper and more hollow lower eyelid that was neither attractive nor youthful. I attempted to demonstrate in this paper that preservation of the lower eyelid fat with transposition of all of the lower fat over the orbital rim and superior vector repositioning of the orbicularis oculi muscle would prevent the hollow lower eyelid syndrome (Figures 69.6 and 69.7).

Although early results were impressive, I was still not satisfied with the tightness and smoothness of the lower eyelid. There are two separate issues I wanted to improve. The first was the fact that regardless of how aggressively I repositioned the orbicularis muscle, the cheek tissue seemed not to reflect the same degree of superior repositioning that I could demonstrate with my fingers before surgery. I did have some experience with subperiosteal cheek dissections, but I felt that the thick cheek flap did not have the elasticity when the periosteum was lifted with the flap. It also seemed that the intermalar distance changed, since the origin of the zygomaticus muscles was elevated in the flap and repositioned.^{19,20} My goal was to preserve the location and original attachment of this muscle origin, but at the same time to move the cheek fat and the orbicularis muscle in a superior medial vector. I discovered I could elevate the soft tissue over the periosteum and extend this elevation both lateral and medial to the origin of the zygomaticus major and minor muscles. To clearly distinguish this flap from the subperiosteal dissections that had become^{21,22} popular due to endoscopic approaches that were being taught around the world, I called it a zygomaticus orbicularis flap (Figure 69.8), and abbreviated the name to the 'zygorbicular' cheek lift. It was very strong and allowed me to effectively reposition the cheek fat that was elevated in the deep plane approach. Moreover I could actually move the cheek fat in a superior medial vector. I became immediately impressed with the very natural youthful appearance of the tissue that was repositioned over the malar eminence.

At the same time, in an attempt to create the youthful 'positive vector' of the lower eyelids, I decided to take a chance and include the septum when I moved the fat of the lower eyelid. I incised the arcus marginalis as before, which was the lower extent of the septum. I then reset this border including lower eyelid fat to the pre-periosteal soft tissue inferior to the orbital rim, and named this procedure the septal reset (Figure 69.9). In this way, the undersurface of the lower eyelid orbicularis was now resting on a

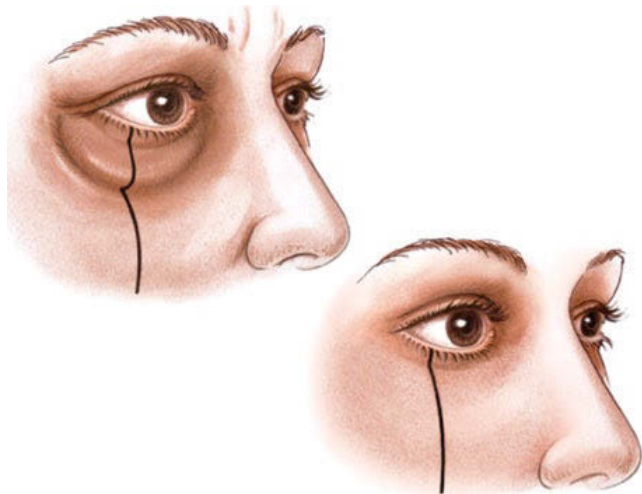


Figure 69.6 The ageing eye has a double convexity, and the youthful eye has a single convexity. The youthful convexity with no evidence of an eyelid-cheek junction is a goal of periorbital rejuvenation.

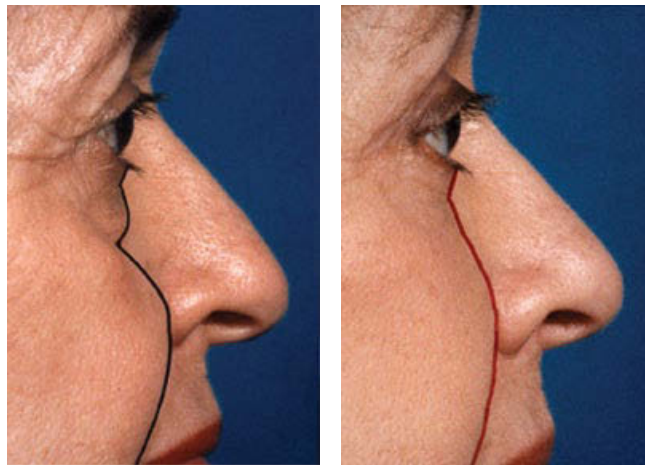


Figure 69.7 This is an example of a patient following an arcus marginalis release with preservation of the lower eyelid fat. The single convexity has been reestablished.

firm foundation rather than on the softness of only the repositioned lower eyelid fat when the septum was not included. This firm lower eyelid, coupled with a very strong purchase of the cheek tissue to the orbital rim with sutures, completed the mission to create an optimal rejuvenated eyelid-cheek junction.

The septal reset²² had been included in the 1998 article on the zygorbicular dissection but it appeared to be to be buried in the content concerning the flap. Therefore it was published again in 2004 along with an article in the same journal by Barton²³ reaffirming the advantages of the septal reset.

Indications of the composite cheek lift

Primary patients

Essentially every patient I see who is seeking facial rejuvenation, with rare exception, is a candidate for a primary 'composite cheek lift' with or without a facelift. A composite cheek lift consists of a

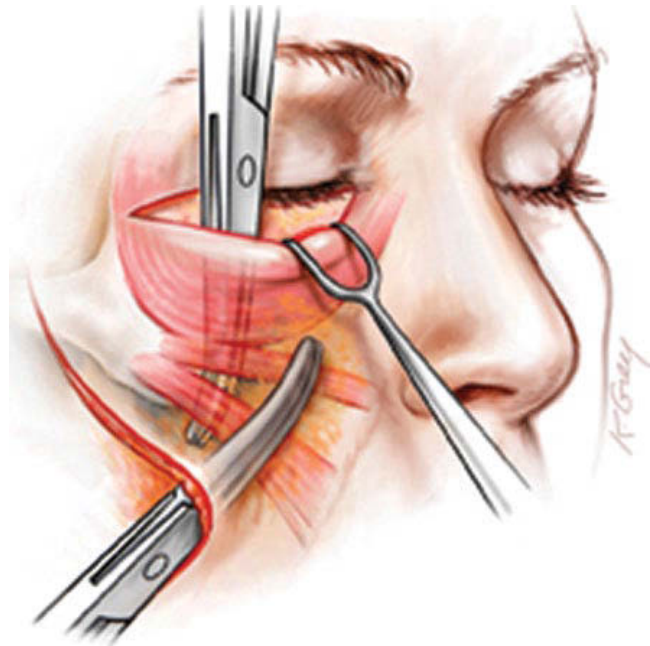


Figure 69.8 Through the lower blepharoplasty incision, the orbicularis-zygomatic complex could be undermined medial and lateral to the origin of the zygomaticus without disrupting the origin as one must do in a subperiosteal dissection.

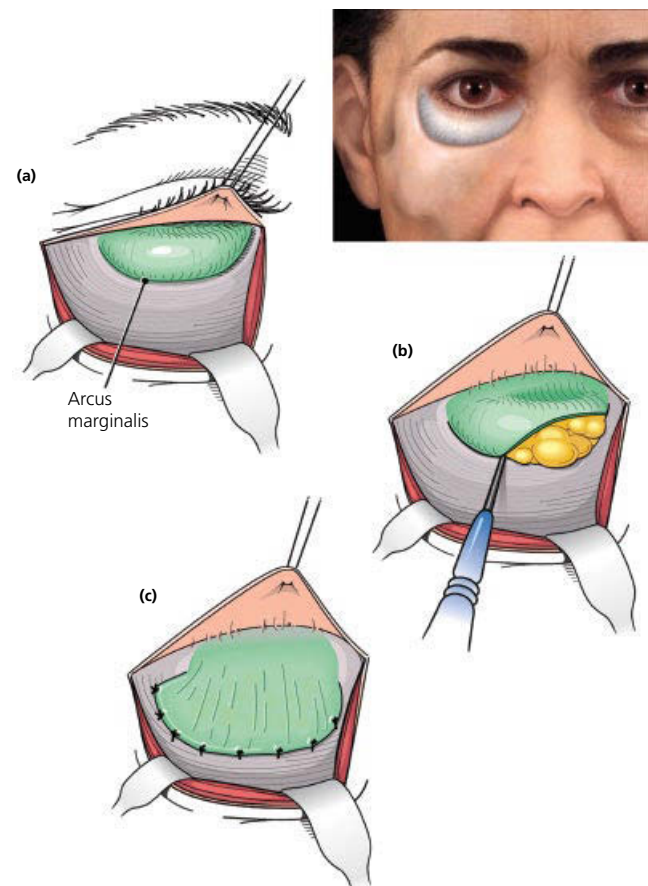


Figure 69.9 After excising the arcus marginalis, the septum could be left intact and then reset over the orbital rim.

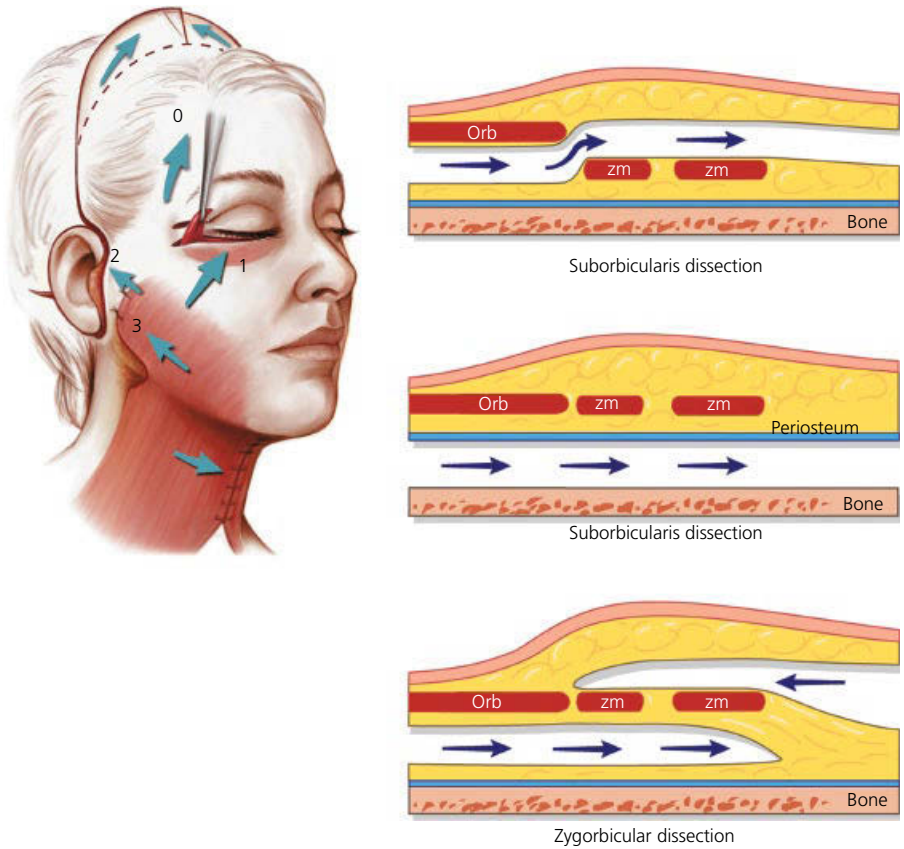


Figure 69.10 In order to shorten the lower eyelid there are only three possibilities.



Figure 69.11 This 50-year-old had only a composite cheek lift and septal reset.

septal reset and a superior medial vector of the zygomaticus orbicularis manoeuvre. In order to effectively impact the anatomy of the lower eyelid the surgeon has three choices: reposition the orbicularis oculi muscle only; reposition the cheek with a subperiosteal

dissection; or use the zygomaticus orbicularis procedure. I find this last option the most advantageous (Figure 69.10).

Obviously, the normal ageing of the human face progresses from the absence of any landmark of underlying bony anatomy



Figure 69.12 This 48-year-old underwent an upper blepharoplasty and composite cheek lift.

to a point, usually beginning in the late thirties, where a definite 'eye socket' with an eyelid–cheek defining border begins to take shape. The bony anatomy of the facial skeleton changes little, if any, between the second and fourth decades of life. It is the ageing of the soft tissues of the face that changes the surface topography of the face. This proceeds throughout life (see Figure 69.5). The ageing of the lower eyelid skin, while variable with different skin types, loses tone and turgor. The amount of lower eyelid fat is also a variable, and usually genetic in distribution and position. This is naturally true throughout the human species, as DNA no doubt determines the appearance of the face humans inherit. In youth, the septum orbitale confines the fat to the infraorbital space. With the loss of tone of the septum orbitale, the lower eyelid fat, whether normal in volume or excessive, is no longer confined to the original space but manifests itself with 'bags' at the eyelid–cheek junction. It is this appearance that commonly directs the patient to the plastic surgeon.

This ageing becomes manifest in the late thirties and early forties and, coupled with the ageing of the skin of the lower eyelid and the obvious prolapse of the underlying fat of the lower eyelid, clearly differentiates the patient from the youthful-appearing anatomy they enjoyed perhaps 10 years earlier. In these years, I commonly perform an upper blepharoplasty and composite cheek lift, since in most cases the remainder of the facial anatomy shows little, if any, advanced ageing. The patient in Figure 69.11 had essentially no facial ageing except the natural hollowness of the eyelid–cheek area. She had an isolated zygorbicular cheek lift with an arcus release and septal reset. This is an excellent example of the youthful appearance that can be obtained with this technique. A patient of the same age with excess lower eyelid fat will obtain the same endpoint with the same procedure. (Figure 69.12).

By the late forties and early fifties, however, with ageing of the lower face and neck coupled with forehead ptosis, the patient is

a candidate for a comprehensive facial rejuvenation. In my practice, if in fact there is sufficient ageing of the overall facial contour, I usually perform a composite facelift, which includes rejuvenation of the forehead, cheek and face, and the neck.

The criteria for impressive postoperative results following facial rejuvenation has changed over the last 40 years. Initially one was happy with a more youthful jawline. Then in the late 1970s, the emphasis was on the neck contours. The mid-1980s seemed to concentrate on ways to improve the nasolabial folds. In my experience, impressive periorbital rejuvenation began in the early 1990s, with much attention directed at the eyelid–cheek junction. My criteria have now changed, with the emphasis and endpoint being a higher cheek mass that had not been previously created (Figure 69.13a,b,c).

In my 2004 article on the septal reset,²⁴ and after many years in practice, I was able to demonstrate many patients under my care when they were very young who, in time, returned for facial rejuvenation. For every type of primary and secondary anatomy, their eyelid–cheek junction became similar to their youthful contour, and in some cases even better. However, in the past 7 years, by omitting an SMAS elevation during the facelift dissection, I discovered that I could even more effectively reposition the upper cheek tissues. Omitting the SMAS elevation now allows me to create a higher cheek mass, which I believe is the endpoint for youth and beauty (Figures 69.14 and 69.15).

Even patients with an appearance of the malar crescent, or malar bags, or festoons can be optimally corrected, since the deformity is created by excessive and redundant orbicularis musculature that can be totally changed with zygorbicular repositioning (Figures 69.16 and 69.17).

Secondary patients

Conventional facelift and eyelid techniques often result in an appearance that is neither youthful nor attractive. In 1995, while I was developing the zygorbicular dissection with the septal reset,



Figure 69.13 (a) This patient underwent a composite facelift, including the septal reset and the zygomaticus orbicularis elevation. The hairline was lowered. (b) The cheek fat repositioning is the key to a higher cheek mass without need for fat injections or alloplastic implants. (c) The shortened vertical height of the lower eyelid and the absence of a discernible eyelid–cheek junction is the endpoint of lower eyelid–cheek rejuvenation.



Figure 69.14 In a comprehensive facial procedure, one must be prepared to surgically change every part of facial anatomy.



Figure 69.15 The three-quarters view can definitively show most parts of the surgical rejuvenation. The best scientific assay of an effective surgical procedure is to compare the same side before-and-after surgery with a half-and-half rather than just viewing side-by-side views.



Figure 69.16 Even with overt festoons that are inherited, a composite rhytidectomy can effectively reposition the orbicularis muscle that creates the 'malar crescent' deformity.



Figure 69.17 This is a primary patient with 'malar bags' preoperatively.

Figure 69.18 This is the classic appearance of the patient who had a previous facelift and standard blepharoplasty, with resulting 'hollow eyes and a lateral sweep'. Both have been effectively corrected.



Figure 69.19 This patient had a previous standard blepharoplasty and rhytidectomy, with a subsequent lateral sweep and malar crescent deformities. In addition to the facelift and eyelid correction, she had a secondary rhinoplasty.



I was very pleased to discover that when patients came for a secondary correction of their previous facelift that they felt was suboptimal, I was able to create an exciting rejuvenation by reversing the stigmata of prior surgery. This was done by elevating essentially all tissues of the face except the upper eyelids, nasal area and perioral area and then repositioning these anatomical segments in accordance with the principle of composite facelifting.

After analysing numbers of these patients, it became clear to me that there were two distinct areas that created the 'facelifted' appearance. The first was the hollow lower eyelid that resulted from previous fat removal. The second was a 'pulled' lower face that seemed swept to the side. I then published in 1998 a sister paper to the paper on the zygorbicular dissection, entitled 'Frequent facelift sequelae: hollow eyes and the lateral sweep'.^{25,26} To my knowledge it was the first paper published analysing the

cause of the surgically created faces seen everywhere. As one would imagine, it was not well received by plastic surgery colleagues as it seemed an indictment of techniques that were time-honoured and easy to accomplish. In almost every case, an impressive improvement can be demonstrated (Figures 69.18 and 69.19).

I had previously described the hollow eyes following traditional blepharoplasty techniques in 1995 with the paper on the arcus marginalis release. Even though I could obtain satisfactory results in secondary patients at that time, it was the advent of the septal reset that was the key that allowed me to effectively correct almost any patient with hollow eyes. As I looked back on almost every patient I had done between 1973 and 1991, I routinely in most cases removed lower eyelid fat. Wherever I went socially, I was fixed on the lower eyelids when I observed people known to have had facial rejuvenation surgery. It was the same

with patients I saw in my office, whether I had done them or they were done by other surgeons. No matter how attractive the facelift results, the lower eyelids never seemed as rejuvenated as other parts of the face.

The second part of the stigmata of a 'facelifted' appearance was what I named the 'lateral sweep' of the face. The effective lateral repositioning of lower face anatomy, frequently made more dramatic with an SMAS manoeuvre, is in my opinion the aetiology of the lateral sweep. The appearance of the lower part of the face, particularly in patients with lighter dryer skin and obvious rhytids, is a deformity obvious to any observer; in the lay press it is commonly called the 'wind tunnel look'. Since the tissues were essentially swept in a lateral vector to the ear, a lateral sweep sounded like an appropriate description. To my knowledge, the lateral sweep and the hollow lower eyelids had never been described before in the plastic surgery literature, except for the article on the arcus marginalis release in 1995. Moreover I am sure that the standard SMAS facelift technique had never been indicted as the primary aetiology of the 'facelifted' appearance since it had become the accepted standard of facelifting in 1976. With the exception of the 'mask lift' created by Paul Tessier, the vast majority of surgeons all over the world were disciples of either a subcutaneous facelift or an SMAS manoeuvre incorporated into a subcutaneous lift.

The mask lift is a subperiosteal dissection that gained popularity particularly with craniofacial surgeons. There is frequently a widened intramalar distance due to the fact that the origin of the zygomaticus major and minor is displaced in a superior lateral vector. The blepharoplasty component remained traditional with either a transconjunctival fat removal or a conventional skin muscle flap. In order to create a higher cheek mass, Tessier advocated the placement of bone grafts on the zygomaticus eminence. The placement of malar silicone implants had been done for many years with conventional facelift procedures. With removal of lower eyelid fat causing a depression which adjoined the increased height of the malar eminence appearance, the eyes seemed more hollow. In addition, the hard tissue augmentation with bone or alloplastic materials frequently created an angular and unattractive 'bony' appearance. Surgeons recognized the high cheek mass as seen in young people, so the popularity of malar augmentation became widespread. Since it is a very simple procedure to accomplish, and since it did create a change that the patient could observe, it remains a very popular and marketable technique. Like so many procedures in aesthetic surgery, 'since so many surgeons are doing it, it must be okay'. This is a phenomenon seen repeatedly with endoscopic facelifts, CO₂ laser, so-called liquid facelifts and, of course, the string lifts.

When I had patients who had these unfortunate facial changes, I was pleased to find that these deformities were reversible when I applied the principles of the composite facelift with the septal reset. Removal of the malar implants is almost always obligatory. Because they are normally placed in a subperiosteal level one



Figure 69.20 This patient had a conventional facelift and blepharoplasty with malar silicone implants. Postoperative results after removal of implants and composite rhytidectomy. Note neck changes, and correction of preauricular incisions and earlobe deformity.

must be careful when removing them so that a level of preperiosteal fat still exists over the orbital rim in order to do the septal reset. The patient in Figure 69.20, in addition to the placement of malar implants, had hollow lower eyes. The preauricular incision was poorly placed away from the tragus, and neck contouring was incomplete.

Conclusion

In order to completely reverse the hollow eye and lateral sweep, one must perform a comprehensive rejuvenation procedure that almost always includes attention to the neck, the lower face, the cheek, and forehead lift. The sequence is as follows: initially the neck is dissected in a preplatysma plane. Excess fat, if any, is removed with scissor dissection. A deep plane dissection is done on the face without elevating the SMAS. Since 2005 I have observed that the tissues of the face can be moved effectively in a superior medial vector without elevating the SMAS, making the traditional lateral vector movement of the SMAS unnecessary. When employing Feldman's corset procedure on the



Figure 69.21 A 13-year follow-up on a primary facelift patient. Note the continuation of the high cheek mass.

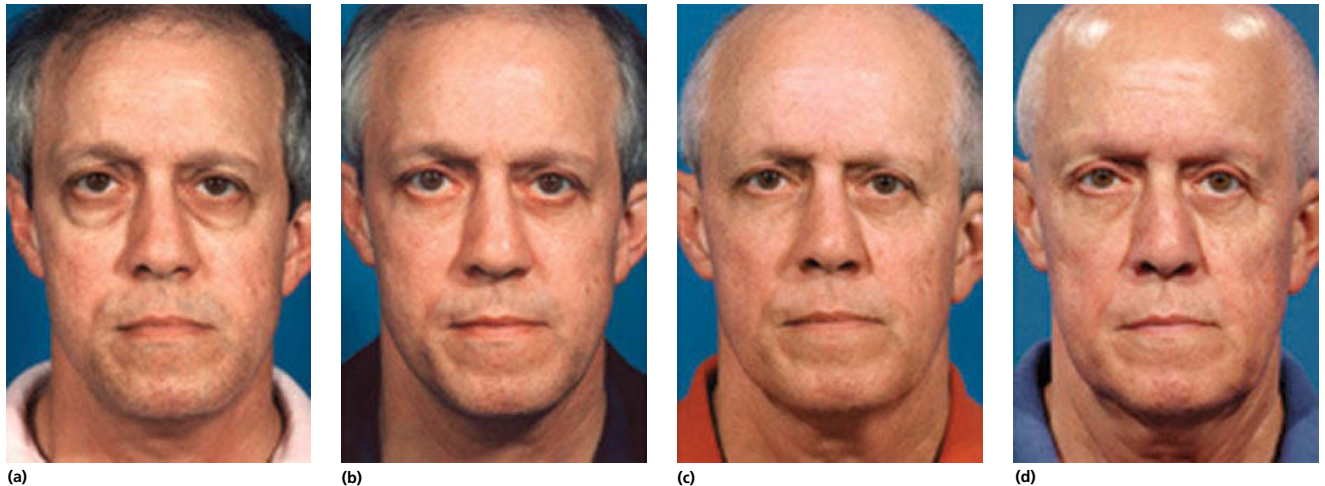


Figure 69.22 This is the first patient who had a septal reset and cheek lift procedure. He is now 17 years postoperatively. All orbital fat was preserved. (a) Preop, (b) one year post, (c) 10 years post, and (d) 17 years post.

platysma anterior edges,²⁷ it makes no sense to counter that very strong platysma tightening in the neck with tension on the platysma of the lower face toward the ear. The dissection on the prezygomaticus fat is exactly as it was described in 1990. One must visualize the zygomaticus major muscle in order to be sure that all of the fat of the cheek remains adherent to the skin. This is absolutely necessary since the zygomaticus orbicularis flap developed from the lower blepharoplasty incision is now joined with the deep plane flap. In this way the cheek fat can be repositioned in a superior medial vector very high.

The lower blepharoplasty incision, which is the gateway to the zygomaticus orbicularis flap, can be done as an isolated cheek lift procedure or is done with a facelift after the deep plane dissection from the pretragal incision. With the septal reset one can be sure that the cheek fat will remain high and

the lower eyelid height will be significantly shortened as a lower eyelid positive vector is created regardless of the original anatomy.

For standard subcutaneous lifts or various conventional SMAS lifts, the surgeon can elect either not to do a forehead lift, or do a lateral vector open forehead lift or an endoscopic forehead lift. In contrast, if one does a composite facelift and cheek lift then the forehead lift becomes obligatory to prevent ‘bunching of the temporal skin’ and the vector must be a superior medial vector for the forehead lift.

The disadvantage of this technique is that many patients for many reasons do not choose a comprehensive procedure based on cost, recovery time and the ability of the surgeon they choose to do a rejuvenating technique. On the other hand, the advantage of this technique is a uniquely balanced and harmonious appearance



Figure 69.23 This patient had a primary composite facelift when she was 70 years old. At 84 she has maintained a high cheek mass with a youthful eyelid–cheek junction. (a) Preop, (b) one year post, and (c) 14 years post.

that not only will never have the stigmata of cosmetic surgery, but is very stable, as long-term follow-ups will demonstrate. Having done the composite facelift with the zygomaticus orbicularis dissection with the septal reset since 1997, I am now in a better position to evaluate long-term results. The most stable part of this procedure is the eyelid–cheek rejuvenation. (Figures 69.21, 69.22, and 69.23). The upper blepharoplasty and obligatory open forehead lift are equally long lasting, but it is the obvious permanent change of the lower eyelid that will continue to give the patient a very youthful appearance, which is the goal and endpoint of periorbital rejuvenation.

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CHAPTER 70

Facelift

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Introduction

Trainees in plastic surgery appreciate early on that one of the defining and most satisfying features of the specialty is the logical biological approach to solving clearly defined clinical problems. There tends to be broad agreement on the principles, objectives and outcomes, as well as the appropriateness of the surgery, across surgeons of different generations and from different 'schools', which unites plastic surgeons while allowing for differences.

However, on exposure to facelift surgery, it is as if an entirely different world has been entered. The trainee is confronted by a situation in which consultant plastic surgeons of similar stature and ability seem to interpret clinical situations differently and perform different facelift procedures based on a different logic. There is often a defensive awkwardness in addressing questions on this. When the patient's immediate result 'on the table' looks good after different facelift techniques, how can trainees decide what approach to take when they come to perform facelift surgery?

Facelift theory

The facelift world is indeed different, because of the many dimensions it embraces. Foremost it is about appearance, which is central to a person's identity. A person's identification of self, their psychological wellbeing and, indeed, their life, revolves around their face. The face is the most defining feature of a person's appearance, which is why there are national portrait galleries filled with images of faces. A person's self-image and self-esteem are powerfully influenced by their appearance. The way people are judged and treated by others, whether supportive or discriminatory, is largely determined by others' perceptions of their character and personality, initially based fallaciously on their facial appearance. People requesting a facelift have experience of this. This aspect places a significant responsibility for the patient's future psychological wellbeing in the hands of the

facelift surgeon; it is an added dimension that trainees are usually not prepared for but must quickly appreciate.

The human face is uniquely adapted for the higher social functions of communication, including expression of emotion. It is therefore inherently difficult to alter a face surgically while retaining the nuances of natural expression, especially as some elements of expression are so subtle as to be scarcely detectable to the naked eye, appreciated only at a subconscious level by the recipient. A compromise in the patient's ability to communicate and to be responded to naturally affects the patient's future wellbeing.

The anatomical demands for facial movement in expression are complex. While the anatomy of the hand is recognized by plastic surgeons as being complex, most of the detail was defined during the classical era of anatomy. Yet after nearly 100 years of facelifting, only recently has a different level of facial anatomy been gradually revealed and this has happened during the lifetime of the current generation of surgeons. For these surgeons, trained in traditional, essentially empiric facelift techniques, this is a significant change. Acceptance and application of the rational 'new anatomy' based techniques tends to be largely in proportion to the surgeon's certainty of anatomical understanding. Fortunately, the detail of this new anatomy is almost complete, and will provide the next generation of plastic surgeons with a better understanding of the pathophysiology of ageing and its logical application in rejuvenation surgery.

Good surgeons are risk averse and appreciate that complex anatomy involves greater surgical risk. This is especially important where complications lead to visible alterations of appearance, resulting in psychological ramifications. The surgeon's choice is stark: to prioritize safety or to prioritize exceptional results! The gap between these two options is reduced by the surgeon's level of anatomical understanding and facelifting experience.

What constitutes an exceptional result cannot be defined absolutely. There is not necessarily agreement about this between the patient and the surgeon, between patients and, indeed, between surgeons themselves. An undetectable facelift is, at the least, a desired part of the result, meaning that the

patient's appearance has improved in a way that is not visibly the result of having had a facelift. A detectable facelift, in contrast, can vary in significance between stigma and status, according to the prevailing cultural and social standards in the particular society. As these standards vary between societies, contrast London with Los Angeles for example, so the definition of a good facelift result may vary according to geographical location.

Facelifts do not fit into the previously mentioned category of 'clearly defined clinical problems', because the end point, namely the patient's satisfaction, has a subjective element. Unconscious factors in a patient's background, whose influence is commonly not appreciated or expressed, have an influence on the patient's motivation and expectations of a facelift, and so on his or her perception of the result. Despite good-quality preoperative consultations and planning, this subjectivity can lead to disappointment, to the chagrin of the surgeon.

For the surgeon to obtain good results in aesthetic surgery requires more than the usual surgical attributes of anatomical understanding and technical skill, with an appropriate measure of psychological patient management. Considerations of aesthetics in appearance are paramount; while facelift surgery is inherently a technical specialty, the importance of the surgeon's aesthetic eye is often unappreciated, being an intangible, yet critical factor in the outcome. Many facelift patients will have a more advanced visual aesthetic than the young technical surgeon who does not intuitively have, or has not yet developed, this level of aesthetic appreciation. Just as a young musician learns by listening to music, the young plastic surgeon should cultivate the habit of analysing faces. Without an understanding of what looks attractive and why, the practitioner of aesthetic surgery does not have an appreciation of the specific outcome to be aimed for.

In considering rejuvenation procedures and the aesthetics of the face, an appreciation of the gestalt of the face, the patient's face as a whole, is no less important than the assessment of the component parts and the way they relate. A key consideration is whether to correct only the part that most concerns the patient or to address the whole. A piecemeal approach in stages can result in a disharmonious and unnatural outcome in some patients, and yet for others it can be a clever way to surreptitiously introduce an undetectable refinement of appearance.

Finally, we should consider the pervasive influence of the media, and its effect on attitudes and opinions, even before people are old enough to contemplate the effects of ageing for themselves. Informative and responsible in-depth analysis of facelifting is rare in the media, compared to the superficial entertainment stories that sensationalize negative aspects of cosmetic surgery. While the media focus is intended to satisfy the fascination of the public, prospective patients can understandably become confused as they try to define what they want and what they can expect for themselves. A consequence of the World Wide Web becoming the foremost source of patients' information is the heightened expectation of facelifts. The influence of marketing in that medium is diminishing the

perception of the reality that a facelift still carries the limitations and risks of a surgical procedure. Even for people who would not consider this option, this incorrect perspective becomes reinforced when facelifts are now marketed in such an appealing way, as a commodity included in a glamorous and affordable vacation package. It is important for the individual surgeon to recognize this modern day background to presurgical consultations and to realize the difficulty of fully controlling patients' expectations of their surgery.

The ageing face

The face is a complex three-dimensional structure, having areas of intrinsic movement and varied foundations of solid bone and supporting soft tissue volumes held together by visible and invisible ligamentous extensions upon which the canvas of our life, emotions and expressions is spread. Genetics, time, environment, lifestyle and, ultimately, gravity all take their toll on our appearance. This canvas bears the blemishes of ageing from inside and out. A tired look, wrinkles, sagging with changed shape of the face, jowls and loose neck are the exacting toll of life's vectors on the face.

The original surgical techniques from the early 1900s were limited to the skin envelope, and because such work had not been thought possible before this time, the improvements to a person's ageing look were considered to be remarkable. To be tackling facial appearance was bold surgery, even if addressing laxity of the facial skin was not. The procedures evolved gradually over the years with progressively more undermining and redraping of the facial skin. The risks were few, mainly to do with bleeding and skin circulation. With patient expectations being relatively low, surgeons were not pushed to risky surgery and the progress in technique was slow.

Then in the 1960s deeper contouring surgery commenced, with direct submental lipectomy introduced by Millard¹ and with profound results in the era before liposuction. The modern facelift era started in the early 1970s with Skoog's revolutionary 'anatomical' approach.^{2,3} Then, Mitz and Peyronie's seminal 1976 paper on the anatomical details of the superficial fascia introduced the important concept of the superficial muscular aponeurotic system (SMAS).⁴ Skoog developed the first sub-SMAS dissection plane facelift. The next milestone, over a full decade after the SMAS description, explained many of the frustrating limitations of SMAS surgery and a way to overcome them. First it was shown that for proper SMAS mobilization of the anterior face, it is necessary to detach the SMAS from fixed attachments to the zygoma.⁵ During the 1980s Furnas discovered that these attachments are actually retaining ligaments and that there is a system of named retaining ligaments that attach the facial soft tissues to the skeleton.⁶ Even now, 25 years on, the full surgical potential of the facial ligaments is not widely appreciated.⁶⁻¹⁰ Understanding how to use the SMAS and retaining ligaments to best advantage has been ongoing.^{8,11-19}

In recent years attention has turned towards the role of soft tissue atrophy, specifically fat, to add to our increasingly nuanced understanding of facial ageing.^{20,21} The final piece in this jigsaw is a unifying theory, recognizing that ageing is a universal phenomenon, not only of the facial skin envelope and the deeper soft tissues, but also of the underlying foundations, the facial skeleton on which the soft tissues are built.^{22–25} Correct rejuvenation technique must address all of these components, taking into consideration the unique individual and regional variations. This chapter provides an overview of the field while the more specialized facial aesthetic surgery chapters provide operative details of the specific techniques.

Overview of facelift techniques

Facelift surgery is a remarkably diverse specialty. In no other field of surgery is such a wide diversity of techniques being practised for the same operation. It may seem strange that this absence of uniformity is regarded as acceptable in this evidence-based medicine era and for a procedure whose quality of result is of such importance to the patient. The absence of consensus determining which technique the individual surgeon should practise partly reflects the paucity of objective evidence (randomized controlled trials). There is insufficient evidence to resolve the debate on which facelift technique provides the ‘best’ result. This leaves the responsibility for choosing the ‘most appropriate’ technique to the individual surgeon, based on the catalogue of ‘best facelift’ results presented by the leading surgeons at presentations and in textbook chapters. Yet when viewing photographs of good results it is not possible to discern which particular technique had been used. A good result, by definition, is undetectable. This confusing situation points to the importance of the ‘surgeon factor’, in which masterfully performed surgery using a ‘lesser’ technique provides a better result than a theoretically better technique with less well-performed surgery. Using an analogy from competitive equestrian eventing, the winner may not be the best horse or the best rider, but the best combination of horse and rider, with the benefit of training and experience together.

In the world of facelifts, however, there is no absolute correct result. It is not about winning and clarity about the result can be hard to define. Success can be defined by degrees of improvement, or the number of years erased, or by some other individual patient requirement. An excellent result may be high in the broad grey zone between overdone and underdone, well expressed by the term ‘age appropriate’. Providing the patient with the youngest look does not necessarily provide the best result. Reversing a person’s appearance to being exactly what it was in the past results in an incorrect-looking rejuvenation that is clearly the result of surgery.

When comparing facelifts for ‘best result’ it is useful to focus on specific areas of the face – for example the lid–cheek,

nasolabial fold, jawline and neck – as some procedures inherently improve some areas better than others.

The best facelift

The ‘best facelift technique’ is, in theory, the one that provides the outright ‘best facelift result’, if indeed there is one. The variation in facelift techniques being performed reflects a number of important related factors that need to be taken into consideration by the surgeon: the social situation of the patient and their psychological expectations, moderated by the competing consumer demand for short recovery time, predictability with low risk, reasonable cost (meaning shorter surgical and recovery time) and naturalness of appearance of the result. The longevity of the result is another factor; this is usually less of a priority in the patient’s mind when making the decision. Balanced against this are the surgeon factors that determine the selection of which of the multiple tissues layers of the facial structure should be the foundation for a patient’s facelift. In reality, the surgeon chooses the ‘most appropriate facelift technique’ for this matrix of factors.

Although improvement of appearance is common to all facelifts, there is great variation in the degree of improvement of the various regions of the face. Concomitant facial rejuvenation procedures are commonly performed at the same time, such as blepharoplasty, temporal lift or browlift, contributing together to the refreshed appearance. The debate about facelift technique selection has become clouded by the emergence of appropriate hybrid procedures that combine with the lift; either additional volume (using lipofilling or other fillers or skeletal implants or a combination) or additional suspension, tightening/lifting using non-surgical thread/facelift combinations and even major resurfacing.

For the patient (whose first impression when looking at their new postsurgery ‘improved’ face must be one of suppressed horror) there is usually relief within a short time that a ‘tell tale’ overdone result has been avoided. Patients are usually pleased with the improvement, without considering that a greater degree of improvement may have been possible by ‘better’ facelift surgery.

Impact of the new anatomical understanding on facelift practice

Understanding the historical evolution of the facelift operation helps to explain the diversity of facelift techniques being performed. This history largely reflects the steps in the surgical application of improved anatomical understanding. In its early years, the facelift benefit was localized to the lateral cheek, as all corrections were in small separate areas, upper eyelid, lower lid, etc. Hence the early medical terms used for facelifts, such as *meloplasty* (‘shaping of the cheek’) and *rhytidectomy* (‘excision of wrinkles’). This progressed to *cervicofacial rhytidectomy* (‘neck and face’) with extension of the improvement to the neck.

The subsequent trend was to merge the improvement of an area into the adjacent areas. When browlifts were introduced

Figure 70.1 The objective of a modern facelift is to restore youthful shape to the face. (a) A 56-year-old woman with significant cheek laxity. (b) One year following a composite facelift, which emphasizes tightening of SMAS laxity of the anterior face. The patient's look is refreshed and the typical facelift pulled look avoided.



they soon overlapped with the upper lid and the temporal lift benefit improved the upper outer cheek. The midcheek, remained a 'no man's land' between the territories of the lower lid blepharoplasty and facelift until the advent of the 'extended lower lid blepharoplasty' to improve the upper part of the midcheek. The facelift correction eventually extended into the midcheek and from there its impact continued to the lower lid. This has allowed the focus of modern facelifts to be less about skin tightening for the correction of wrinkles and all about restoring the youthful shape of the face, from the lower lid down to the lower neck²⁶ (Figure 70.1).

Subcutaneous facelift

The earliest application of surgery in facial rejuvenation early in the twentieth century²⁷ consisted of the simple excision of carefully located and shaped ellipses or half moons of skin at the back of the face (hairline, pretragal). These were performed under local anaesthesia without undermining and using direct closure with controlled skin tension and often involving multiple sessions. By the late 1920s the benefit of undermining the skin for a short distance was appreciated.²⁸ Interestingly, even with this advance in technique a pattern was emerging, acknowledging the advance as being 'too much for the would-be surgeon'.²⁹ Similarly, poor facelift results were being seen, with an unnatural appearance resulting from tightly stretched skin.³⁰

Modern subcutaneous lifting started being performed in the 1930s as a result of linking the multiple skin excisions with a continuous incision, along with the release of subcutaneous attachments. These tentative first steps in facial aesthetic surgery gained momentum with surgical experience. Increased demand coincided with the increased social affluence of the post World War II years and general medical progress, with the advent of antibiotics and improved anaesthesia. The technique entails dissection of large, random-pattern subcutaneous skin flaps of the cheek and

jowls with a superolateral suspension vector and resection of excess skin.³⁰ To improve the cheek correction, the skin dissection progressively extended towards the nasolabial fold, usually, but not always, stopping a few centimetres short of this landmark. Furnas, in describing the retaining ligaments of the cheek, noted that early surgeons recognized the upper area of dissection over the lower zygoma as being more difficult to dissect and more vascular in the area known as McGregor's patch.⁶ Subsequently, the reason for this was found to be the dermal attachments of the zygomatico-cutaneous ligament, which extends through the underlying SMAS and divides at the subcutaneous level. The extent of neck skin undermining below the mandible has variously extended towards the mentum. After haemostasis, with appropriate tension applied to the skin flaps, the excess skin is marked and key sutures placed before trimming the redundant skin and closure of the incisions, with or without drains. A thin face with little soft tissue ptosis, excess skin and little bony resorption or indeed a revision facelift may be an appropriate case where a subcutaneous technique can still be considered.

There are innumerable variations in the extent of undermining. The dermal attachments across the face are not uniform, but reflect the underlying pattern of facial ligamentous attachment and this is the rational basis for deciding the extent of surgical release.

There are also variations in the depth of the subcutaneous dissection, the decision being determined by the surgical objective. A superficial subcutaneous dissection has the advantage of being the most remote from the facial nerve branches, hence minimizing the risk of facial nerve damage. Where the subcutaneous layer is thin, particularly over the temple and in the elderly, there is less than usual protection of the underlying temporal branches of the facial nerve. In secondary facelifts, the nerves are at greater risk due to their fixation by fibrosis from previous surgical intervention, and especially when this is more than usual as a result of previous haematoma. When

a separate SMAS flap is planned, it is beneficial to leave most of the retinacula cutis (in the subcutaneous layer) attached to the SMAS, as this fibrous component reinforces the strength of the SMAS flap.³¹ When superficial subcutaneous dissection is performed, a meticulous dissection technique is required to avoid damage to the dermal circulation and visible irregularities.

In contrast, a deep level of subcutaneous dissection is required when the surface of the SMAS is to be exposed preliminary to superficial SMAS platysma manipulation techniques. At this deeper level the subcutaneous dissection is considerably easier, with less bleeding, but the dissection plane is closer to the nerves. This level of dissection has been described as the superficial musculo-aponeurotic plane (SMAP).^{32–34} This plane of dissection, directly on the surface of orbicularis oculi overlying the zygoma, when extended medially provides direct access to the malar fat pad, the thicker subcutaneous fat inferomedially that forms the volume of the nasolabial fold. Specific suture suspension of the malar fat pad provides a controlled correction of the nasolabial fold contour, but unfortunately at the expense of prolonged postoperative oedema.⁸

The skin flap technique maintains popularity with surgeons because of its surgical simplicity, safety, short operative time with relatively speedy recovery, and relatively satisfactory early postoperative results. The long-term shortcomings with this technique are inherent, due to the viscoelastic properties of skin. Tightening the skin envelope alone cannot maintain shape. Additional manipulation of the underlying parenchyma is required, as in other plastic surgery procedures, most notably breast reductions. Depending on skin tension has other specific limitations, including scar widening, traction on the pinna and skin necrosis.

SMAS manipulation techniques

Following Skoog's technique and the subsequent anatomical description of Mitz and Peyronie, it became evident that utilization of the underlying SMAS was the key to controlling tension on the skin closure.⁴ This was the first accepted advantage of SMAS surgery but it took many years, with further anatomical studies and improved understanding of the potential of the SMAS, for it to be appreciated as the tool for true facial reshaping and more lasting outcomes.^{8,11,12,14,16,17,35,36}

SMAS plication/imbrication

The original version of this technique was to first perform the standard type of skin-only facelift dissection and then to add mobilization of the SMAS, as a completely separate layer, locating the SMAS incision directly beneath the facelift incision. The SMAS dissection over the preauricular area is difficult due to the absence of a natural cleavage plane, as a result of the strong adherence of the SMAS to the parotid capsule (the fixed SMAS). The early SMAS mobilizations were limited in extent to the anterior border of the parotid, as originally advised by Mitz and Peyronie, to avoid the risk of damage to the facial nerve branches. It was only later appreciated that it is necessary for the

SMAS to be mobilized forward of the parotid capsule (the mobile SMAS) if the medial cheek and jawl is to be tightened (Figure 70.2).

After the SMAS dissection, the subsequent handling of the SMAS could be varied according to individual facial morphology, specifically the SMAS thickness, included plication (simple infolding of the excess SMAS) and imbrication (undermining of the SMAS and subsequent overlapping), tightening perpendicular to the nasolabial fold using buried sutures. After haemostasis, redundant pre- and postauricular skin is marked in a similar superolateral vector, with key sutures placed at the top of the helix, prior to excision.

Variations of this technique continue to have wide popularity. Its advantages include relative safety in relation to the sub-SMAS facial nerve branches, while providing effective manipulation of the mobile SMAS, combined with relatively quick postoperative recovery, yielding a more lasting improvement than skin-only rhytidectomies. Limited release of the SMAS from its retaining ligaments and deep attachments is a limitation of this technique. Superolateral traction on the SMAS without release of its more anterior retaining ligaments (masseteric cutaneous) restricts the tightening of the more anterior laxity towards the nasolabial fold and oral commissure. For this reason correction of the anterior midface has traditionally been limited.

The deep plane and composite rhytidectomy

The role of the SMAS was the major topic of discussion in facelifts through the late 1980s and 1990s. In this context the newly introduced deep plane and then composite facelift techniques introduced by Hamra became hot topics.^{12,37} These operations were an evolution of the original Skoog operation. Skoog did not describe his operation as being sub-SMAS as it preceded the introduction of the term. He described the plane of dissection used in his revolutionary facelift in correct anatomical terms as being under the superficial fascia.^{2,3} For surgeons of the time, for whom considerations of anatomy were not usual in the context of facelifts, this concept was not

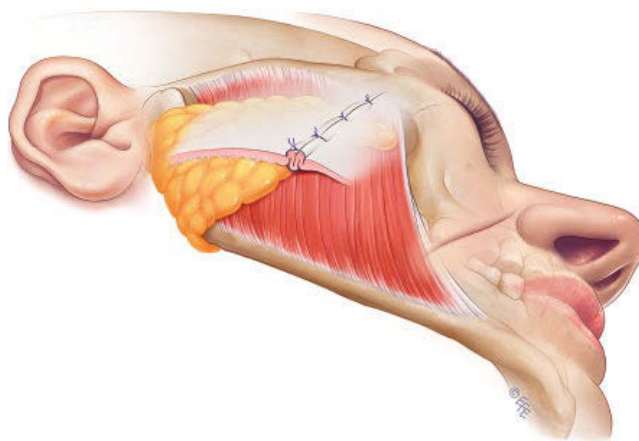


Figure 70.2 SMAS imbrication.

readily accepted until a surgical term – the deep plane – was introduced. Hamra's deep plane concept, being clearly descriptive of the dissection plane, was less anatomically confusing and the term became widely adopted and remains in use to this day. Although it is worth noting that Hamra's surgical plane of dissection transitioned from being sub-SMAS to becoming more superficial, as the dissection continued medially over zygomaticus major.

Some clarification regarding use of the term 'composite' is required, as two different, yet similar, meanings are attributed to it. In the context of flaps, a composite flap is one in which the several concentric tissue layers in a compound flap remain in their natural fused anatomical relationship. A scalp flap is the archetypal composite flap, consisting of the outer three layers – skin, subcutaneous layer and galea aponeurotica – forming a vertical composite. Elevation of a scalp flap involves dissection through the subgaleal areolar tissue glide plane beneath the flap (layer 4), allowing it to be separated from the underlying fixed deep fascia, the periosteum.

In Hamra's composite facelift, described in 1990, the term composite was used to denote the surgical extension medially of the same SMAS layer to incorporate the orbicularis oculi. According to Hamra's description, it is the inclusion of the lower lid orbicularis in the flap (elevation and repositioning) that determined the composite. Otherwise, a deep plane facelift does not impact the lower eyelid despite obtaining vertical cheek movement. Hamra's composite concept later progressed to include a Hamra composite cheek lift.³⁷ Essentially, this consists of a package of procedures: elevation of the lower lid through the deep plane (i.e. extended submuscular flap), using a 'zygobuccal' dissection and redraping the lid cheek in a superior medial vector after performing a septal reset procedure.³⁸

These techniques provide direct benefit to the midcheek and nasolabial fold. A standard facelift incision is made with 2–3 cm subcutaneous skin dissection. The fixed SMAS is incised over the parotid and a sub-SMAS dissection is continued to the malar eminence superiorly, dividing the zygomatico-cutaneous ligaments and their masseteric counterparts. Dissection is continued medially over zygomaticus major muscle towards the nasolabial fold, dividing the remaining zygomatico-cutaneous ligaments and providing access to the malar fat pad. Musculocutaneous flap is thus developed which is advanced and rotated superiorly. In the neck, the anterior neck is opened with approximation of the upper platysma edges and excision of the redundant medial platysmal border in the lower neck. This communicates with a superficial, preplatysmal dissection plane from the preauricular approach, with dissection carried 8–10 cm below the mandibular angle and defatting of the flap where indicated. This leaves separate dissection pockets in the face and the neck, which do not anatomically communicate. Excess skin is excised and closed, with tension on the SMAS applied in the temporal fixation.¹²

Hamra further addressed a limitation of facelifts – the failure to correct the upper cheek. He recognized that correction of the ageing changes of the lid-cheek junction (the lengthening of the

lower lid with lowering of the position of the upper cheek) are essential for complete facial rejuvenation. He further observed the benefit of correction of the orbicularis oculi laxity in the roof of the prezygomatic space, which is responsible for the appearance of malar mounds (crescent, bags) and modified his facelift to address this. To correct laxity of the orbicularis oculi of the upper cheek required an inferior extension of the submuscular lower lid blepharoplasty dissection, with communication through into the pocket of the previously dissected deep plane facelift. It was necessary to release the restraining effect of the ligamentous attachments along the orbital rim (later recognized to be the orbicularis retaining ligament) for this to occur.³⁸ In contrast to the superolateral vector usually used in redraping a lower lid blepharoplasty flap, a superomedial vector is applied to oppose the inferolateral vectors of upper cheek ageing. The tightening is achieved at the completion of the facelift by an orbicularis flap suspension to the lateral orbital rim.^{13,37}

Hamra's composite facelift clinical series demonstrated impressive outcomes as a result of the anatomically composite musculocutaneous flap that provides robust vascularity with reduced tension on the skin closure. In addition, the surgically composite access to the malar fat pad and superomedial cheek-lift vector corrects the lid-cheek area more effectively than do the extended and high SMAS counterparts. Similar to these techniques, potential damage to facial nerve branches in the dissection field and higher operative technical requirements with longer postoperative recovery are disadvantages that have limited usage of this specific technique.

Lateral SMASectomy

In the 1990s Daniel Baker developed this practical technique (Figure 70.3), which became extremely popular, usually in association with a 'short scar' (shorter skin incision) technique.³⁹ Baker recognized the lack of benefit from dissection of the fixed lateral SMAS, whereas the real benefit came from tightening the mobile SMAS over the anterior masseter area. Accordingly, the tedious sub-SMAS dissection over the parotid was avoided.

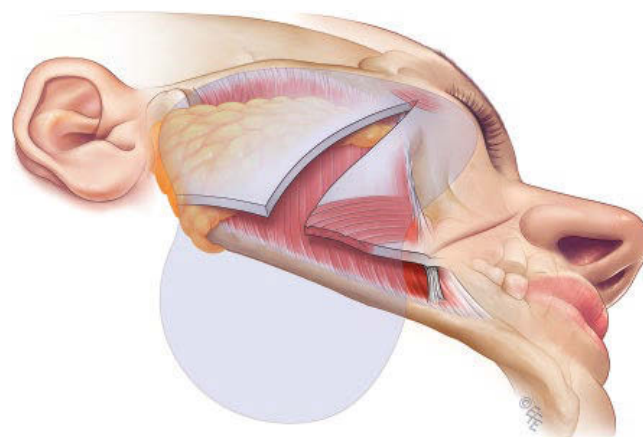


Figure 70.3 SMASectomy.

Baker also popularized the 'short scar' technique by limiting the postauricular skin incision and undermining behind the ear. Of course, this is successful in proportion to the amount of neck skin redundancy and the degree of its correction. The standard SMASectomy dissection begins with the lateral cheek skin flap dissected in the subcutaneous plane to within 2–3 cm of the nasolabial fold, and 5–6 cm below the level of the mandible into the neck. The mobile SMAS anterior to the parotid border is identified and, depending on the individual patient's facial morphology, the redundant SMAS here is either resected or plicated. Where a resection is planned, the mobile SMAS anterior to the parotid is manipulated in a vector perpendicular to the nasolabial fold from the lateral malar eminence to the angle of the mandible. The redundant SMAS is marked and superficially excised before the anterior, mobile SMAS is secured against the fixed lateral SMAS overlying the parotid, commencing inferiorly and moving superiorly. In a thinner face this technique allows for 'stacking' of the SMAS to augment that lateral cheek. Various modifications of the technique have been reported.⁴⁰

Similar to simple SMAS plication, this technique corrects the lateral cheek and jowls, and in addition provides for resection of redundant SMAS where indicated. Its shortfall is the failure to address the deep ligamentous attachments of the mobile SMAS along the anterior masseter border. While relative simplicity is its attraction, by design this technique does not fully address the midface.

Extended SMAS

This is an early SMAS technique that evolved to incorporate the understanding of the ligamentous anatomy on the zygoma and its relationship with the SMAS of the anterior face (Figure 70.4).^{16,41}

Visible contour changes of the cheek are a key part of the ageing process, particularly the reduction of prominence over the zygoma and loss of the contrasting infrazygomatic (submalar) youthful concavity over the buccal recess. The zygomatic liga-

ments are recognized as suspending the cheek soft tissues, including the malar fat pad from the zygoma.^{6,9} Descent of this facial fat has been the explanation for these contour changes and presumed to be secondary to attenuation of the zygomatic ligaments with age. In reality, given the stout nature of these ligaments, the weakening more likely occurs in the SMAS attachments of the ligaments.

The principles of this technique are based on the anatomical details of the retaining ligaments of the face, as well as consideration to vectors of facial and neck rejuvenation originating from the pioneering work of Bruce Connell.¹⁷ A bidirectional manipulation of skin and SMAS is advocated. A standard facelift skin incision is used; the skin is dissected widely in standard fashion to a few centimetres short of the nasolabial fold and below the mandible. The skin overlying the lateral two thirds of zygoma is mobilized. It is imperative to leave some fat on the SMAS, especially medially as it thins out to reduce proneness to tearing during the subsequent SMAS dissection and elevation. Following the standard posterior dissection of the fixed SMAS over the parotid, the forward extension of the SMAS incision commences 1 cm below the zygomatic arch (to protect the frontal branch of the facial nerve) and continues to the junction of the arch and body of the zygoma. Then the SMAS incision is angled towards the lateral canthus for a distance of about 3 cm, following which the SMAS incision is continued at 90°, away from the lower lid towards the superior nasolabial fold.¹⁶ The SMAS dissection provides access to the zygomatico-cutaneous ligaments, including the strong medial ligament just medial to zygomaticus minor that extends from the zygomatico-maxillary suture.⁷ The preauricular SMAS incision is extended below the mandibular border for 5–6 cm along the posterior border of the platysma. In the malar region the flap is elevated superficial to the orbicularis oculi as well as superficial to the zygomaticus major and minor muscles. Mobility of the SMAS flap is obtained by freeing it from the zygomatic prominence along with release of the masseteric ligaments. Often some extended dissection of the SMAS is undertaken medial to the zygomaticus minor, beyond the boundaries of the subcutaneous dissection, in the plane between the malar fat pad and levator labii superioris.

The malar SMAS flap is redraped in a superolateral vector parallel to zygomaticus major and fixed to the zygomatic prominence. The excess tissue is either excised or used as a soft tissue auto-augmentation over the zygoma. The remaining cheek SMAS flap is rotated in a perpendicular direction to the mandible to contour the jowl area. To accentuate the jawline and contrasting submental contour and neck concavity, a strong posterior vector is obtained by securing it to the mastoid using an inferiorly based flap of a preauricular SMAS as a strip transposed behind the ear. Drains are usually required on account of the extensive subcutaneous dissection.^{16,41}

This SMAS rejuvenation technique is based on sound anatomical principles of releasing the SMAS from its deep fixation by key retaining ligaments. Its advantage over the other SMAS

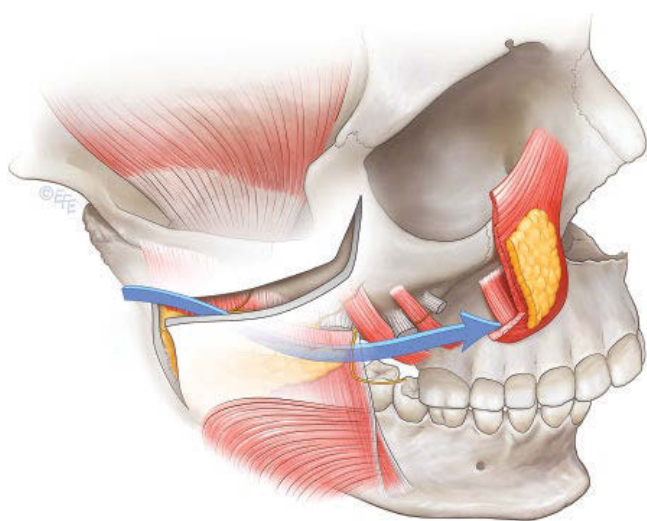


Figure 70.4 Extended SMAS facelift.

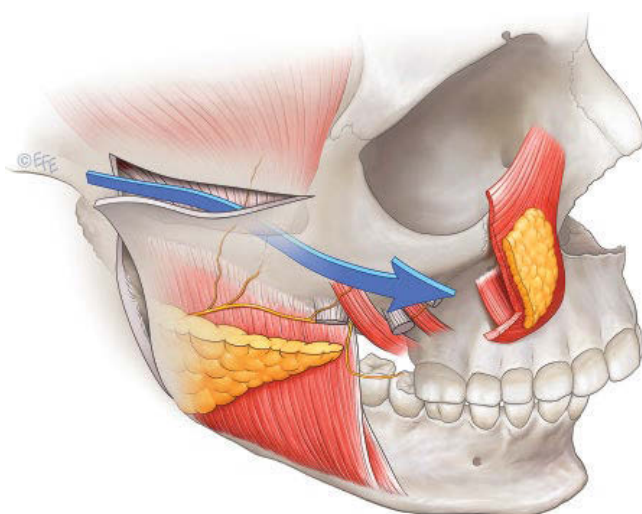


Figure 70.5 The high SMAS facelift.

techniques discussed so far is the access to the malar fat pad and improvement of the anterior part of the midface.

The high SMAS facelift (Fig 5)

The 'high SMAS facelift' conceived by Barton in the early 1980s and also advocated by Owsley, another pioneer in the application of SMAS anatomy, continues to grow in popularity among experienced plastic surgeons.⁸ It is not dissimilar to the extended SMAS but utilizes a simpler and higher approach to the upper SMAS to improve the benefit of SMAS redraping over the body of the zygoma and into the area of the lid-cheek (Figure 70.5). This area is not addressed with standard SMAS operations (imbrication/plication and lateral SMASectomy).¹⁴ Leverage of the upper SMAS over the zygoma results from raising the transverse SMAS incision, from inferior to the zygomatic arch to slightly cephalad.

Although a standard facelift incision is used, the skin undermining is minimized to 4–5 cm in the preauricular area and extends above the zygomatic arch and forward to the lateral border of the orbicularis oculi. The inferior subcutaneous dissection in the neck is continued superficial to the platysma from mastoid to the midline. To establish the correct plane, the fixed SMAS dissection commences over the parotid, then at the anterior border of the parotid the mobile SMAS is undermined using blunt dissection, protecting the facial nerve branches. The risk of damage to the frontal branches of the facial nerve is avoided by keeping the transverse SMAS incision superficial, where the nerves are deeper on the periosteum. The lateral edge of orbicularis oculi is used as a guide to the origin of the zygomaticus major. A thin layer of fat is left over this muscle to protect those zygomatic facial nerve branches passing superficially to innervate the lateral orbicularis oculi. The pattern of nerve supply and anatomical boundaries of the prezygomatic space are detailed by Mendelson *et al.*⁹ Releasing the SMAS from the fixation of the zygomatico-cutaneous retaining ligaments and

accessing the subcutaneous plane over zygomaticus major allows mobilization of the midface cheek mass, including the malar fat pad. The flap is strong as it is reinforced by the multiple ramifications of the zygomatic ligament and is lifted in a vertical direction and secured to the deep temporal fascia above the arch, where it not only suspends the cheek but also provides supports to the lower eyelid.¹⁹

The advantage of this technique is that, similar to its extended counterpart, it incorporates midface rejuvenation in addition to the jowls and neck. Elevation of an anatomically composite skin-SMAS flap provides a better vascularized flap than separate skin and SMAS flaps, with less bruising, induration and haematoma risk. The reluctance over the years to use a high SMAS incision had been the concern about the risk of damage to the temporal nerve branches as they cross the zygomatic arch. However, this has been strongly repudiated by good anatomical descriptions and the combined results from Owsley and Barton of 3000 cases with no permanent temporal branch injury.^{42,43} The traditional concern about not being able to redrape the skin and SMAS in different vectors is not a realistic shortcoming.⁴²

Subperiosteal facelift

In 1980, Tessier extended his craniofacial approach to facial rejuvenation. Conceptually, the technique not only repositions the origins of midface muscles but also provides access for manipulation of the orbicularis oculi through a 300° range.⁴⁴ Utilizing a bicoronal or hairline and, later, endoscopic approach and subperiosteal dissection plane, the lateral and midface are detached from the zygoma and maxilla. The origins of the superficial muscles, zygomaticus major and minor, levator labii superioris, levator anguli oris, as well as orbicularis oculi and related ligaments are inherently detached with this dissection and are repositioned. In the lateral cheek the dissection continues with detachment of the overlying masseteric fascial layer from the zygomatic insertion, but is limited by its masseteric attachments. A fronto-glabellar dissection, if used, can continue to the tip of the nose, preserving the medial canthal ligament. The entire facial soft tissues is thus freed from its underlying skeletal attachment, is advanced 2–3 cm in the superior direction and is fixed against the deep temporal fascia.

Initial reports of this technique reported unacceptably high rates of frontal facial nerve palsies (up to 20%), as well as zygomatic branch palsies. The injuries were sustained over the zygomatic arch and body, respectively, where the zygomatic branch may be caught in suspending sutures.⁴⁵ An extended technique was developed after further anatomic studies addressing these concerns. The vital dissection over the zygomatic arch where facial fascial layers coalesce was modified. About 2 cm above the zygomatic arch an incision is made in the continuation of the subperiosteal pocket into the superficial layer of the deep temporal fascia, breaching the superficial temporal fat pad to protect the overlying temporoparietal fascia and frontal nerve branch. A periosteal incision at the superomedial margin of the zygomatic arch along its length allows access to the lateral cheek where the masseteric fascia

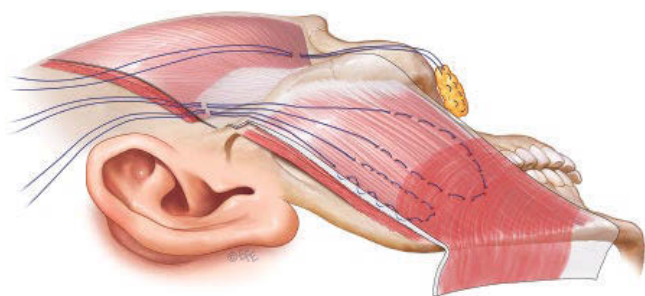


Figure 70.6 The MACS lift.

is detached from its zygomatic origin. This is combined with a minimal preauricular subcutaneous dissection. The redundant skin is excised after suspension of the face and the wounds closed.⁴⁶

The disadvantages of this technique include the steep learning curve and need for increased familiarity with craniofacial principles, but most significantly, the prolonged swelling that leads to longer postoperative recovery times. The periosteum strongly reattaches to the bone, carrying the facial soft tissues including the retaining ligament and muscle origins previously detached. Studies demonstrate that origins and insertion of the facial muscles do not attenuate with ageing.⁴⁷ Relocating the origins of these mimetic muscles inevitably leads to unnatural vectors of facial animation, which may account for the name 'mask lift'. Although effective, interest in this procedure diminished with advances in anatomical understanding. The original attraction had been the quality of correction obtainable over the zygoma, while avoiding the nerve risks of sub-SMAS dissection in this area, at the time when this lack of anatomical understanding was prohibitive.

Minimal access cranial suspension (MACS lift)

This technique was developed more recently, at a time when each advance in facelifting seemed to be further anatomically complicated. The original attractions of the MACS lift were its simplicity and safety, with short operating time and short postoperative recovery. Developed by Tonnard and Verpaele, it is based around a subcutaneous dissection with unusual short scar, utilizing a simple SMAS manipulation with a vertical skin vector, without displacement of the hairline (Figure 70.6). The original MACS lift addressed the lower third of the face, the jowls, marionette lines and neck.⁴⁸ A subsequent modification addresses the midface and malar fat pad and is called the extended MACS lift.^{49,50}

A zigzag temporal pre-hairline incision is made from the level of the lateral canthus and the lower end of the incision finishes at the earlobe, not extending postauricular. In the extended variant the temporal incision is extended 1–2 cm cephalad. Wide skin undermining is performed for 5–6 cm, extending to approximately 2 cm below the mandibular angle until the posterior platysma border is visualized. The facial shaping is achieved by a pair of U-shaped loop sutures anchored above to the deep temporal fascia above the zygomatic arch, 1 cm anterior to the helical root and weaved into the SMAS below. In the standard

technique the posterior suture extends down to the posterosuperior border of the platysma and because of the mobility of the platysma, as the pursestring suture is tied the platysma laxity and displacement is tightened, which contours the cervicomental angle and the pre-liposuctioned neck. A second, more open O-shaped suture originating from the same site is angled at 30° towards the jowls. Tightening the second suture places the mobile SMAS under concentric tension, reducing the laxity that contributes to the jowls and marionette lines. Where an extended technique is performed, a third U-shaped suture is placed in the furthest anterior limit of the deep temporal fascia and is directed towards the malar fat pad, suspending it with an oblique vector. The redundant skin is then excised in a purely vertical vector. The redundant subciliary skin at the lower lid is excised (4–8 mm on average) through a lower lid blepharoplasty incision.^{48,50}

Well performed, the results are surprisingly good for such a non-invasive technique, making it an excellent facelift procedure for the occasional facelift surgeon. Selection of the technique is based on a personal risk–benefit analysis, where an extensive skin undermining is considered preferable to more involved SMAS techniques. The shorter operative time advantage has diminished due to the requirement to enhance the result with further suspension sutures, yet the safe plane of dissection and reduced risk of major complications are the real advantages of this technique. It is often performed under local anaesthetic and sedation; patients are frequently discharged home the same day.

The focus on a pure vertical preauricular lift, which is considerable, is designed to avoid the skin redundancy behind the ears that results from neck correction, and instead transfers the skin excess to the temple, where it is removed without displacement of the temporal hair. The necessity for a pre-hairline scar is considered a disadvantage compared to the usual postauricular scar and is debated. The location of the loop sutures avoids a 'flat' facelift look. Although the anatomical basis of the technique does not require any release of the facial retaining ligaments at the sub-SMAS level (with its attendant drawbacks), the procedure does cleverly take advantage of the mobility of the platysma and the midcheek SMAS. Its critics consider the disadvantages of the technique manifold. The results are lasting as the SMAS plication suture causes multiple small imbrications with fibrosis induced around the suture, whose benefit is maintained after the suture has resorbed.

Directions in the further evolution of the facelift operation

Although facelifting has progressed beyond understanding of the SMAS and the retaining ligaments, concerns about the facial nerve location remain a major consideration in the selection of facelift procedure. The nerves and retaining ligaments are two of the main structures that traverse the sub-SMAS plane (the deep plane between the superficial and deep fascias). However, the largest area in this interval is occupied by the sub-SMAS (glide plane) facial spaces, whose clinical significance has only recently become appreciated. Although the spaces function to



Figure 70.7 Minimal dissection composite facelift. Skin flap undermining is limited to the cross-hatched area. Sub-SMAS dissection only is employed forward of this, and is limited to the pre-masseter spaces. No dissection is required forward of the yellow line at the anterior border of the masseter.

allow facial movement, their presence accounts for many of the characteristic ageing changes of the face, such as malar mounds and jowls. For surgeons, there are significant advantages in knowing that the spaces are effectively, 'pre-dissected' and are 'safe spaces'. These characteristics allow for a relatively rapid, bloodless and safe surgical access into the face, as the facial nerve branches and vessels remain outside the spaces. The nerves are at all times behind the walls of the spaces. Where the nerve branches become superficial and are exposed to shearing forces, they travel under the 'protection' of retaining ligaments. In surgery, the nerves are only at potential risk when dissection is performed outside the spaces; and then only at real risk where they are in close relation to the retaining ligament the surgeon intends to lyse. In short, the nerves and major ligaments are found close together outside the walls of the spaces.

The surgical principle is to first locate and enter and then define the boundaries of the spaces using blunt dissection. Then, when dissecting in the area between the boundaries of the spaces, the dissection technique is changed, to a precise, controlled vertical spreading. This technique safely and progressively releases the areolar tissue, allowing gentle definition of the nerves and their relationship to the ligaments that need release. For safety, the nerve may be eased aside before the ligament is released. Then for the facelift reconstruction a suture is placed into the upper end of the ligament where it embraces the overlying SMAS.

An important future direction, to make facelifts more acceptable, is faster postoperative recovery. Postoperative bruising following an anatomically composite facelift does not occur under the composite flap, it occurs only in the posterior subcutaneous dissection part. With a change in surgical emphasis it is possible to design the facelift flap so that there is, essentially, no residual area of subcutaneous dissection and therefore no bruising on the face as a result of the facelift (Figure 70.7). A complete composite facelift is the absolute opposite of the MACS lift, which requires the use of an extended subcutaneous flap. To perform a complete composite, the location of the vertical

SMAS incision is planned so that the posterior edge of the mobilized SMAS flap reaches, and is sutured to, the fixed SMAS at the level of the skin incision. This enables the entire facelift flap to be composite (i.e. without any subcutaneously undermined skin) so that bruising and induration is avoided.⁵¹

Complications

Haematoma

This remains the most common early postoperative complication of facelifts. Correct treatment of haematomas is required to avoid secondary consequences of skin necrosis, prolonged facial oedema or pigmentation. Large series have reported rates of 0.01–4.5% and as high as 8% in males.^{37,52–55} In general, there is much less bleeding with sub-SMAS dissection compared to subcutaneous dissection and the haematoma rate is considerably less. In their series of 1078 consecutive facelifts, Waterhouse and Jones reported a 4.5% haematoma rate.⁴⁰ They identified statistically significant relationships between anterior plastysmaplasty, systolic blood pressure, male gender, smoking and consumption of non-steroidal anti-inflammatory drugs (NSAIDs) and postoperative haematoma requiring evacuation.⁵⁴

Nerve injury

The sensory innervation of the facial skin flap is always damaged from subcutaneous dissection, the extent of numbness being in proportion to the extent of dissection. This usually resolves in 15–18 months. The greater auricular nerve branches are at risk during the neck dissection.

All facial motor nerve branches are considered at risk during facelifts. Traction neurapraxia is the most frequent injury, leading to temporary muscle paresis that resolves with time. A major neurapraxia may be evident for 12–16 weeks. The buccal branch is thought to be the most commonly affected, but fortunately, because it has multiple interconnecting branches with intraneural overlap, its clinical significance is small. Frontal and marginal mandibular branch injuries are more clinically significant as these are terminal branches. Permanent facial nerve injury is a major complication and fortunately rare, being reported in several large series with a range of 0–0.3%. Most nerve injuries recover during the postoperative months.^{37,46,55}

Skin loss

Traditional facelift techniques utilize large random, often thin, flaps sutured under tension that predispose to areas of skin loss from circulatory deficiency. These are most often postauricular and managed conservatively to allow secondary healing. Skin loss is an avoidable complication with recognized predisposing risk factors, smoking being the major risk. Smoking was implicated in 74% of patients with skin loss in a major retrospective analysis by Rees and smokers estimated to be 12.5 times more likely to suffer skin necrosis.⁵⁶ Another prospective series estimated skin necrosis in 19.5% of smokers and only 5% of non-smokers.⁵⁷

Unsatisfactory scars

Skin hyper- or hypopigmentation, hypertrophic and keloid scarring are possible general scarring complications. Earlobe distortion and temporal hairline deformities are consequences of incision design error and traction from excess skin tension, as are loss of hair and scar widening.

Infection

Infections are not a problem in facelift surgery and occur in less than 1% of cases.⁵⁸

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CHAPTER 71

Neck lift

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Introduction

Why perform a neck lift?

Why perform a neck lift? Why not just lift the cheeks and jawline or perform other procedures that rejuvenate the eyes or upper face? The simple answer is that a well-contoured neck is an artistic imperative to an attractive and appealing appearance. A good neckline conveys a sense of youth, health, fitness and vitality, and lends an appearance of decisiveness, sensuality and beauty (Figure 71.1). Neck improvement is of high priority to almost every patient seeking facial rejuvenation, and the results of 'facelift' procedures are largely judged by the outcome achieved in the neck. If the neck is not sufficiently improved our patients will feel we have failed them.

How should a neck lift be performed?

It is unlikely there will ever be a consensus on how a neck lift should be performed and it is a fact that no one procedure will be best for all patients. The technique used cannot be arbitrary, will depend on deformities present, and will necessarily vary from patient to patient. Success or failure in treating the neck lies in the diagnosis of problems and the application of a logical surgical plan, and any surgeon capable of identifying the anatomic basis of patient problems and forming a sound plan for their correction can achieve excellent outcomes.

So how should one perform a neck lift? Perhaps it is easiest to start by stating what not to do. Despite the fact that it is a common practice, it is not enough to perform submental liposuction and tighten the skin in most patients as such an approach ignores a number of anatomical problems present in many patients seeking neck improvement, including platysmal laxity, platysma bands, excess subplatysmal fat, large submandibular glands, digastric muscle hypertrophy and developmental factors such as the size and shape of the bony jaw and chin. Removing subcutaneous fat and tightening skin over these problems does not correct them, and the presence or absence of each must be looked for in order to create and apply an appropriate surgical plan (Figure 71.2).

Treatment options

Patients seeking neck improvement have a range of options available to them depending on the problems present, the degree of improvement they seek, and the time, trouble and expense they are willing to undergo to obtain the improvement they desire. And while it is incumbent upon us to discuss these options and the advantages and disadvantages of each, patients are also seeking our guidance as to what is possible, what is practical, and what is really best. It is not enough to steer patients to procedures we are comfortable with. We are professionally bound and ethically obliged to refer patients for the care they need and desire if we are not able to provide it ourselves.

Submental liposuction

Submental liposuction is the simplest and likely the most commonly performed procedure to improve neck contour in the range of options available to patients. It does not constitute a true neck lift, however, and only occasionally produces optimal outcomes. Patients and surgeons are predictably drawn into a sense of denial of this by the fact that submental liposuction will occasionally produce worthwhile improvement, and it is these atypical outcomes that are predictably displayed in offices, placed in advertisements, shown on TV and websites, published in the beauty press, and even included in presentations at plastic surgery symposia. Adding to the deception is that fact that these photographs have usually been taken in the early postoperative period when swelling is still present, which obscures irregularities and underlying but untreated deep layer problems.

In reality, submental liposuction is an incomplete solution to neck problems for most patients. As a standalone treatment it suffers the significant drawback that it falsely assumes poor neck contour to be solely the result of the accumulation of subcutaneous fat, and it is conceptually flawed in that it does not address platysma laxity and other deep layer problems, which together typically play a much larger and more important role in the ageing neck and neck contour deformities. As a result, most patients undergoing this procedure achieve improvement, but



Figure 71.1 Patient seen before and after neck lift. A good neckline conveys a sense of youth, health, fitness and vitality, decisiveness, sensuality and beauty. (The patient has also undergone facelift, foreheadplasty, eyelid surgery and facial fat injections.) Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.



Figure 71.2 Patient seen before and after neck lift. It is not enough to perform submental liposuction and tighten the skin in most patients. Such an approach ignores anatomical problems present in many patients seeking neck improvement. A careful evaluation shows patient preoperatively (*before*) to be troubled by platysmal laxity, platysma bands, excess subplatysmal fat, large submandibular glands and digastric muscle hypertrophy. Removing subcutaneous fat and tightening skin over these problems does not correct them and cannot produce the type of improvement shown (*after*). Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

Figure 71.3 Typical outcome seen with submental liposuction, which is an incomplete solution to neck problems for most patients as it falsely assumes that poor neck contour is solely the result of the accumulation of subcutaneous fat, and it does not address platysma laxity and other deep layer problems which usually play a much larger role in the ageing neck. Note that the large submandibular salivary gland is still present, platysma laxity has not been corrected, and aggressive fat removal has made platysma bands more obvious.



not comprehensive correction of their neck problems, and ultimately relying on this technique as the only method to obtain improved neck contour will seldom be successful (Figure 71.3).

Submental liposuction is also a frequent cause of many vexing patient problems and complications as it often leads to unintended but inappropriate removal of subcutaneous fat essential to a natural and youthful appearance, and this can, in turn, expose underlying problems of deep layer origin. Misapplied

and overused, or when aggressive 'ultrasonic' and 'laser' techniques are overzealously employed, submental liposuction can also result in extreme over-resection of precious subcutaneous fat and very unnatural and objectionable appearances. These problems are typically not evident in the operating room or in the early post-op period when cervicosubmental tissues are swollen however, but appear later, and once present, are very difficult to correct (Figure 71.4).



Figure 71.4 Overexcision of subcutaneous fat with liposuction. The patient (*left*) has had aggressive 'small cannula micro-liposculpture' by an unknown surgeon, which resulted in too much fat being removed. The neck and submental region are irregular, unnatural and unattractive. A different patient (*right*) has had 'laser liposuction' performed by an unknown surgeon. Inappropriate over-resection of fat has resulted in harsh and unnatural contours along with unattractive exposure of platysmal bands.

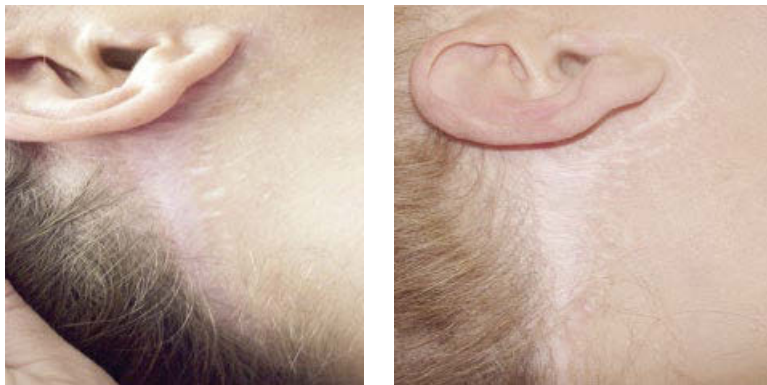


Figure 71.5 Overexcision of postauricular skin. It is a common error to attempt to lift the neck by tightening neck skin. It is not possible, however, to create a sustained improvement in neck contour in this manner and ill-conceived overtightening of neck skin will result in wide postauricular scars. Neck contour is properly created by modification of deep layer structures and only skin that is truly redundant should be removed. (Procedures performed by unknown surgeons.)

Liposuction, in combination with ill-conceived overtightening of neck skin, can add the additional objectionable problems of hairline displacement and wide postauricular scars, and compound the overall deformity (Figure 71.5).

Despite its many drawbacks and propensity to precipitate troublesome and vexing problems, the conceptual and comparative technical simplicity of submental liposuction is particularly appealing to inexperienced surgeons and non-surgeons performing aesthetic surgery procedures. Indeed, these characteristics of the technique almost guarantee that, effective or not, it will continue to be overused and inappropriately recommended to patients who are not good candidates for the procedure for the foreseeable future.

'Short-scar' neck lift (neck lift with submental incision only)

Although submental liposuction alone will rarely produce optimal neck improvement, a neck lift performed through a submental incision without any removal of skin can create attractive cervical contour in many patients (Figure 71.6).

This is due to the fact that, unlike liposuction, a neck lift performed through a submental incision allows deep layer problems and platysmal laxity typically present in the majority of patients seeking neck improvement to be addressed. This,

however, begs the question, how can good neck contour be created without removing and tightening the skin, and what happens to the 'excess' skin if only the deeper layer treatment is made and no skin is excised? The answer to this question is twofold: First, it is important to appreciate that in a properly performed neck lift contour is created by modification of deep layers of the neck, *not* by tightening the skin. Skin is intended to be a covering layer and serve a covering function. It is meant to stretch and give as we move and express ourselves. It is not intended to be a structural supporting layer, or to hold up sagging muscle and fat or lift hypertrophied structures lying beneath it. The second part of the answer lies in the increase in neck surface area that occurs when deep neck contour is improved. Improving neck contour by removing excess pre-platysmal fat when present, excising redundant subplatysmal fat and performing other deep layer manoeuvres as indicated, followed by tightening a lax platysma will result in a deepened cervicomenal angle, a longer curvilinear distance from the mentum to the sternal notch, and a more concave and geometrically larger and longer neck surface. When neck skin is re-draped and redistributed over the deeper, more concave surface, 'excess' skin is absorbed and none need be removed. These simple but not intuitive or immediately obvious facts underlie the reason that skin excision need not be performed in



Figure 71.6 Neck lift with a submental incision only. Although submental liposuction alone will rarely produce optimal neck improvement, a neck lift performed through a submental incision without any removal of skin can create an attractive cervical contour in many patients. Both of these patients had their procedures performed through a submental incision only. No incision was made in the periauricular area and there are no periauricular scars. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

patients with good skin quality and mild to moderate skin excess to obtain a good result.

Once the fact that increased neck surface area is produced by deepening the cervicomental angle is acknowledged and accepted it becomes clear why it is counterproductive to excise any skin from the submental incision as this can create a 'bow-string' effect and actually blunt the cervicomental angle. The submental skin incision is used for access to the neck only in a properly performed neck lift, and if skin excision is necessary it is more practically, effectively and logically removed from the postauricular area.

There is a limit to the amount of neck skin that can be absorbed and managed in this manner, however, and an isolated neck lift performed through a submental incision only is typically best for male patients and younger women with mild to moderate skin excess, good skin elasticity and minimal or modest ageing in the midface, cheek and jawline.

Neck lift with a chin implant

The difference between the presence of poor neck contour and microgenia is commonly misunderstood, and it is a common misconception that placement of a chin implant improves neck contour. A chin implant is a treatment for microgenia – not a poor neckline – and the presence or absence of microgenia and the need for a chin implant is a cephalometric determination that is independent of the condition of the neck. Placement of a chin implant when microgenia is not present is a conceptual and artistic error that will create unnatural appearances.

When true microgenia is present, however, placing a chin implant in combination with a neck lift will produce a more harmonious and balanced profile and a more aesthetic and attractive cervicofacial profile (Figure 71.7).

'Extended' neck lift and neck lift with facelift

If a significant amount of redundant skin is present, it must be excised to obtain the best result, and in such situations it is most logically and effectively excised in the postauricular areas using periauricular skin incisions. Skin excision from the submental incision is conceptually flawed and will actually degrade neck contour rather than improve it, and skin excision using large midline Z-plasties will result in a poorly concealed scar, and bizarre changes in beard hair inclination in men.

Skin excision can be combined with treatment of the neck through the submental incision as an 'extended' or 'long-scar' neck lift, or as part of a facelift procedure (Figure 71.8).

Practically speaking, most patients in need of a neck lift with skin excision also need a facelift as it is unusual to encounter patients with excess skin in the neck but not elsewhere on the face. This is particularly true in women and it is aesthetically inappropriate to perform an isolated neck lift in most women who present for facial rejuvenation. Lifting only the neck but not the cheeks, jowls and jawline can create an unnatural and unfeminine appearance.

Combining a facelift with a neck lift is almost always the best approach to obtain a balanced, natural and harmonious rejuvenation of the female face, although not all patients will recognize or accept this (Figure 71.9).



Figure 71.7 Neck lift with a chin implant. The difference between the presence of poor neck contour and microgenia is commonly misunderstood, and it is a common misconception that placement of a chin implant improves neck contour. When true microgenia is present, however, placing a chin implant in combination with a neck lift will produce a more harmonious and balanced profile and a more aesthetic and attractive cervicofacial profile. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.



Figure 71.8 Extended neck lift. Skin excision can be combined with treatment of the neck through the submental incision as an 'extended' or 'long-scar' neck lift, or as part of a facelift procedure. Both of these patients had skin excised as part of their procedures, and an incision was made in the periauricular area. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

Surgical planning

Planning the submental incision

Whether performing a neck lift alone or in combination with a facelift, optimal improvement in the neck can generally not be obtained in most patients unless a submental incision is made. Traditionally this incision is placed directly in and along the

submental crease in a well-intended but counterproductive attempt to conceal the resulting scar (Figure 71.10).

This incision plan should be avoided, however, as it will surgically reinforce the crease and accentuate a 'double chin' or 'witch's chin' deformity. Exposure of the submental region will also be compromised, and difficulty will be encountered when suturing or dissecting low in the neck. A more posterior placement



Figure 71.9 Comprehensive rejuvenation of the face. Combining a neck lift with a facelift and related procedures is almost always the best approach to obtain a balanced, natural and harmonious rejuvenation of the female face, although not all patients will recognize or accept this. This 55-year-old woman underwent facelift, neck lift, limited incision forehead lift, upper and lower blepharoplasties, chin augmentation, and fat transfer to the lips and cheeks. Addressing all regions of the face has produced a natural balanced outcome, and a far better result than neck lift alone (see also Figure 71.1). Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

of this incision will eliminate these problems, but still result in an inconspicuous and well-concealed scar (Figures 71.11 and 71.12).

The submental incision should be placed well within the mandibular shadow and well posterior but parallel to the submental crease at a point lying roughly one half the distance between the mentum and hyoid. This usually corresponds to a site situated 1–2 cm posterior to the crease (Figure 71.13).

The incision should be approximately 3–3.5 cm in length, but may be made longer as long as neither end will be advanced up upon a visible portion of the face when skin flaps are shifted. Healing will be best, and the scar will be best concealed, if it is made as a straight, and not as a curved line (Figure 71.14).

Planning treatment of the platysma

Assessing platysma deformity

The cervicosubmental region of each patient must be carefully examined both at rest and during platysmal activation if complete assessment of platysmal deformity is to be made. This

is best accomplished by asking the patient to ‘push the jaw forward and tighten the neck’. It is often helpful if the surgeon demonstrates this manoeuvre first for the patient to be sure it is correctly performed. Frequently an insignificant-appearing deformity at rest will be obvious upon muscle activation (Figure 71.15). Failure to recognize and appropriately correct these ‘dynamic deformities’ is the reason too many facelift patients appear improved in repose, but unnatural or bizarre in conversation, animation and during other activities that result in platysma muscle activation.

Examination of the cervicosubmental region with and without platysmal activation allows one to distinguish between ‘hard’ dynamic and ‘soft’ adynamic cervical bands. Soft, adynamic bands change little during platysma activation and are predominantly a problem of loose skin or horizontal platysmal laxity. Hard dynamic bands become tight or exaggerated upon platysmal activation and indicate a problem of platysmal hyperfunction.

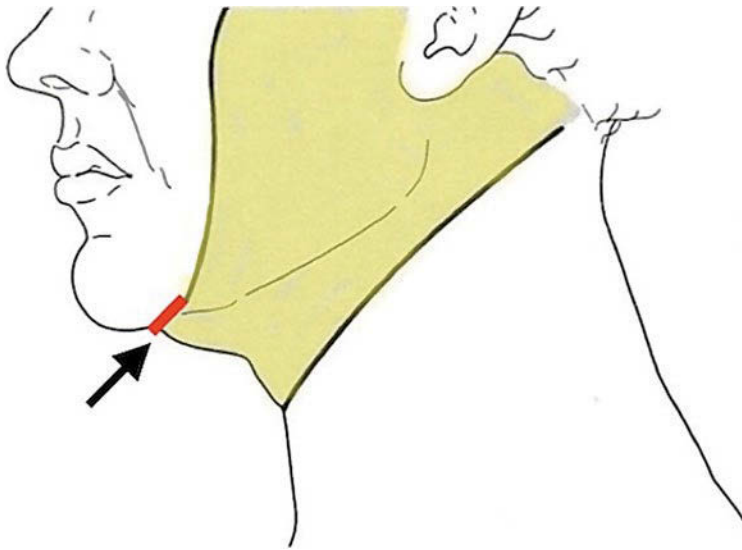


Figure 71.10 Traditional, but incorrect, plan for the submental incision. The incision should not be placed directly along the submental crease (arrow) as this will reinforce the submental retaining ligaments and accentuate the 'double chin' appearance. Note that typical plan of skin undermining (yellow area) also promotes a double chin appearance because the crease is not undermined, retaining ligaments are not released, and the fat of the chin and the neck cannot be readily blended to create a smooth transition between them.

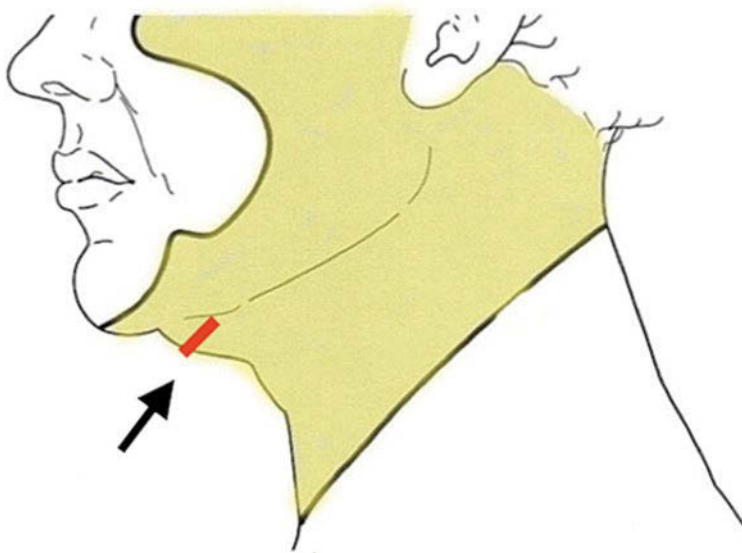


Figure 71.11 Correct location for the submental incision. Placing the submental incision 1.5 cm *posterior* to the submental crease prevents accentuation of the 'double chin' and witch's chin deformities and provides for easier dissection and suturing in the anterior neck (compare with Figure 71.10). Note that this incision plan allows the submental crease to be undermined and submental retaining ligaments to be released, and the fat of the chin pad and neck to be blended to create a smooth transition between them (Figure 71.12). Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

Treatment of horizontal platysma laxity and 'soft' bands

If submental support is poor due to horizontal platysmal laxity or platysmal diastasis and optimal improvement in the anterior neck is desired, *anterior platysmaplasty* is planned (Figure 71.16). Anterior platysmaplasty is the procedure in which the medial borders of the platysma muscle are sutured together to help consolidate the neck, reduce horizontal platysmal laxity and improve neck appearance when the patients flexes the neck and looks down (Figure 71.17). It is *not*, however, adequate or effective in the treatment of excess subplatysmal fat, prominent submandibular glands, hypertrophy of the anterior bellies of the digastric muscles, or the treatment of 'hard' dynamic platysmal bands. Treatment of these problems will require that other manoeuvres be performed.

Treatment of platysma hyperfunction and dynamic 'hard' bands

It is important to distinguish between 'hard' dynamic bands and 'soft' adynamic platysmal bands as treatment will vary depending on the type present. Patients with soft bands are usually adequately treated with skin excision or skin excision and platysmaplasty alone. Patients with 'hard' bands, however, require *transverse platysma myotomy*. The result of platysma myotomy is immediate, dramatic, long-lasting and superior to any suture, suture suspension or 'corset' technique. The length of trans-section required can be varied depending on the location and configuration of bands present, and will thus differ from patient to patient.

Platysmal bands are generally seen to lie in medial and lateral locations. Each band is situated near the medial and lateral



Figure 71.12 Correction of the 'double chin' deformity. (a) Patient with 'double chin' seen preoperatively. (b) Same patient seen after facelift and neck lift. The submental incision was made posterior to the submental crease to allow undermining and release on the submental retaining ligaments and blending of chin and neck fat. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

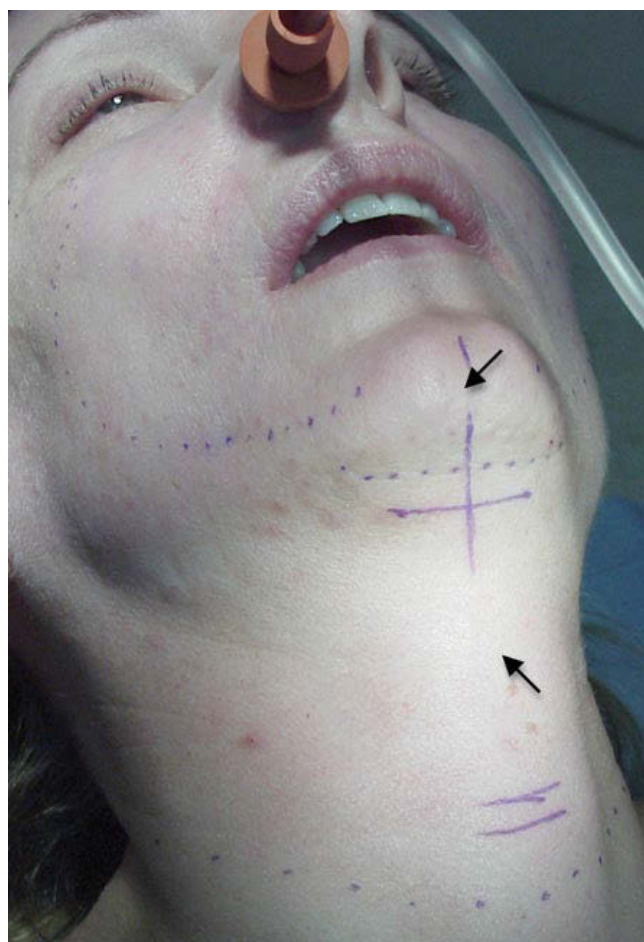


Figure 71.13 Plan for the submental incision. The submental portion of the facelift incision (solid line) should be placed posterior to the submental crease (dotted line), approximately one half the distance between the mentum and hyoid (arrows). Usually this corresponds to a point about 1.5 cm posterior to the submental crease.

platysma muscle borders, but does not usually correlate precisely with them. Patients with anterior platysmal bands only are planned to undergo transverse platysmal myotomy medially through the section of muscle on which the band is present at the level of the cricoid cartilage (Figure 71.18). The platysma muscles need not be completely transected when an anterior band only is present, just the region where the platysmal band is located.

If lateral, as well as anterior bands are present, the myotomy is carried more laterally to include them as well (Figure 71.19). If poor definition is present over the lateral mandibular border, in addition to anterior and lateral neck bands, platysmal myotomy is planned to continue 'full width' superolaterally up to the anterior border of the sternocleidomastoid muscle (Figure 71.20). A similar full-width myotomy of this type is also indicated in a firm obtuse neck with a 'short platysma' and poor definition over the mandibular angle, but no bands.

In many necks a subtotal myotomy only is indicated and need be performed. As a practical matter, however, complete transection of the platysma is performed in most patients to ensure the best contour possible, and to limit the possibility of band recurrence or that residual platysmal bands will be present. Despite assertions to the contrary, full-width platysmal myotomy will not result in an overly thin appearing neck or reduced support of the submandibular glands when properly performed. Medial wedge resections of platysma at the hyoid and vertical excisions of platysma bands, although shown in many textbooks, are not effective and usually result in unaesthetic exposure of underlying cervical anatomy and other secondary deformities. These procedures are not recommended and experience has shown that they should not be performed. Similarly, attempts to place rigid support across the cervicomental angle in the form of a mastoid to mastoid 'noose' of suture or commercially available straps to lift platysmal bands is conceptually flawed and practically problematic. Experience has shown that these manoeuvres do not work and are the source of many patient problems.



Figure 71.14 Healed submental incisions. Placement of the submental incision posterior to the submental crease will still result in an inconspicuous, well-concealed scar and simultaneously provide better access and exposure when working in the deep layers of the neck. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.



(a) Repose



(b) Platysma contraction

Figure 71.15 Dynamic assessment of the cervicosubmental region. The neck is examined in repose (a) and as the platysma is contracted (b). Dynamic platysma muscle irregularities are often referred to as 'platysma bands'.

Planning the modification of the submental region

Traditional neck lift techniques do not adequately address many aspects of ageing in the submental region and it is not enough in most situations to limit treatment of the neck to pre-platysmal lipectomy alone or with postauricular skin excision. For many patients subplatysmal fat accumulation, submandibular salivary gland 'ptosis' (enlargement) and digastric muscle hypertrophy will contribute significantly to the neck deformity present and necessitate additional treatment.

In all but the unusual or young patient the majority of cervical fat accumulation will be present in a subplatysmal location, and little if any will need to be removed from the pre-platysmal layer. Indeed, as patients age fat stores generally shift from a pre-platysmal location to a subplatysmal one, and the small amount of subcutaneous fat present in the typical patient presenting for a neck lift is necessary and must be preserved if a soft, youthful and attractive appearance is to be obtained. Although the necks of patients undergoing liposuction may look good initially when swelling is present, once it resolves, they typically display an unnatural and unaesthetic 'guttled'

appearance. Once one acknowledges this fact, the futility and undesirability of cervicosubmental liposuction in most patients becomes obvious.

Abnormal collections of subplatysmal fat can be identified by preoperative palpation of the neck with and without platysmal activation. Fat lying predominantly in a pre-platysmal position will generally feel 'soft' and will remain in the examiner's grasp upon platysmal muscle activation. Fat lying in a subplatysmal position, however, will have a firmer feel, and will tend to be pulled superiorly out of the examiner's grasp when the platysma is contracted (Figure 71.21).

Two typical scenarios where subplatysmal fat is likely to be present is in patients with firm, obtuse necks who have been troubled by lifelong cervicomenal fullness and patients presenting for secondary surgery who have poor cervical contour (Figure 71.22). In these individuals, partial subplatysmal lipectomy will likely be required, but complete removal of all subplatysmal fat is rarely indicated and deep cervical fat (intra-digastric fat) should not be removed. When subplatysmal and/or deep cervical fat has been erroneously overexcised, an objectionable depression in the submental region may result,

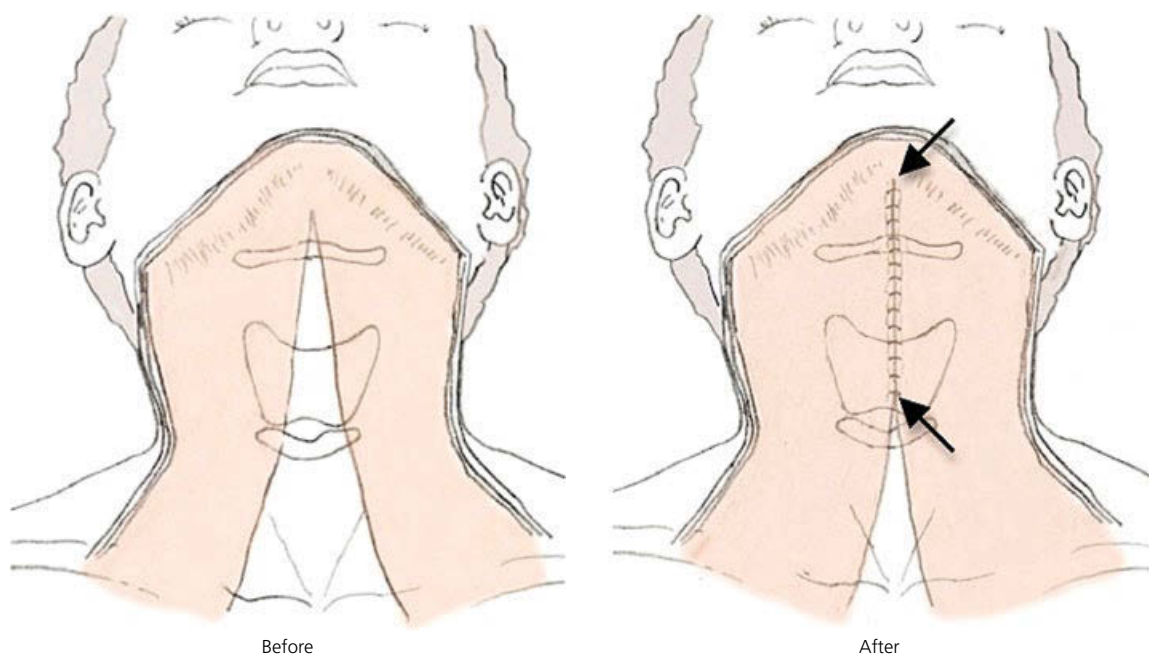


Figure 71.16 Anterior platysmaplasty. Repair of platysmal diastasis reduces horizontal platysmal laxity, improves submental support and helps consolidate the neck. It is not adequate or effective treatment of platysmal bands, however.

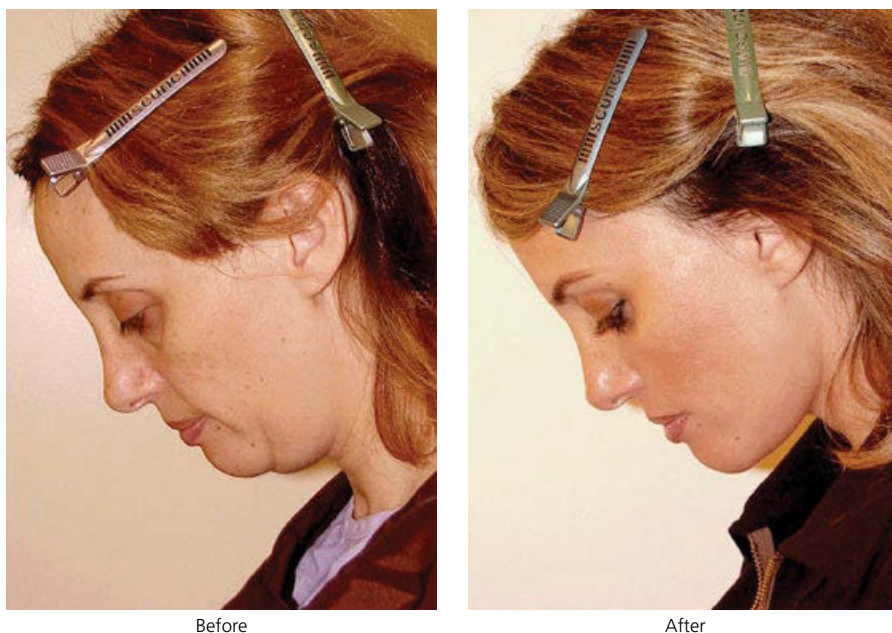


Figure 71.17 Anterior platysmaplasty. Platysmaplasty improves submental support and enhances neck appearance when the patient looks down. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

commonly referred to as the 'dug out neck' deformity (Figure 71.23). Typically this depression is more evident when the neck is flexed somewhat or when the patient swallows.

Planning treatment of the prominent submandibular gland

Preoperative assessment must be made of the submandibular glands as they often contribute to the appearance of a full, 'obtuse', and 'lumpy' neck. Large submandibular glands are most

easily seen in the secondary facelift patient who has had prior aggressive cervicosubmental lipectomy (Figure 71.24). Large glands are frequently hidden by submental fat or lax platysma muscle in the patient with a full neck presenting for a primary procedure, however, and a plan that does not recognize this fact will lead to disappointing and unexpected bulges in the lateral submental regions postoperatively.

Submandibular glands are usually palpable as firm, smooth, discrete, mobile masses in the lateral submental triangle, lateral

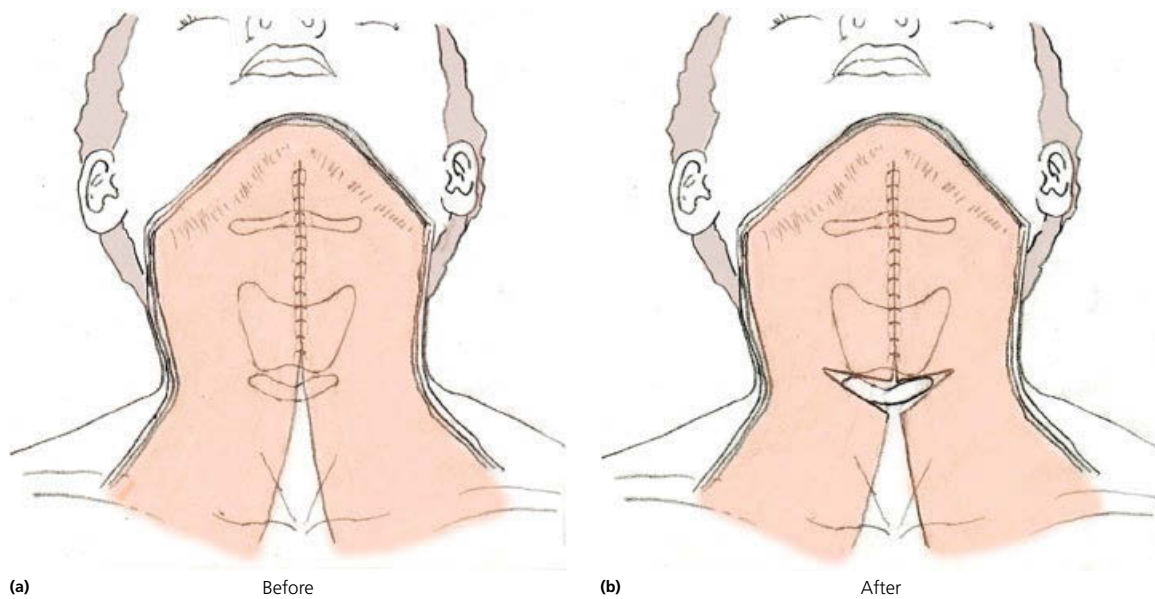


Figure 71.18 Plan for treatment of 'anterior' platysmal bands. Transverse platysma myotomy is performed in the region of the platysma muscle where the platysmal band is present. **(a)** Anterior neck after platysmaplasty but before anterior platysma myotomy. **(b)** After anterior platysmal myotomy. (Note: myotomy is most easily and effectively performed after platysmaplasty has been completed.)

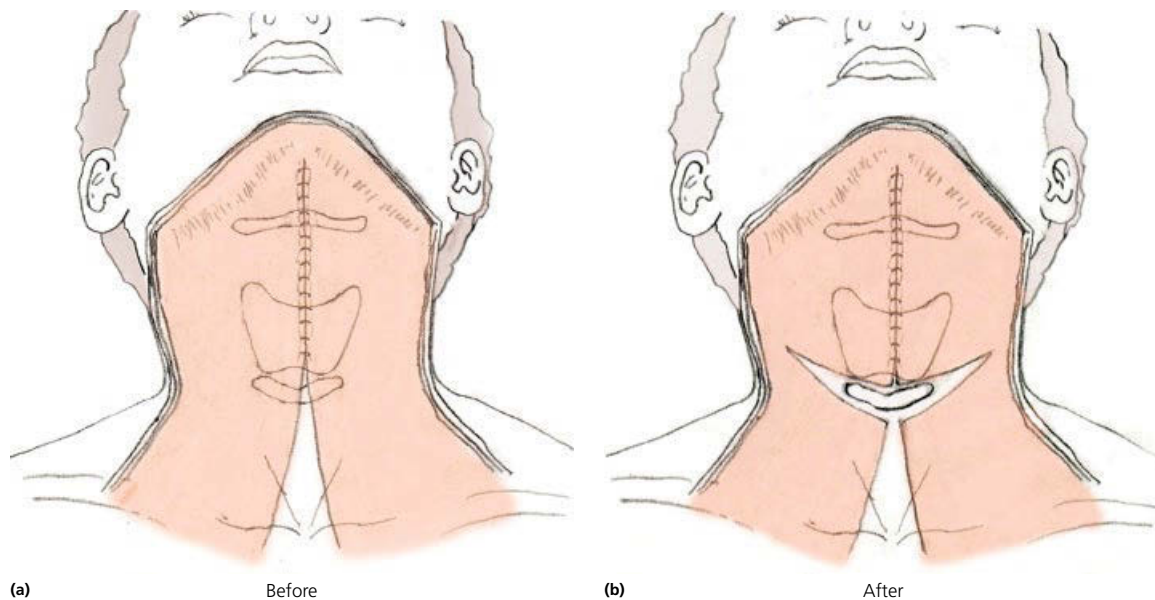


Figure 71.19 Plan for treatment of 'anterior' and 'lateral' platysmal bands. If lateral bands are present, the myotomy is carried more laterally to include them as well. **(a)** Anterior neck after platysmaplasty but before anterolateral platysmal myotomy. **(b)** After anterolateral myotomy. (Note: myotomy is most easily and effectively performed after platysmaplasty has been completed.)

to the anterior belly of the digastric muscle and medial to the inner aspect of the ipsilateral mandibular border. Submandibular glands lying superior to the plane tangent to the inferior border of the mandible and the ipsilateral anterior belly of the digastric muscle do *not* disrupt neck contour and will usually *not* require treatment. Glands protruding inferior to this plane are likely to be problematic, however, if excess cervical fat is removed, the platysma muscle is tightened and redundant skin is excised. These glands warrant treatment.

Despite claims to the contrary, experience has shown that prominent submandibular glands are actually large, *not* ptotic, and that the platysma contributes little to their position or support. Attempts at tightening the platysma when a prominent gland is present usually results in modest improvement that is short lived. The same is true with the various suture suspension methods that have been proposed and now largely abandoned. Careful sculpting of the periglandular fat will sometimes allow 'small' prominent glands to be disguised, but large, prominent

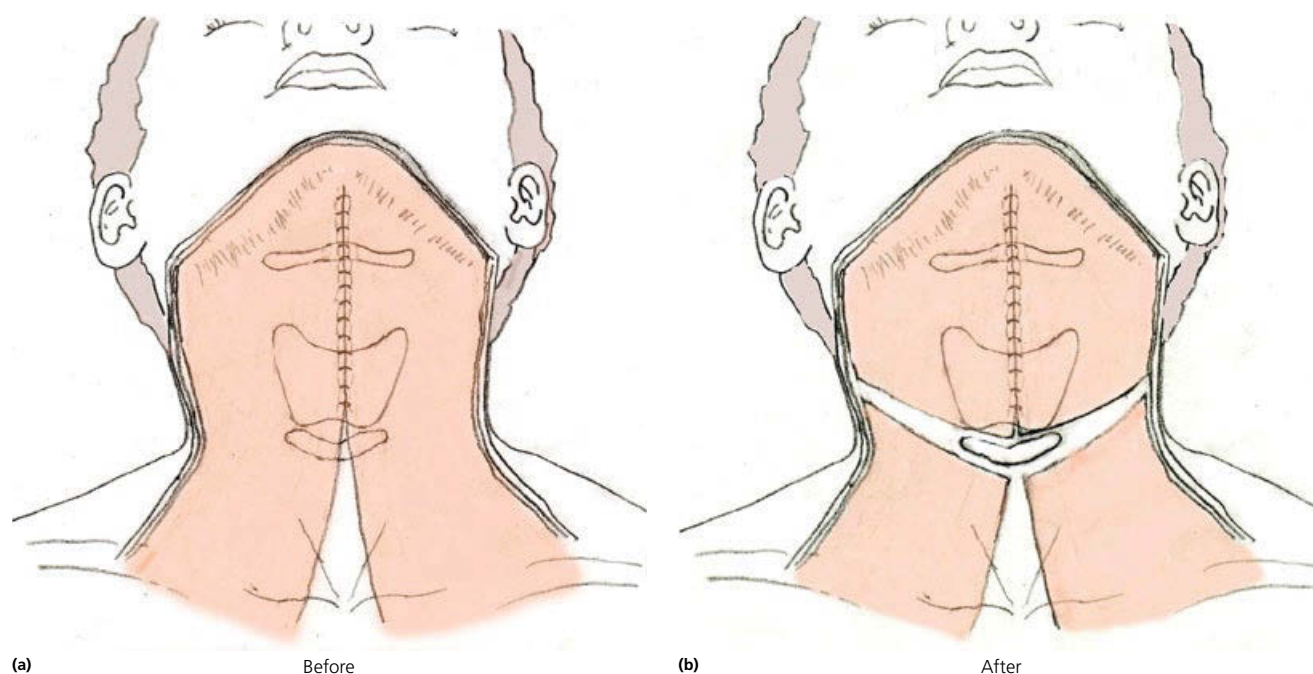


Figure 71.20 Plan for 'full-width' platysmal transection. Full-width myotomy not only corrects anterior and lateral bands, but also provides improved definition over the lateral mandibular border. (a) Anterior neck after platysmaplasty but before full-width platysmal myotomy. (b) After full-width platysmal myotomy. (Note: myotomy is most easily and effectively performed after platysmaplasty has been completed.)



Figure 71.21 Assessing the location of cervicosubmental fat. (a) Submental 'waddle' is grasped with the face and neck in repose. (b) The patient is then asked to activate the platysma muscle. In this example, fat is pulled superiorly from the examiner's grasp, indicating a predominantly subplatysmal position. Fat lying predominantly in a subcutaneous pre-platysmal position would tend to remain within the examiner's grasp when the platysma is activated.

glands will require that the protruding portion be resected if optimal improvement is to be obtained (Figure 71.25).

The decision to perform submandibular gland reduction should be made in conjunction with the patient after appropriate

discussion of the advantages and disadvantages of the procedure. Patients should know that the procedure may prolong submental induration and oedema, and carries a small theoretical but unlikely risk of resulting in bleeding,



Figure 71.22 Patients likely to have subplatysmal fat accumulation. (a) Young patient with firm, obtuse neck who has been troubled by lifelong cervicomenmental fullness. These patients typically have large collections of subplatysmal fat and are suboptimal candidates for submental liposuction. (b) Patient presenting for secondary surgery who has poor cervical contour. It can be seen that superficial subcutaneous fat has been stripped out at the primary procedure and the remaining fat is almost completely in a subplatysmal location.

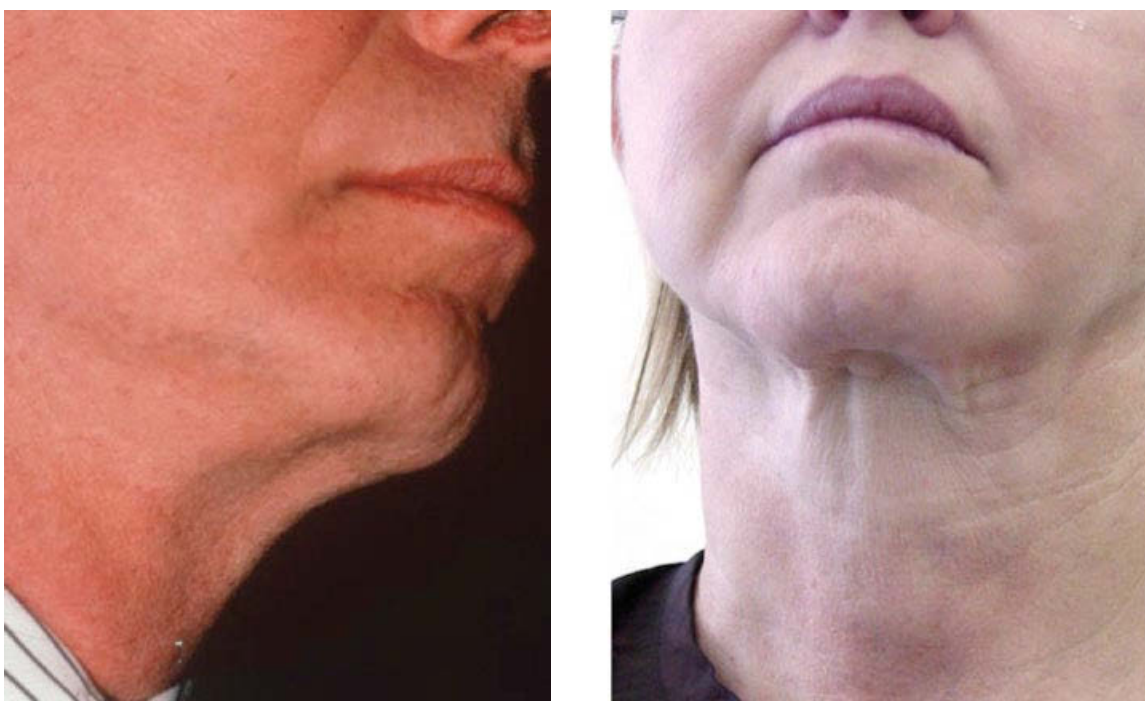


Figure 71.23 The 'dug out neck' deformity is a term used to describe situations in which too much subplatysmal and/or deep cervical fat has been erroneously overexcised, resulting in an objectionable depression in the submental region.

sialoma, salivary fistula and dry mouth symptoms. Patients should also be informed, however, that such problems are rare, that only the protruding portion of the gland is excised and that the majority of the gland is left in place and is not removed.

Planning treatment of prominent anterior belly of the digastric muscle

A small subgroup of patients will present with large, bulky anterior bellies of their digastric muscles that are evident as linear paramedian submental fullness (Figure 71.26).



Figure 71.24 Prominent submandibular glands are usually evident as protruding masses in the lateral submental triangle, lateral to the anterior belly of the ipsilateral digastric muscle and mandibular border. **(a)** Patient with residual prominent submandibular gland after facelift and submental liposuction. His prior procedure has made the prominent glands more obvious. **(b)** Patient with residual prominent submandibular gland after 'weekend neck lift'. Aggressive resection of subcutaneous fat has exposed the prominent gland, which was not treated.



Figure 71.25 Prominent submandibular glands. Despite claims to the contrary, experience shows that prominent submandibular glands are actually large and not 'ptotic' and will require that the protruding portion be resected if optimal improvement is to be obtained. **(a)** Patient with prominent submandibular gland that contributes significantly to poor neck contour. **(b)** Same patient seen after neck lift that included submandibular gland reduction. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

Large anterior bellies of the digastric muscles are most easily seen in the secondary facelift patient who has had prior aggressive cervicosubmental lipectomy. Large muscles are frequently hidden by excess submental fat or lax platysma muscle in the patient presenting for primary procedures, however, and failure to identify them may lead to unexpected and objectionable sub-

mental bulges postoperatively. When large, prominent digastric muscles are identified, subtotal *superficial digastric myectomy* should be considered. The final decision as to whether partial digastric myectomy should be performed is often best deferred until the day of surgery and the improvement produced by other modifications of the submental region can be evaluated.



Figure 71.26 Prominent anterior belly of the digastric muscle. The anterior belly of the digastric muscle can be seen as objectionable linear paramedian fullness in the submental region. Prominent digastric muscles often go unnoticed at the time of the primary procedure due to the fact that they are frequently hidden by cervical fat and lax platysma muscle.

Surgical Technique

Anaesthesia

Properly and comprehensively performed neck lift techniques can be time consuming, technically demanding, and can test the patience and composure of almost any surgeon. It is highly recommended that any surgeon new to these techniques enlist the services of an anaesthetist. This is particularly important when the procedure is to be performed upon a patient who is apprehensive, has a history of anaesthetic difficulties, hypertension or other significant medical problems.

The majority of our neck lifts are now performed under deep sedation administered by an anaesthetist using a laryngeal mask airway (LMA). The use of a laryngeal mask allows the patient to be heavily sedated without compromise of their airway, but she or he need not receive muscle relaxants and can be allowed to breathe spontaneously. An LMA is also less likely to become dislodged during the procedure than an endotracheal tube, and will not trigger coughing and bucking when the patient emerges from the anaesthetic. The *flexible* LMA is particularly useful in neck surgery. When this device is used and the breathing circuit is separately draped, the breathing circuit can be moved from side to side as needed to obtain unobstructed access to the cervicosubmental region. Most surgeons when initially considering using an LMA airway are understandably fearful that the bladder on the device when inflated will distort the cervical region and interfere with neck assessment and treatment. Fortunately, experience has shown this not to be a valid concern.

Preoperative preparations and care

Each patient receives a full surgical scrub of the entire scalp, face, ears, nose, neck, shoulders and upper chest with full strength (1:750) benzalkonium chloride (BAK or Zephiran) solution after anaesthesia is begun and bland ophthalmic ointment is instilled into each eye. The patient's head is then placed through the opening of a 'split sheet' or a split adhesive-backed disposable transverse laparotomy sheet, leaving the entire head and neck

region unobscured from the clavicles up. No 'turban' or 'head drape' is used. This allows unimpaired examination of the cervicofacial profile in its entirety throughout the procedure and provides complete access to the periauricular areas when required.

The breathing circuit is draped separately from the patient by wrapping it with a sterile paper drape secured with Steri-Strips (sterile adhesive wound tapes) or other sterile fastener of choice. This allows it to move during the procedure as the patient's head is turned from side to side. When drapes are applied in this manner it is not necessary to secure the LMA with tape or by other means as long as its position is watched by the anaesthetist and/or other members of the operating room team present. If an endotracheal tube is used, it can be conveniently and effectively secured to the perioral skin with a TegaDerm or similar sterile adhesive-backed plastic dressing.

Administering local anaesthesia

Local anaesthetic is administered even if deep sedation or general anaesthesia is used. This limits stimulation of the patient and the overall amount of narcotics and anaesthetics needed. A significant and helpful haemostatic effect is also obtained when adrenaline (epinephrine)-containing solutions are used.

Sensory nerve blocks are performed using 0.25% bupivacaine (Marcaine) with adrenaline 1:200 000. This allows the neck or face to be injected with a reduced degree of stimulation. Skin marked for incision is then infiltrated with the same solution. Areas of subcutaneous dissection are generously infiltrated with 0.1% lidocaine (lignocaine) (Xylocaine) with adrenaline 1:1 000 000 solution. It is not necessary, however, to over-inject local anaesthetic in a 'tumescent' fashion.

Infiltration of the preauricular cheek and postauricular areas is carried out using a 22 gauge spinal needle if an 'extended' neck lift or a facelift is being performed. Infiltration of the neck and submental region is carried out with a 1.6 mm multi-hole blunt-tipped infiltration cannula. No direct infiltration beneath the superficial muscular aponeurotic system (SMAS) or platysma is

necessary if the overlying subcutaneous tissues are infiltrated generously at the beginning of the procedure as described above.

Skin flap elevation

The submental incision should be made 1.5 cm or more posterior to the submental crease approximately half way between the mentum and hyoid. Making the incision in this location helps conceal it in the shadow of the mandible, avoids surgical reinforcement of the crease and accentuation of the 'double chin' and 'witch's chin' deformities, and provides better exposure of the deep neck.

Once the submental incision has been made, the surgeon should stand at the head of the table during dissection of the submental region, while the assistant retracts the edges of the incision with a pair of 10 mm double-pronged skin hooks. Skin undermining should be made using Metzenbaum scissors and the dissection should be made subcutaneously, leaving the majority of the subcutaneous/pre-platysmal fat on the platysma surface. This makes fat excision and sculpting easier later in the procedure if required, and precludes the need for difficult and tedious excision of fat from the undersurface of the cervical skin flap. Unlike the dissection of the facelift skin flap, in which a conscious effort must be made to prevent the flap from becoming too thick and the SMAS being compromised, a slightly deeper dissection should be made in the neck to preserve a slightly thicker layer of subcutaneous fat. Preservation of a thicker layer of fat helps avoid a hard or over-resected appearance in the cervicosubmental area, and objectionable overexposure of underlying neck anatomy postoperatively.

Submental dissection is continued inferiorly and laterally. If an 'extended neck lift' or concomitant facelift is being performed, this dissection will join the upper lateral neck dissection made through postauricular and peribulbar incisions.

Upon completion of subcutaneous undermining of the anterior neck and submental regions, a retrograde dissection should be made subcutaneously up onto the inferior chin. This will ensure the submental restraining ligaments have been divided and that the submental crease has been released from its bony attachments. Some bleeding is usually encountered during this manoeuvre, but this should not prevent one from completing this important step.

If a facelift or 'extended' neck lift is being performed and the upper lateral neck is being approached from a postauricular incision, care must be taken to avoid injury to the great auricular nerve. This nerve provides sensation to the lower two thirds of the ear and travels superficially superiorly in the upper lateral neck over the sternocleidomastoid muscle fascia. The most superficial portion of the nerve is anatomically constant and lies approximately 6.5 cm inferior to the auditory meatus, midway between the anterior and posterior borders of the sternocleidomastoid muscle and it is helpful to mark this spot preoperatively on the neck skin surface to guide one during dissection. If the dissection is performed correctly, however, the nerve will lie deep to the plane of skin flap elevation and will be

covered by a thin layer of subcutaneous fat. It is neither necessary nor advisable to attempt to specifically identify or isolate this nerve through additional dissection.

Patients with thin faces, or those undergoing secondary face-lifts, will not uncommonly have little subcutaneous fat between the superficially situated portion of the great auricular nerve and the skin, however. For this reason, extra caution must be taken in these patients if elevating the skin flap in this area, and a clear knowledge of nerve anatomy is required.

Cervical lipectomy

Cervical lipectomy is a technically demanding manoeuvre that requires patience, perseverance and artistic sensitivity. Contrary to what is traditionally practised, if a facelift is being performed in conjunction with the neck lift, subcutaneous lipectomy (including liposuction) should be performed *after* deep neck manoeuvres and shifting and suturing of the SMAS and platysma, and not at the beginning of the procedure. If subcutaneous lipectomy and/or submental liposuction is performed at the beginning of the procedure, fat will be mistakenly excised from regions of the neck that will be raised onto the face when SMAS flaps are advanced and suspended, and this can result in inadvertent creation of harsh or irregular mandibular contours (Figure 71.27). In almost all cases it is best to leave the subcutaneous fat layer untouched until all other manoeuvres are completed and improvements obtained with them assessed.

The key to obtaining good outcomes in the neck is in understanding the distribution of fat in the cervicosubmental region and choosing a treatment plan accordingly. Cervical fat is present in three distinct anatomic layers – pre-platysmal, subplatysmal and deep cervicosubmuscular ('intra-digastric'). In almost all cases cervical lipectomy should be limited to the two more superficially situated regions. Lipectomy technique is not as important as the contours created and it should be the aim of the surgeon to produce an attractive neck, and not simply one devoid of fat.

Exploration of the subplatysmal space is indicated if preoperative assessment suggests a significant collection of subplatysmal fat is present, large digastric muscles are noted or large submandibular glands are identified, or if uncertainty exists as to any aspect of the subplatysmal condition. Significant accumulations of subplatysmal fat can also be demonstrated intraoperatively by placing gentle superior traction on the cheek flaps (if facelift) or cheeks (isolated neck lift) and flexing the neck. If the neck appears full upon this manoeuvre, consideration should be given to exploration of the subplatysmal space and the removal of redundant subplatysmal fat and/or protruding portions of other enlarged structures.

Subplatysmal exploration is performed through the submental skin incision. The subplatysmal space is entered by incising the superficially situated fascia between the medial platysma muscle borders using a Metzenbaum scissors. A combination of blunt and sharp scissors technique is used to isolate each muscle edge. The muscle edge is then grasped and retracted by the assistant

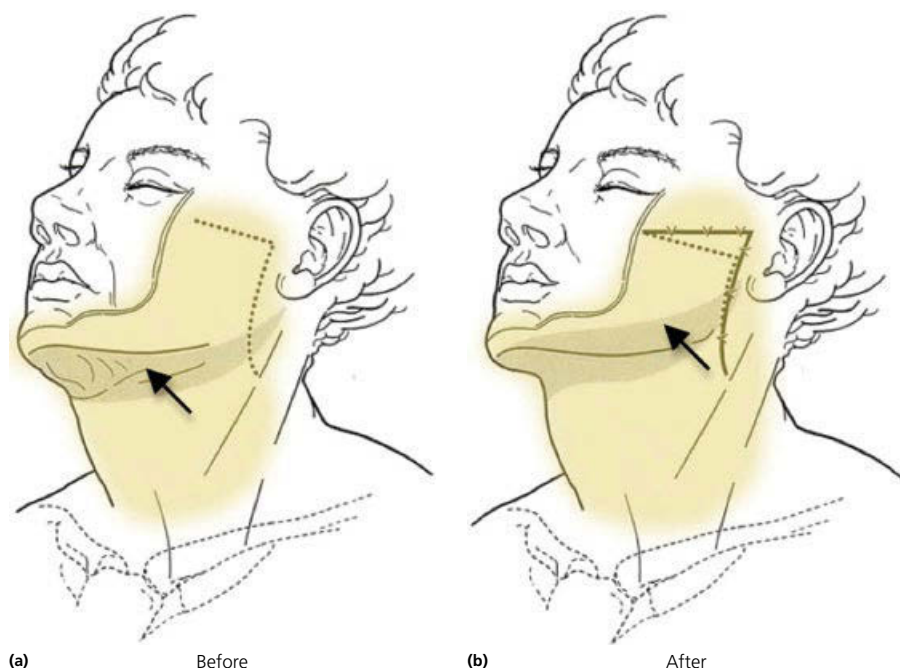


Figure 71.27 Sequencing excision of preplatysmal fat when simultaneous facelift and necklift are performed. If submental liposuction is performed before the SMAS is elevated, denuded areas will be subsequently advanced onto the lower face. This can result in a harsh mandibular contour. To avoid this problem, preplatysmal fat should be removed, if indicated, after the SMAS flap has been raised. (a) Incorrect plan for preplatysmal fat excision (grey shaded area). Submental liposuction is performed at the beginning of the procedure before SMAS flap is raised. (b) After SMAS elevation, areas stripped of fat (grey shaded area) are moved up onto the lower face.

using a 10mm double-pronged skin hook or an Allys forceps. The dissection is subsequently carried laterally, over the anterior belly of the digastric muscle hugging the underside of the platysma. If this dissection is carefully made, a well-defined and relatively avascular plane can usually be identified. Small communicating vessels are not infrequently encountered, however, especially near the medial muscle borders. These should be carefully fulgurated and divided as required. Subplatysmal fat should be left on the deep surface of the neck and *not* raised with the platysma flap. This will facilitate fat removal, as it is technically more difficult to resect fat from the undersurface of the muscle.

The plane tangent to the anterior bellies of the digastric muscles with the neck in neutral or slightly flexed position should be used as the landmark for subplatysmal fat removal. All fat lying superficial to this plane should theoretically be removed if optimal contour is to be obtained. If fat excision is made with the neck flexed, inadvertent overexcision can occur.

In practice, overall neck contour must be considered, as it is the curvilinear plane across the submental region tangent to the borders of the mandible and the anterior bellies of the digastric muscles that ultimately determines attractive neck contour. If large submandibular glands and/or anterior bellies of the digastric muscles are present and it is elected that they should not be treated, subplatysmal fat removal should be more conservative if accentuation of these problems is to be avoided. Fibrous fat in the pre-hyoid region should also not be arbitrarily removed, especially in women, as accentuation and masculinization of the larynx can occur. If prominent submandibular glands and digastric muscles are appropriately treated, however, more aggressive resection of subplatysmal fat can be made.

Subplatysmal fat will be evident as a centrally situated triangular-shaped fat pad with its base lying at the hyoid and tip

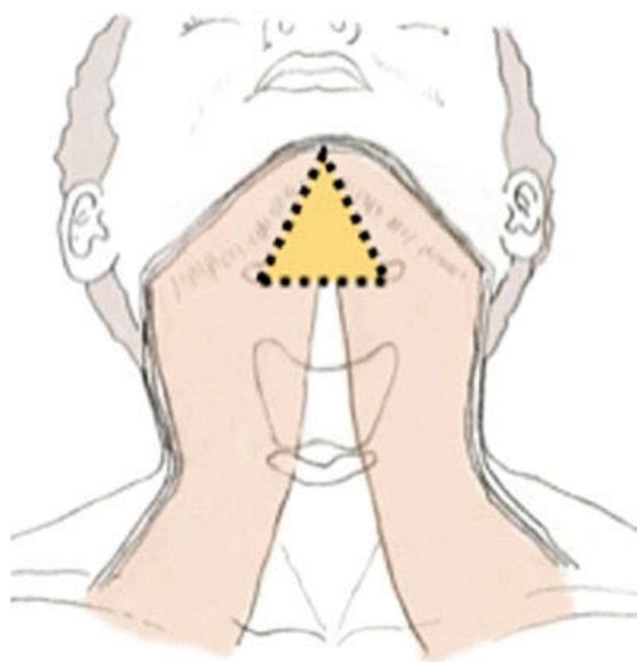
near the mentum (Figure 71.28). It should be removed incrementally, as appropriate, and as determined by the position and size of the digastric muscles and the size of the submandibular glands. Fat removal should not be arbitrary, and close attention must be paid to the new contours created. Subplatysmal fat excision often results in division of tributaries of small pretracheal vessels and a long shielded cautery forceps and good suction should be available to obtain adequate haemostasis when required.

Fat situated deep to the plane tangent to anterior bellies of the digastric muscles and beneath the deep cervical fascia (deep cervical or 'intra-digastric' fat) should not be removed and the overwhelming temptation to 'clean out' the deep cervical space resisted. Platysmaplasty, invaginating platysma in a 'corset' fashion, suturing the anterior bellies of the digastric muscles together, and other similar manoeuvres cannot compensate for overzealous excision of deep fat and, in time, an objectionable submental depression will appear in the necks of patients so treated.

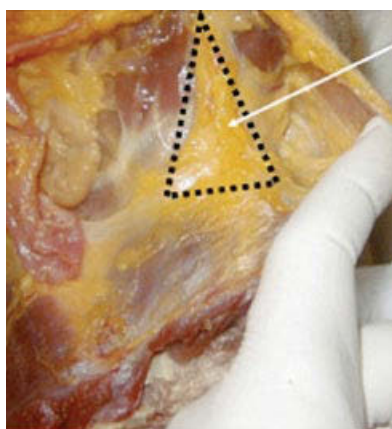
Submandibular gland reduction

If large submandibular glands are present, they can usually be seen and palpated lateral to the ipsilateral belly of the anterior digastric muscle on each side within its respective submandibular triangle, protruding inferiorly to a plane tangent to the digastric belly and the mandibular border. The protruding portion can be incrementally resected through the submental incision before submental platysmaplasty is performed to improve neck contour, if indicated (Figure 71.29).

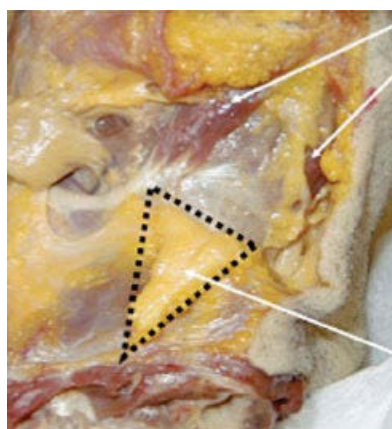
Prominent submandibular glands will be encountered as subplatysmal dissection is carried over the anterior belly of the ipsilateral digastric muscle. The prominent gland will appear as a



(a)



(b)



(c)



(d)

Figure 71.28 Subplatysmal fat. (a) Illustration showing the position of the triangular-shaped subplatysmal fat pad located in the submental space with its tip near the mentum and base at the hyoid bone. (b) Cadaveric view of the submental region and subplatysmal fat similar to that shown in illustration above but closer-up. The arrow is pointing to the subplatysmal fat that has been outlined with a dashed line. The right and left borders of the platysma have been elevated showing the contents of the submental space. The subplatysmal fat pad can be seen overlying the digastric muscles to the upper right and left of the triangle. The hyoid bone at the base of the triangle. (c) Cadaveric demonstration of subplatysmal fat. The head is slightly turned to the viewer's right and the chin is at the upper right hand corner of the photo. The two upper arrows point to the anterior bellies of the right and left digastric muscles and the lower arrow to the subplatysmal fat pad. In this photo, the fat pad has been reflected inferiorly but still attached at its base. The hyoid is now visible along with the interdigastric space. There is a small amount of deep cervical fat visible between the two upper arrows that has been reflected to the right. (d) Intraoperative demonstration of the subplatysmal fat specimen lying over the submental space from which it was removed. The patient's chin is pointing superiorly and is presenting the upper left corner of the photo. The neck is in the lower right corner of the photo.

smooth pink to tan-coloured mass covered by a smooth capsule. Reduction is begun by incising the capsule overlying the gland inferomedially just lateral to the anterior belly of the digastric muscle. The submandibular gland will be evident once exposed in this manner due to its distinctive lobulated appearance. Its inferior portion can be grasped and easily separated from adjacent tissue inside its capsule using a gentle blunt scissors spreading

technique. Although all vital structures are outside the glandular capsule, care should be taken when mobilizing the gland superolaterally, as both the retromandibular vein and the marginal mandibular branch of the facial nerve are in proximity in that area.

An examination of the gland once mobilized inside its capsule will show it to be large, and not merely 'ptotic', and this observation forms the basis of the recommendation that partial resection

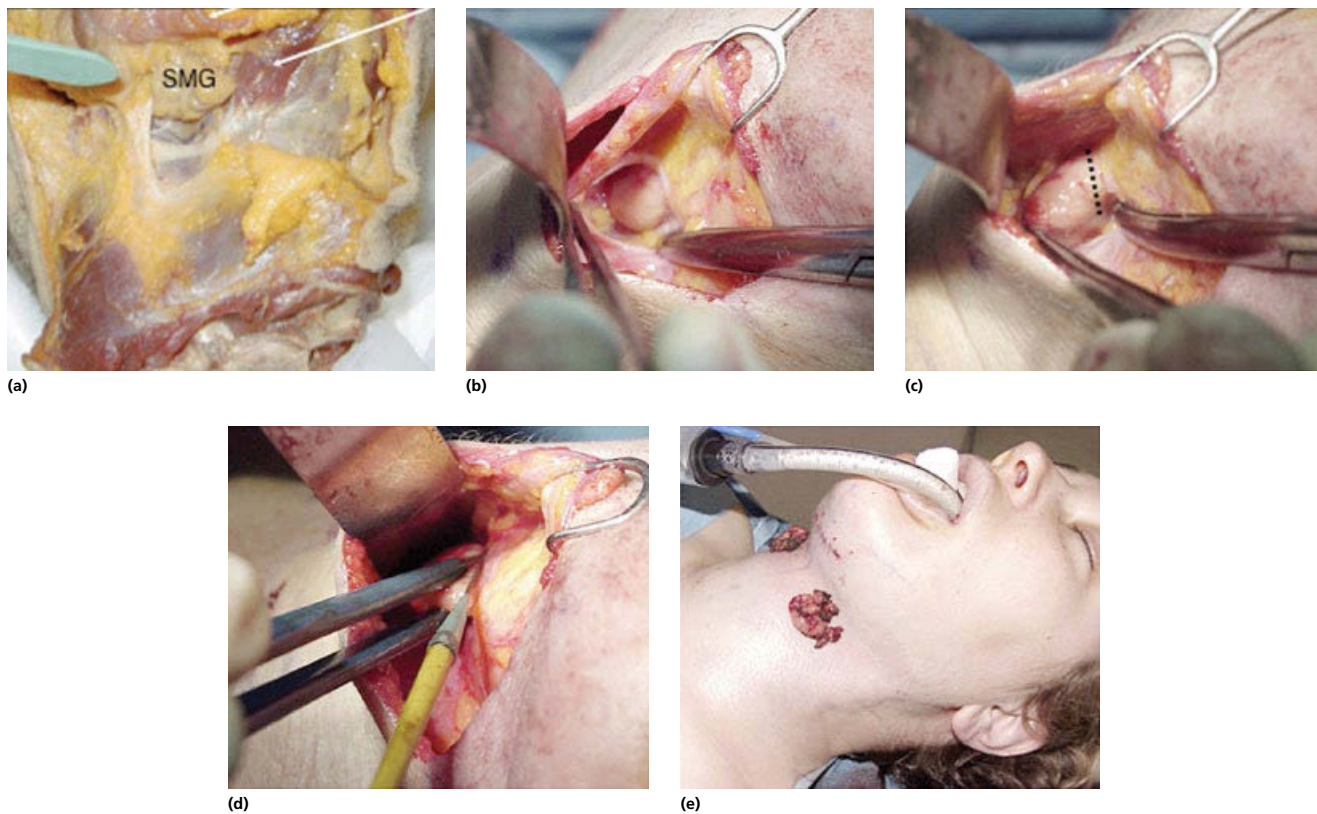


Figure 71.29 Surgical approach to submandibular gland reduction. (a) Cadaveric demonstration of submandibular triangle. Chin is in the upper right corner of the photo. The excessive portion of the submandibular gland (SMG) is found protruding inferiorly to a plane tangent to the digastric (lower arrow) and the mandibular border (small upper arrow). (b) Intraoperative photo demonstrating exposure of the submandibular gland. A submental incision has been made approximately 1 cm posterior to the submental crease and the neck subcutaneously undermined. The right platysma muscle has been elevated and is retracted with a double-pronged skin hook and a malleable retractor. The gland will be seen as a distinct bulge just lateral to the ipsilateral anterior belly of the digastric (scissors tips rest on digastric). The capsule has been incised inferomedially and the submandibular gland isolated using blunt dissection. (c) Intraoperative photo prior to resection of the excess portion of the gland. The gland has been mobilized and gently pulled inferiorly. The dotted line represents the level at which the inferior portion of the gland will be excised. (d) Intraoperative photo just prior to excision of the protruding part of the gland. This should be performed incrementally with electrocautery in such a manner that the portion protruding inferior to the plane tangent to the ipsilateral anterior belly of the digastric muscle and the ipsilateral mandibular border will be resected. (e) Patient seen immediately after neck lift that included partial submandibular gland reduction but no skin resection. The photo shows the excellent contour created without skin tightening. The excised portions of the submandibular glands are also demonstrated on both the right and left sides.

of the protruding portion be performed. Examination of the exposed gland will also clearly demonstrate that attempts to reposition it more superiorly by suture suspension, or by tightening overlying platysma muscle will ultimately be fruitless.

A key step in the safe performance of the procedure is adequate mobilization of the gland, and a modest overmobilization will make both gland resection and the control of any intraglandular bleeding that might be encountered much easier.

Once adequately mobilized inside its capsule, the inferior portion of the gland is grasped, and pulled gently inferiorly and medially out of its fossa and away from adjacent structures. The redundant portion is subsequently excised incrementally under direct vision in a medial-to-lateral direction along the planned line of resection with coagulating current cautery. Excision of the excess part of the gland should be performed in such a manner that the portion protruding inferior to the plane tangent

to the ipsilateral anterior belly of the digastric muscle and the ipsilateral mandibular border will be resected. It is usually best that initial resection is conservative and that the gland is incrementally reduced thereafter as required. Never is it necessary or appropriate to remove an entire gland. This would most likely result in a depression or other contour abnormality, and could precipitate an objectionable depression or 'dry mouth' condition.

Excision of the redundant and protruding portion of the submandibular gland should be performed using an extended flat-tipped cautery and a long, high-quality pair of insulated forceps. A flat-tipped, extended cautery is better for controlling bleeding of an intraglandular vessel, should it be encountered, than a needle-point cautery tip. As in all surgery of the neck and submental region, a fiberoptic headlight or retractor is mandatory, as is a competent assistant and a second scrubbed team member to pass instruments and sutures as needed. One person cannot

safely perform both these roles. A suction cannula or smoke-evacuating retractor should also be placed, and the position of the cautery tip relative to vital adjacent structures must be known at all times during the resection. If a smoke-evacuating retractor is used, a second suction set-up for a Yanchor suction should be available in the event that bleeding is encountered during the resection and suction is needed to obtain haemostasis.

Submandibular gland reduction must not be performed without good light, proper instruments, requisite exposure, adequate suction, sufficient personnel and considered caution, as intraglandular vessels may be encountered. These vessels are more commonly encountered when larger resections are necessary and are most easily managed by immediate and thorough fulguration before further dissection is made. Typically, bleeding will occur from both the gland and the specimen side and this is usually best controlled by quickly moving the cautery back and forth between each end of the cut vessel. If only the gland side is cauterized, bleeding will often continue from the specimen side, obscuring the surgical field. If bleeding is brisk and not easily controlled by simple cauterization as described above, the cut edge of the gland should be compressed with forceps and the vessel isolated. It may then be grasped with a second instrument and cautery applied. In the unlikely event that bleeding cannot be controlled by these means the submandibular fossae should be packed with gauze and digital pressure firmly applied for several minutes. In most cases when the gauze is carefully removed, the bleeding will be seen to have stopped and a search for the bleeding point can be made and any suspicious areas cauterized. The submandibular artery enters the submandibular gland superolaterally and will not be encountered when performing typical resections made for aesthetic purposes. Dissection should always be made carefully in this area, however, and as one becomes more practised in the procedure and more comprehensive resections as a result are made, this vessel will often be seen. In most cases, if care is exercised, resection of the most superolateral portion of the protruding portion of the gland can be made without injuring it or the need to ligate it. Should the vessel be inadvertently injured suction should be applied and it should be grasped with a high-quality DeBakey-type forceps. It can then be grasped with a right-angle or similar forceps and ligated.

Once excision of the protruding portion of the gland is complete, the submental region is irrigated and a check for haemostasis along the gland's cut edge is made. It is not necessary or productive to attempt to oversee the cut edge of the gland, or to close the glandular capsule. These manoeuvres are not only technically difficult, but could result in injury to nearby neurovascular structures. It is also not necessary or productive to overcauterize the cut edge of the gland in an attempt to shrink or seal it. This typically results in more pain and swelling, and prolonged submental induration. Subtotal submandibular gland excision to date has not resulted in any cases of haematoma, airway compromise, salivary fistula or gustatory sweating in our practices.

Experience has shown that when submandibular gland reduction is performed, a drain should be placed in the subplatysmal space for at least several days after the procedure to reduce oedema and induration in the submental area and to speed the patient's overall recovery. It is safest to perform the platysmaplasty before drain placement, and then to thread the drain into place. If the drain is placed before platysmaplasty is performed there is a chance that the drain could be caught in one of the sutures, making its removal problematic.

Submandibular gland reduction, although important in the rejuvenation of many necks, will not in and of itself result in attractive neck contour. Other problems must be identified and addressed if optimal results are to be obtained.

Submandibular reduction should be regarded as an advanced technique. It should be undertaken by surgeons well versed in cervical anatomy and experienced in more basic manoeuvres used to rejuvenate the neck. It is highly recommended that any surgeon uncertain of the anatomy of the submental triangle or the exact relationships of important structures in this area review them carefully before undertaking this dissection.

Partial digastric myectomy

After subplatysmal lipectomy and submandibular gland reduction have been performed, assessment should be made of the anterior bellies of the digastric muscles and whether partial digastric myectomy would produce further improvement in neck contour. A subset of patients will be encountered who will be identified as having objectionably large anterior digastric muscle bellies. Usually these are most evident after subplatysmal lipectomy and submandibular gland reduction has been performed, but they may sometimes be evident preoperatively in patients seeking secondary, and occasionally, primary surgery. In these situations additional improvement in neck contour may be obtained by performing superficial, subtotal anterior digastric myectomy.

Superficial, subtotal anterior digastric myectomy is performed under direct vision, through the submental incision after the subplatysmal space has been opened, the platysma muscle mobilized, and subplatysmal fat and protruding portions of the submandibular gland resected, if indicated. The redundant portion of muscle can be excised by either a tangential strip excision technique or by a partial division and excision technique. Although both are reliable and effective, in most cases tangential strip excision provides the simplest and most intuitive and straightforward means of removing the protruding portion of the muscle belly.

Tangential strip excision is performed by visually gauging the muscle redundancy, grasping the redundant portion with a DeBakey or similar forceps, and tangentially excising a strip of muscle longitudinally from the muscle belly with Metzenbaum or similar scissors. Excision is begun near the muscle origin at the mentum and continued to the muscle belly insertion at the lateral hyoid. The muscle is then reassessed and the action repeated until the protruding portion is fully removed and optimal contour has been established. Typically, tangential excision will result in modest bleeding from the excised area

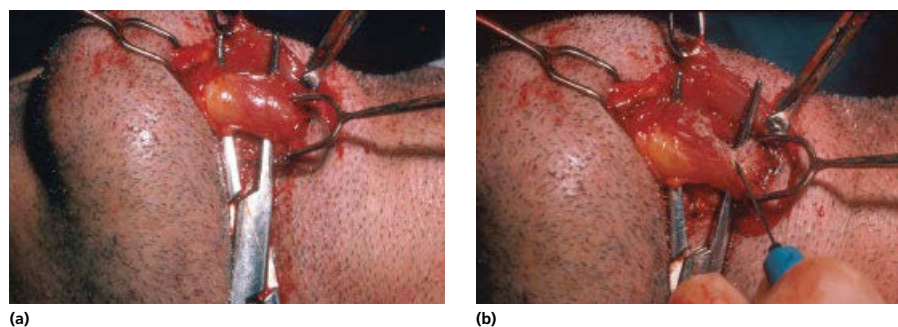


Figure 71.30 Partial division and excision technique of digastric muscle reduction. (a) Excess muscle present is gauged and isolated on a tonsil forceps by pushing the tips of the instrument through the mid-muscle belly at the level of optimal contour and spreading to split the muscle along the length of its fibres. View from patient's right of right digastric muscle through submental incision. Patient's chin is at the left upper corner of the photo and the neck is on right. (b) The isolated muscle segment resting on the instrument consisting of the superficial, protruding portion of it is then excised with scissors or cautery by dividing it near the mandible and the hyoid. The neck is re-examined and the manoeuvre repeated again if necessary until an improved contour is obtained.

that is easily controlled with cautery once muscle reduction is complete. Alternatively, muscle can be excised in this manner directly with a cautery instrument.

In the partial division and excision technique of digastric muscle reduction the excess muscle present is gauged and isolated on a tonsil forceps by pushing the tips of the instrument through the mid-muscle belly at the level of optimal contour and spreading to split the muscle along the length of its fibres. The isolated muscle segment resting on the instrument, consisting of the superficial, protruding portion, is then excised with scissors or cautery by dividing it near the mandible and the hyoid (Figure 71.30). The neck is re-examined and the manoeuvre repeated if necessary until an improved contour is obtained. Usually this entails the excision of the superficial-most half or less of each muscle.

It should be noted that the myectomy, regardless of how it is performed, is a partial and subtotal reduction of the protruding portion of the digastric muscle only, and not an arbitrary excision of the entire muscle belly. In some cases a near total resection will be indicated and can be performed. In addition, partial, superficial digastric myectomy, although an important manoeuvre in the rejuvenation of many necks, will not in and of itself result in attractive neck contour. Other problems must be identified and treated if optimal results are to be obtained. Finally, it should be noted that partial superficial digastric myectomy, although straightforward and conceptually simple, should be regarded as an advanced technique. It should be undertaken by surgeons familiar with cervical anatomy and experienced in more basic manoeuvres used to rejuvenate the neck.

Anterior platysmaplasty

Anterior platysmaplasty (see Figure 71.16 and 71.31) is commonly performed and provides an improved result in most necks. The purpose of platysmaplasty is to consolidate the neck, to reduce irregularities due to horizontal platysmal laxity and to optimize neck appearance when it is flexed and the patient is looking down. Contrary to popular opinion, it cannot create a

sustained improvement in neck contour in and of itself if deep layer problems (subplatysmal fat excess or submandibular gland and digastric muscle hypertrophy) are present, and it is *not* adequate treatment for platysmal hyperfunction and dynamic platysmal bands in most patients.

Typically, platysmaplasty consists of suturing the medial muscle borders of the platysma muscles together from mentum to thyroid cartilage in one or two layers. If redundant muscle is present medially, it is excised and discarded before suturing is performed so that a smooth, edge-to-edge approximation of the medial muscle borders can be made. Experience has shown that this produces a better and more consistent outcome than when excess muscle is invaginated and a multilayer 'plication' or 'corset' is performed, and reduces the likelihood that objectionable midline fullness or bands will result after healing and relaxation of tissues has occurred.

When performing a facelift and neck lift together, platysmaplasty should be performed after cheek SMAS flap dissection and suspension to prevent an accentuation of the effects of ageing and gravity and compromised improvement on the face. Although raising and suspending cheek SMAS flaps first makes platysmaplasty suture placement more difficult, it allows optimal repositioning of the cheek and jaw, and provides for the best overall improvement. If platysmaplasty is performed first, the cheeks and jaw will be pulled inferiorly towards and into the neck, and the effectiveness of the SMAS flap will be significantly diminished.

Platysmaplasty is usually performed using multiple simple interrupted sutures of 3–0 braided polyester (or suture of choice) on a medium to large tapered needle. Long instruments are needed and patience will be required. Optimal improvement generally cannot be obtained if repair is made in one layer or if a running suture is initially used. If the first layer is performed using a running suture some gathering and a 'purse string' effect can occur. This will result in shortening along the line of repair and can cause bowstringing and postoperative midline band formation. If the initial approximation is made using interrupted sutures this problem is averted.

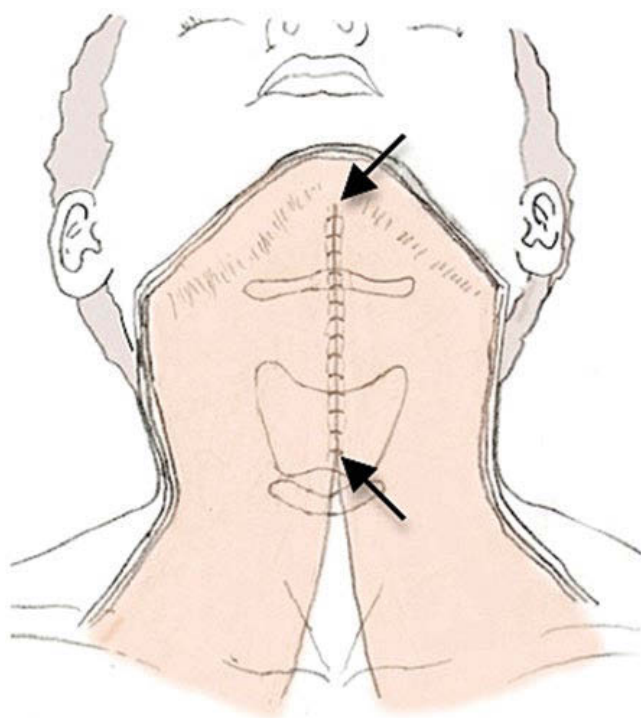


Figure 71.31 Anterior platysmaplasty. repair of platysmal diastasis from mentum to thyroid cartilage (arrows) improves submental support and helps consolidate the neck. It is not adequate or effective treatment of platysmal bands however.

Once initial approximation has been made with interrupted sutures, the line of repair can be oversewn and reinforced with a simple running or running inverting suture without a purse string and shortening effect. The repair need only be snug and should not be tight. If a permanent suture is used, care should be taken to saturate each suture with antibiotic solution prior to placement and to bury all knots.

Platysmaplasty should be performed with the neck in a neutral position. If the closure is made with the neck extended, excessive tightness may result when the patient looks down after surgery. This is particularly the case if platysmaplasty is performed in conjunction with a postauricular transposition flap of cheek SMAS.

If a neck lift is performed in conjunction with a facelift and postauricular transposition flaps are planned, suspension of the flaps to the mastoid should be performed after anterior platysmaplasty has been performed. If the transposition flaps are suspended first, the platysma muscles can be shifted laterally and it may be difficult to join them in the midline. Suspension to the mastoid is performed with interrupted sutures of 4-0 polyglactin (Vicryl) (or other suture of choice). The more proximal portions of the flap are secured with a simple running suture of the same material. This consolidates the deep layer repair in the peribulbar area and prevents the flap from bunching up or rolling up on itself.

Transverse platysma myotomy

Although anterior platysmaplasty will often result in an attractive neck at rest and on the operating table, objectionable appearing dynamic platysma bands and muscle striations will often still be evident in conversation and during animation after surgery if additional steps are not taken. These problems can be minimized by performing *transverse platysma myotomy* (see Figures 71.18, 71.19 and 71.20). The extent of platysma trans-section will depend upon the deformities present and thus will vary from patient to patient.

Platysma myotomy should be performed after anterior platysmaplasty has been completed as the platysma muscle will be uniformly distributed over the anterior neck and under slight tension. Myotomy should be performed low in the neck at the level of the cricoid cartilage. At this level the muscle is thin and will bleed less, and the cut edges are less likely to be visible postoperatively. In addition, a smooth transition across the cervicomenal angle is maintained and lower lip dysfunction is avoided. If trans-section is made higher, these benefits are lost. This is particularly true when myotomy is performed at the level of the hyoid along the cervicomenal angle or when wedges of platysma muscle are removed at that location, as is shown in many plastic surgery textbooks. The platysma muscle is much thicker at that level, the cut edges are more likely to bleed and/or be visible after surgery. In addition, a high trans-section or resection of this sort can result in a severe and unaesthetic transition for the submental region to the neck, and adversely effect platysma action and result in asymmetrical movement of the lower lip during speaking and expression.

Anterior platysma myotomy is best begun through the submental incision just inferior to the inferior-most suture placed when platysmaplasty was performed. The medial platysmal border is identified over the cricothyroid area and grasped and lifted away from the deep cervical fascia with a long DeBakey-type forceps. Myotomy is then made by nibbling through the muscle in small increments with Metzenbaum scissors. As the muscle is divided it usually separates a centimetre or more, exposing the fascia beneath it. Serrated and 'super-sharp' scissors should not be used as they tend to pick up and cut adjacent tissue, and can result in injury to the anterior jugular vein. Unintended injury to the anterior jugular vein will result in annoying bleeding that can be difficult to control. Gentle spreading with scissors tips before each cut is made helps separate the platysma from underlying cervical fascia and facilitates dissection. Myotomy is continued laterally and slightly superiorly according to the preoperative plan. If a 'full-width' division of the platysma is planned, the lateral-most portion of the trans-section is completed through the postauricular incisions by identifying the lateral muscle border, incising it and incrementally extending the incision until it is brought into continuity with the incision made in the muscle anteriorly through the submental incision.

Final contouring of cervicofacial fat

Once correction of deep layer neck problems has been made and platysmaplasty, platysma myotomy and suspension of postauricular transposition flaps have been performed as indicated, final contouring of subcutaneous superficial cervicofacial fat should be performed. This can be done under direct vision using Metzenbaum scissors or a small suction cannula. Final fat sculpting should be continued until all contours are smooth and regular, but it is essential that not all fat is removed if an attractive appearance is to be obtained. In addition, if a 'double chin' or 'witch's chin' is present, an extended dissection of the perimental area and chin pad should be made and fat contributing to these problems excised. Mental and submental fat should then be sculpted and blended to ensure a smooth transition for the chin to the submental area. In full necks, pre-platysmal lipectomy can be performed with a suction cannula using an 'open' liposuction technique by placing the cannula hole down under the skin flap and holding the skin flap down as resection is made. In thin necks, simple excision is made using a scissors technique.

Drain placement

Drains are used routinely and experience has shown that they reduce postoperative ecchymosis and seromas, and allow patients to return to their work and social lives sooner. As a practical matter, drains are generally necessary when aggressive deep neck manoeuvres (subplatysmal fat excision and submandibular gland reduction) are performed, and 'tissue glues' advocated by some surgeons to consolidate the subcutaneous plane cannot be expected to prevent collections in the subplatysmal area. It should be acknowledged that drains will not substitute for poor haemostasis in either plane, however, and will not prevent haematoma formation.

One or two 10 F round 'end-perforated' Jackson-Pratt style closed-circuit suction drains are typically placed subcutaneously in the neck when neck lift is performed. Each drain is placed through a small stab incision on the occipital scalp (in a facelift or extended neck lift) or postauricular sulcus (in a short-scar neck lift) and is anchored at its exit site with a 4–0 nylon suture. Drain placement will be easy in extended neck lifts or when a facelift is concurrently performed as a postauricular incision will be present. In short-scar neck lifts, in which a submental incision only is present, a small liposuction cannula can be used to tunnel from the postauricular stab incision to the neck, and then used to pass the drain back out the incision in a retrograde fashion. The perforated portion of the drain is then positioned beneath the cervical skin across the anterior neck to the opposite side. When extensive modifications have been made to the anterior cervical and submental regions, or when diffuse oozing is encountered, one drain is placed subcutaneously on each side. This provides overlapping drainage of the anterior cervical area. Two drains are generally routinely placed in most male patients as well.

If submandibular gland resection is performed and/or a large amount of subplatysmal fat is removed, an additional drain is placed and its perforated portion is placed in the subplatysmal space after platysmaplasty has been performed. This is most easily accomplished working through the submental incision and by placing a long tonsil or right-angle forceps under the medial edge of the right or left platysma muscle at the inferior-most extent of the platysmaplasty suture line, advancing it laterally and slightly superiorly under the muscle, and pushing the tip of the forceps through the muscle into the subcutaneous space. The tip of the drain is then grasped, pulled retrograde under the muscle and back to the midline, and then cut to length as indicated and tucked underneath the muscle on the opposite side. It is not recommended that a drain be placed in the subplatysmal space before platysmaplasty is performed. When this is done there is a small but significant chance that it could be inadvertently sutured in place and rendered difficult to remove.

Skin closure

If a short-scar neck lift has been performed, a submental incision only will be present and this can be conveniently closed in two layers. Prior to closure it is important to confirm that the wound edge thickness and amount of subcutaneous fat present on each side of the incision are similar. Typically, the wound edge of the cervical side will have a thinner edge and have less subcutaneous fat due to contact with instruments and retractors during preceding steps in the procedure, and the mental, superior side will be thicker and have more fat. If a discrepancy is present, as it often will be, fat is carefully removed from the thicker superior side. The first layer of closure consists of several subcutaneous sutures of 5–0 Monocryl (or other suture of choice) to ensure that fat is present behind the closed wound and so that a depressed scar is avoided. Final approximation is then made with simple interrupted sutures of 6–0 nylon (or other suture of choice).

If an extended neck lift is performed, or a facelift is performed in conjunction with a neck lift procedure, skin will be excised from the postauricular area. The postauricular flap is shifted posteriorly and somewhat superiorly, roughly parallel to transverse neck creases and the mandibular border, in such a manner that skin is suspended under minimal or no tension and that little or no skin need be trimmed from the anterior (postauricular) flap border. Excessive trimming of skin from the anterior border of the postauricular skin flap will require the flap to be shifted along an incorrect superiorly directed vector, and this will produce a compromised result. The first point of suspension is located in the postauricular area at the anterior–superior aspect of the occipito-mastoid incision. The flap is secured at this point with a simple interrupted suture of 4–0 nylon. No incision into the flap is necessary and no deep suture is used.

Once this suspension suture has been placed, the flap overlying the inferior portion of the ear should be carefully divided and the lobule exteriorized. This is a key step in the procedure

that must be performed incrementally and with great care if a visible scar, lobular mal-position, and objectionable 'pixy ear-lobe' is to be avoided. If the incision to exteriorize the lobule is properly made, the apex of the incision should rest snugly against the inferior-most portion of the conchal cartilage. If the incision into the cheek flap is made too far anteriorly or inferiorly, however, artistically appropriate resetting of the lobule will not be possible, and the outcome of the overall procedure significantly compromised.

Postauricular closure along the auriculomastoid sulcus is performed next, and should be completed before trimming and closure of the occipital portion of the incision and resetting of the lobule. It is begun by conservatively trimming the anterior border of the postauricular skin flap into a soft curve to match the curve of the incision made in the auriculomastoid sulcus. The incision is then closed with several interrupted sutures of 4-0 nylon. No deep suture is necessary or used.

Little if any skin should be trimmed from the anterior margin of the postauricular flap along the auriculo-mastoid sulcus. If it appears that a large amount of skin needs to be excised from this area, the postauricular flap has been shifted incorrectly both too far anteriorly and superiorly. In such instances, the previously placed suspension sutures should be removed and the flap re-advanced along a more appropriate, and more posteriorly directed vector.

It is also a significant error and conceptually flawed concept to excise any skin over (superior to) the apex of the occipitomastoid incision and to shorten the postauricular flap along the long axis of the sternocleidomastoid muscle as commonly practised and shown in many plastic surgery textbooks. Despite an apparent redundancy in this area when the patient is supine on the operating table, there is never any true skin excess at this location. This pseudo-excess of skin is present only because of the patient's high shoulder position in the supine position. It will vanish when she or he sits up and the shoulders drop to a normal position. Skin along the axis of the sternocleidomastoid muscle is also needed for side-to-side head tilt. Inappropriate excision of skin over the apex of the occipito-mastoid defect is the ultimate underlying cause of hypertrophic healing in the postauricular region and of a wide postauricular scar.

Trimming and closure of the occipital portion of the incision should be performed after closure of the postauricular area and suspension without trimming along the transmastoid portion of the postauricular incision. A facelift marker should be used to gauge skin flap redundancy along the occipital incision, and all points along the flap should be intentionally trimmed with 2-3 mm of redundancy. It must be remembered that the goal of skin excision is to remove redundancy, and not to tighten the cervical skin flap. If trimming is performed correctly, wound edges should abut one another, and no gaps should be present, *before* sutures are placed. The incision is then closed in one layer with multiple half-buried vertical mattress sutures of 4-0 nylon, with the knots tied on the scalp side and simple interrupted sutures of 6-0 nylon. No deep sutures are required and staples

should not be used. This plan will provide precise wound edge alignment and prevent cross-hatched scars (suture marks). In addition, if the incision is closed under no tension as described an inconspicuous scar will be obtained.

If the postauricular skin flap has been shifted along a proper vector, and tissue distributed appropriately under no tension over the occipital area, a precise fit should be obtained. If neck skin redundancy is large, or if the postauricular flap has been suspended under tension, a 'dog-ear' will often be present at the inferior-most portion of the occipital incision. This should not be chased down the occipital hairline as an obvious scar will result in the fine hair on the nape of the neck. A better result is obtained under these circumstances if the dog-ear is inset posteriorly into the occipital scalp, above the junction of thick and fine hair, and secured with simple interrupted sutures of 4-0 nylon. This requires that a small ellipse of *scalp* be excised from the *scalp side of the incision*, but moves the end of the incision into dense hair where it is well concealed.

Dressings

After all planned procedures have been completed and all incisions have been closed, the patient's hair is washed with shampoo and conditioner and if long it is placed in a soft braid. The hair is then allowed to dry or is blown dry in the recovery room. A final inspection of sutured incisions is made. If poor alignment is found in any area, it is locally re-prepped and sutures are removed and replaced as required.

No dressing is required or applied. Patients are typically discharged with a hat, scarf and sunglasses following the procedure. The traditional facelift and neck lift dressings consisting of tightly applied mineral oil-soaked cotton and rolled gauze are unnecessary if closed suction drains are used, and a strong argument can be made that they are counterproductive and of potential harm to the patient. These dressings place unnecessary and potentially harmful pressure on thin skin flaps in a critical stage of their healing and can compromise already tenuous circulation in them. In addition, they preclude inspection and monitoring of the operative site and can disguise and delay the diagnosis of serious problems such as haematoma, flap ischaemia and pressure necrosis. Finally, patients typically find the traditional face and neck lift dressing cumbersome, confining, unhygienic and claustrophobic, and their family members are often frightened by them.

Postoperative care

Performance of the procedure itself only fulfills part of our obligation to the patient and the care they receive postoperatively is arguably as important as the surgery itself. Diligent postoperative care will also ensure the best result and limit the likelihood that problems and complications will occur.

Patients are discharged with specific written instructions as to how they are to care for themselves, and the surgeon should

always be available to answer questions and see any patient, if needed.

Sleeping and head position

Patients are instructed to sleep flat on their backs without a pillow. A small cylindrical neck roll is permitted if the patient requests or requires it. This posture assures an open cervicomental angle and averts dangerous folding of the neck skin flap and obstruction of regional cervical lymphatics that inevitably occurs if the patient is allowed to 'elevate their head on pillows' as is commonly recommended after head and neck surgery. If patients are allowed to elevate their head on pillows, their neck will inevitably end up flexed and their skin bunched and folded. In addition, a flat-in-bed position encourages swelling to drain posteriorly to the back of the head instead of inferiorly to the anterior neck, where it is not harmful, less noticeable and more rapidly transmitted away from the head and neck area when the patient sits upright. This is particularly helpful if a facelift has been performed as part of the procedure.

Patients are shown an 'elbows on knees' position that ensures an open cervicomental angle while sitting. This posture places the patient's book, magazine, paperwork, notebook computer or meal in a position that allows reading, writing, eating or TV watching to be performed comfortably and safely with their chin up and their neck skin smoothly and uniformly distributed over their neck. If patients are allowed to sit upright during these activities their neck will inevitably end up flexed and their skin bunched and folded.

Cool compresses

Patients are asked to rest quietly and apply cold compresses to their face and eyes for 15–20 min of every hour they are awake for the first 3 days after surgery. For most patients, oedema peaks at about this time. It is not necessary or productive to apply ice compresses continually throughout the day or at night.

Medications

All patients are provided oral (usually non-narcotic) analgesics, sleeping pills, oral dissolving antiemetic tablets, preservative-free ophthalmic ointment and preservative-free 'artificial tears' solution with instructions for their use. Patients are required to use ophthalmic ointment each night for the first three weeks after surgery or until all signs of eye irritation are gone. Artificial tears are used throughout the day on an as-needed basis.

After-surgery diet

Patients are allowed to take their usual diet as tolerated after surgery but are encouraged to avoid sweet, salty, sour, dry and difficult-to-chew foods for several weeks. It is particularly important to avoid these sorts of foods, which stimulate salivation, for 7–10 days if submandibular gland reduction has been performed. Patients are asked to abstain from the intake of alcohol for two weeks after surgery and until they are no longer taking pain killers or sleeping medication.

Wound care

Patients are instructed to begin a daily routine of showering and shampooing no later than 3 days after surgery, even if their drains are still in place. This helps remove crusting about the suture lines, keeps incisions clean and usually improves the patient's general outlook and wellbeing. Patients are also informed that they need not be as thorough as usual when washing their hair and assured that shower water, shampoo and conditioners are not harmful and will not interfere with healing or cause infection.

Hair care

Patients are allowed to use their usual hair-care products but must be warned that their scalp may be partially numb after surgery and that they must be careful to ensure that shower water is not too hot and that hair dryers are not used on high heat settings. Patients are also instructed not to 'perm', tint, dye, highlight, colour or otherwise chemically treat their hair for two weeks after surgery to limit the chance of hair breakage or hair loss. 'Hot curlers' and 'curling irons' must be used with care for several months following forehead lift surgery as patients might unknowingly burn their foreheads and scalps when doing so. Patients are advised to apply hair gel, hair cream, hair spray and similar products sparsely, or preferably not at all, until all sutures have been removed.

Drain removal

At least one drain is usually left in the neck until the first suture removal visit. This is because drain output often quickly falls on the first or second day after surgery during the time the patient is mostly supine and resting, but then typically picks up again when the patient begins to spend more time upright and starts to move his or her head about more. Leaving the neck drain in longer, for 4–5 days, will reduce the likelihood that small collections will form and it will speed the overall resolution of oedema, ecchymosis and induration in the neck area.

Suture removal

When sutures are removed will vary depending on the type of procedure performed. If a short-scar neck lift has been performed, a submental incision only will be present and sutures are removed on the 4th or 5th day. If an extended neck lift or facelift and neck lift have been performed, 6–0 sutures are removed on the 4th or 5th day and half-buried vertical mattress sutures of 4–0 nylon with the knots tied on the scalp side are removed on the 7th to 9th day. If sutures are left in longer, tell tale and objectionable 'suture marks' are likely to occur.

Return to work after surgery

How long it is before patients return to work and their social lives depends upon their tolerance for surgery, their capacity for healing, the type of work they do, the activities they enjoy and how they feel overall about their appearance. Patients are asked to set aside 7–10 days to recover, depending on the extent of their surgery, and additional time off is recommended if a facelift and related procedures are simultaneously



Figure 71.32 Before and after surgery views of a woman aged 65 who has had prior upper and lower eye lifts by an unknown surgeon. *Before:* Note lax skin, obtuse cervicosubmental contour, paucity of subcutaneous neck fat and 'double chin' appearance when the patient looks down. A large submandibular salivary gland is clearly visible in the lateral submental triangle in the lateral view, and the patient appears to have a 'low hyoid'. *After:* The same patient 13 months after facelift, neck lift, forehead lift, upper and lower eyelifts, perioral dermabrasion and fat transfer to cheeks and lips. The neck lift procedure included excision of excess subplatysmal fat, submandibular salivary gland reduction, superficial digastric myectomy, anterior platysmaplasty and full-width platysma myotomy. Note well-defined jaw line and attractive, youthful-appearing neckline, even when the patient looks down. Comprehensive neck surgery has corrected problems that are commonly misinterpreted as a low hyoid position. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.



Figure 71.33 Before and after surgery views of a woman aged 46 who has had no prior surgery. *Before:* Note full neck, obtuse cervicosubmental contour, and 'double chin' appearance when the patient looks down. Palpation of the lateral submental triangle revealed a firm mobile mass consistent with an enlarged submandibular salivary gland. Her preoperative appearance suggest the presence of a 'low hyoid'. *After:* The same patient 11 months after facelift, neck lift, forehead lift, lower eyelifts and facial fat injections. The neck lift procedure included excision of subplatysmal fat, submandibular salivary gland reduction, superficial digastric myectomy, anterior platysmaplasty and full-width platysmal myotomy. Note attractive, youthful-appearing neckline, even when the patient looks down. Comprehensive neck surgery has corrected problems that are commonly misinterpreted as a low hyoid position. The double chin deformity has also been corrected. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

performed. If the patient is doing well and not experiencing problems, she or he is allowed to return to light office work and casual social activities at that time. It is often wise to begin with a limited workday at first and to adjust schedules thereafter. If a patient's job entails more strenuous activity or physical labour, a longer period of convalescence may be required. Patients are advised not to drive for the first 10 days

after surgery and until they are feeling well, their vision is clear and they are off pain medications.

After surgery activity

Patients are advised to avoid all strenuous activity during the first two weeks after surgery. Two weeks after surgery, patients are allowed to begin light exercise and gradually work up to



Figure 71.34 Before and after surgery views of a woman aged 55 who has had prior eye lifts and a perioral chemical peel performed by an unknown surgeon. *Before*: Note full neck, obtuse cervicosubmental contour and 'double chin' appearance that is exacerbated when the patient looks down. Palpation of the lateral submental triangle revealed a firm mobile mass consistent with an enlarged submandibular salivary gland. Note paucity of subcutaneous neck fat suggesting submental liposuction had been performed at her previous procedure. *After*: Same patient 1 year and 4 months after facelift, neck lift, forehead lift, upper and lower eye lifts, perioral dermabrasion and facial fat injections. The neck lift procedure included excision of a large amount of subplatysmal fat, submandibular salivary gland reduction, superficial digastric myectomy, anterior platysmaplasty and full-width platysma myotomy. Note well-defined jaw line and attractive, youthful appearing neckline even when the patient looks down. The double chin deformity has been corrected by releasing the submental retaining ligaments and blending the fat of the chin and the submental area. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

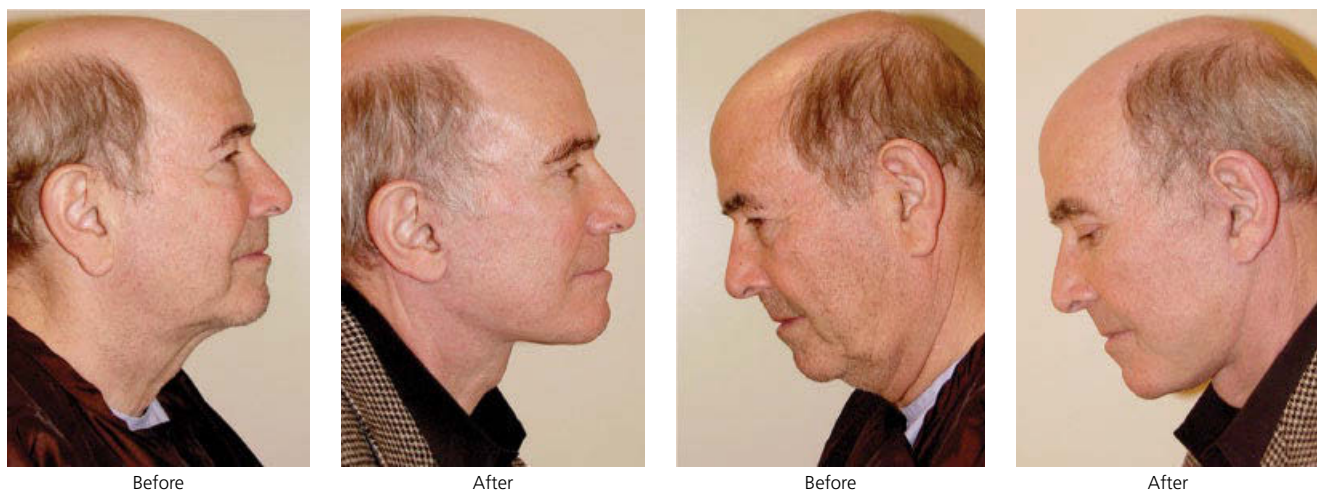


Figure 71.35 Before and after surgery views of a woman aged 68 who has had no prior plastic surgery. *Before*: Note poor cervicosubmental contour and 'double chin' appearance when the patient looks down. An enlarged submandibular salivary gland can be seen beneath the skin in the lateral view and confirmed on palpation. Note paucity of subcutaneous neck fat that is typical for patients in older age groups. Platysma bands are visible in the anterior neck. *After*: Same patient 1 year and 9 months after facelift, neck lift, forehead lift, upper and lower eye lifts, partial facial fat transfer and ear lobe reduction. The neck lift procedure included excision of amount of subplatysmal fat, submanbibular salivary gland reduction, superficial digastric myectomy, anterior platysmaplasty and full-width platysma myotomy. Note well-defined jaw line and masculine-appearing neckline even when the patient looks down. Surgical procedures performed by Timothy J. Marten, MD, FACS. Source: Photos courtesy of the Marten Clinic of Plastic Surgery.

their presurgical level of activity. Four to six weeks after surgery, they are allowed to engage in more vigorous activities, including most sports, as tolerated.

Case examples

Figures 71.32, 71.33, 71.34 and 71.35 show patients before and after procedures.

Further reading

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CHAPTER 72

Rhinoplasty

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Introduction

The nose is a complex three-dimensional structure with critical structural and functional roles, which, by virtue of its position, serves as the central component of the face. Its relationship to surrounding structures is in part responsible for a harmonious, pleasing visage as a whole. Functionally, the nose provides an airway and acts to warm, filter and humidify air passing through it. Noses that deviate from ideal structural proportions – whether subtle variations of normal or more dramatic posttraumatic or neoplastic deformities – have motivated rhinoplasty surgeons since ancient India 800 B.C.E.

Anatomy

A thorough appreciation and understanding of relevant anatomy is necessary for all surgery. Obviously, recognition of the nuances and subtle variations of nasal anatomy is absolutely vital for execution of a successful rhinoplasty.

Skin

A thorough rhinoplasty evaluation must assess skin type and thickness. The surgeon must anticipate the advantages and limitations afforded by a particular skin type. In general, thin skin is unlikely to camouflage irregularities in the underlying cartilaginous and osseous structures, particularly with grafting. In contrast, thick skin conceals imperfections in the nasal skeleton and may conceal surgical changes performed on underlying cartilage and bone.¹ Sebaceous skin can give the tip a bulbous appearance. Despite individual variability in relative skin thickness, general patterns remain consistent across different skin types and ethnicities. The skin is thickest at the nasion. More caudally, the remaining upper half of the nose tends to have skin that is thinner and more mobile than lower half. In the tip area, there tend to be more sebaceous glands, but in the infratip region, the columella and ala again are usually covered with thinner skin.^{2,3}

Cartilaginous and bony support

An understanding of the bony and cartilaginous structural components of the nose should be complemented by correlation with landmarks of the surface anatomy (Figure 72.1). Caudal to the glabellar protrusion, the highest point of the surface of the nose is referred to as the nasion. The root of the nose, just caudal to the nasofrontal suture line, is referred to as the radix, minor adjustments of which can change the overall character of the nose. The rhinion describes the position along the dorsum where the nasal bones articulate with the upper lateral cartilages and the pronasale refers to the region extending from the supratip break, including the tip and infratip lobule, to the anterior-most aspect of the columella. The tip-defining points are the anterior-most points of the nose corresponding to the intermediate crura of the lower lateral cartilages.

Nasal bones

The bony vault is pyramidal in shape and comprises the paired nasal bones that articulate with the frontal bone superiorly and the ascending maxillary processes laterally. The bony pyramid is narrowest at the level of the intercanthal line and widens inferiorly.² The nasal bones are thickest cephalically and become thinner caudally.^{2,4} The nasal bones are supported superiorly at the nasofrontal suture line, laterally at their articulation with the ascending process of the maxilla, and dorsally at their fusion with the perpendicular plate of the ethmoid. The junction between the upper third and lower two thirds of the nose where the cartilaginous septum meets the nasal bones is referred to as the 'keystone area', as this area is essential for structural support, and stabilizing the nasal dorsum for height and projection.³

Upper lateral cartilages

The upper lateral cartilages articulate with the nasal bones and extend cephalically deep to the nasal bones. Beneath the nasal bones, they are fused as one unit. The upper lateral cartilages are supported primarily by their connection with the nasal bones: they do not rest on or articulate with the pyriform process, but rather gain their support as they cantilever off of their

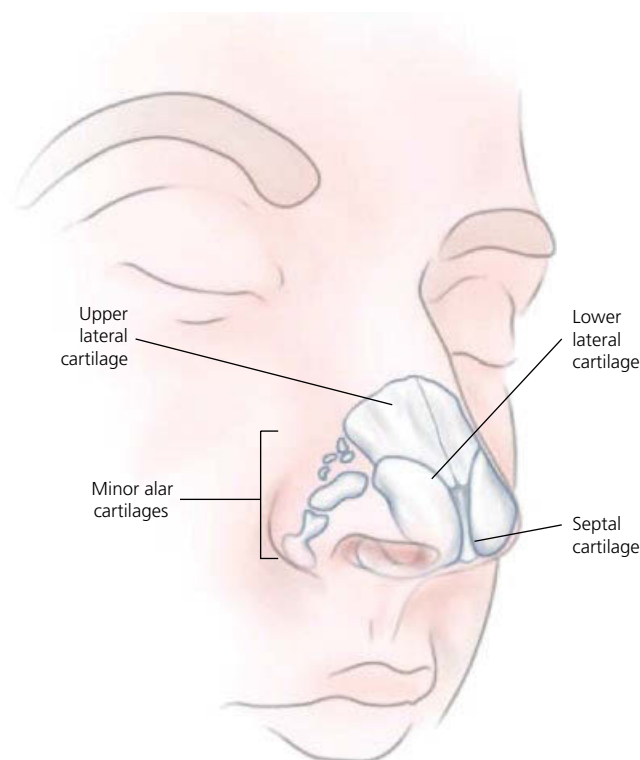


Figure 72.1 The cartilaginous components of the nose in relation to surface anatomy.

articulation with the nasal bones. On their deep surface they are lined by perichondrium that is continuous with the septal perichondrium.³ Their articulation with the septum forms the internal nasal valve; the angle should be between 9 and 15°.⁵

Lower lateral cartilages

The lower lateral cartilages (LLCs) are paired structures in the caudal third of the nose that can be subdivided into three segments: the lateral, intermediate/middle and medial crura (Figure 72.2a–c). The lateral crus supports the anterior half of the alar rim before turning cephalically. The scroll area is the region where the superior edge of the LLC curls downward as it meets the inferior edge of the upper lateral cartilage. This area provides some support for the internal nasal valve. Superior and lateral to the scroll area, one can find several small sesamoid cartilages connected by fibrous tissue that wraps around the alae laterally.^{2,3} The intermediate crus has the most variability in shape and may be convex, boxy, broad or concave; it forms the tip-defining point. It should be noted that skin over this region is particularly delicate as there is a deficiency of subcutaneous tissue: the skin directly abuts the perichondrium necessitating meticulous dissection.³ The medial crura sit next to each other in the columella. The foot-plate is the area closest to the nasal spine where variable degrees of divergence exist. The columellar segment, the middle portion of the columella, extends towards the intermediate crura where the cartilages may diverge again to reach the tip-defining points.

Several analogies have been proposed to describe the dynamics of tip rhinoplasty, relating the medial and lateral crura of the

LLCs and their attachment to the caudal septum to tip support, projection and rotation. The ‘tripod concept’ of the nasal base is the most popular analogy for understanding the interdependent relationships of the nasal base structures. This theory proposes that the nasal base structurally functions like a tripod with the paired medial crura acting as one leg of the tripod and each of the lateral crura acting as one of the other two tripod legs. Altering the length or strength of the legs will result in changes of the rotation and projection of the tip. Although the tripod theory has ample utility in the understanding and planning of surgery,^{6,7} it has limitations in that it may not be the most accurate predictor of the true forces on the nasal tip. Westreich *et al.* proposed an alternate model, which extrapolates on the tripod concept described as a ‘cantilevered spring tripod’. They measured stiffness and elastic modulus of the different cartilages and found that the septal cartilage had the greatest tensile strength. They also found that the strength and support of the nasal tip depended greatly on the thickness of the cartilage and perichondrial attachments more than the elastic modulus of the particular cartilage. With these findings, they proposed considering the nasal tip as a cantilever from the caudal septum.⁸

Septum

The septum comprises bony and cartilaginous portions. The bony septum is made up of the perpendicular plate of the ethmoid bone superiorly, the vomer inferiorly and posteriorly, and the maxillary crest, which forms the caudal-most projection of the premaxilla, anteriorly and inferiorly. The cartilaginous septum is composed of a single continuous quadrangular cartilage that articulates with the vomer and the perpendicular plate of the ethmoid posteriorly and superiorly and is supported inferiorly by the maxillary crest. The anterior aspect of the quadrangular cartilage supports the nasal dorsum and provides tip projection. The anterior septal angle supports the nasal tip with attachments to the medial crura of the LLCs. The posterior septal angle articulates with the anterior-most portion of the maxillary crest. The amount of cartilage in the quadrangular cartilage varies among patients and is typically less robust in non-white patients.³

Superficial muscular aponeurotic system

In the nose, the superficial muscular aponeurotic system (SMAS) is a continuation of the subcutaneous muscular aponeurotic system, which is contiguous from the galea to platysma. Muscles associated with the nose within the SMAS can be categorized into elevators and depressors of the nasal tip. The elevator muscle group include the procerus, levator labii superioris, alaque nasi and the anomalous nasi. Depressors include the alar and transverse portions of the nasalis, the dilator naris posterior, depressor septi and compressor narium.

Blood supply

Understanding the blood supply of the nose is important in order to avoid ischaemic consequences if this rich blood supply is inadvertently and sufficiently disrupted. The dorsal nasal artery, a

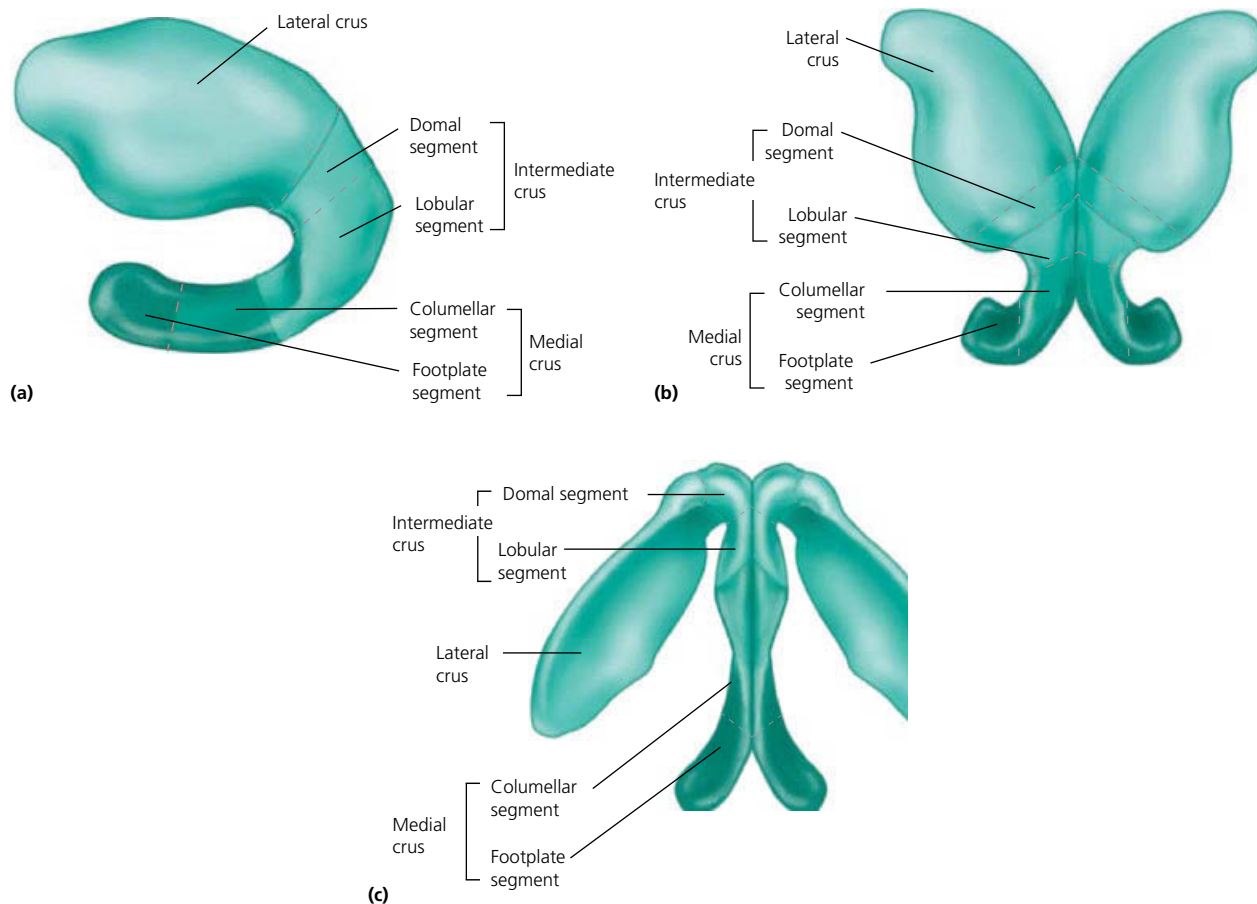


Figure 72.2 (a) Lateral, (b) anterior and (c) inferior views of the lower lateral cartilages.

branch of the ophthalmic artery, penetrates the orbital septum above the medial palpebral ligament and courses along the side of the nose, providing robust blood supply to the dorsal nasal skin. It ultimately anastomoses with the lateral nasal artery, a branch of the angular artery (itself the terminal branch of the facial artery), with numerous branches that enter the subdermal plexus thereby supplying the lateral surface of the caudal nose. The tip of the nose is supplied both by the external nasal branch of the anterior ethmoid artery (a branch of the ophthalmic artery), the columellar artery (a branch of the superior labial artery) and branches of the lateral nasal artery. The external nasal branch of the anterior ethmoid artery passes between the nasal bone and the upper lateral cartilage to supply skin of the nasal tip. Branches of the superior labial artery supply the nostril sill and columellar base (Figure 72.3). Intranasal blood supply is from branches of the anterior and posterior ethmoid arteries, the superior labial and greater palatine arteries, and the maxillary artery.

Venous drainage of the external nose generally consists of veins with names that correspond to the associated arteries, which veins drain through the facial vein, the pterygoid plexus, and ophthalmic veins, the pterygoid plexus and the facial vein. Lymphatic drainage of the nasal tip follows the same path and appears to be limited to the lateral walls, sparing the columella.⁹

Nerve supply

Branches of the second and third divisions of the trigeminal nerves provide sensory innervation of the nose, whereas motor innervation is supplied by the zygomatic branch of the facial nerve. From V_2 , the ophthalmic nerve gives off the supratrochlear and infratrochlear nerves to supply sensation to the rhinion, radix sidewalls. The anterior ethmoid nerve gives off external branches that travel within the SMAS layer to supply sensation to the tip; therefore, careful dissection beneath the SMAS over the tip will preserve tip sensation. From V_3 , the maxillary nerve branches and supplies sensation to the lateral vestibule and columella (Figure 72.4).

Patient assessment

The process of rhinoplasty begins with patient evaluation. Arguably, the foundation of a successful rhinoplasty is established at the initial patient visit, including a thorough, accurate evaluation of the nose; its relation to the rest of the patient's face; identification of appropriate and realistic goals given the patient's anatomy; clear communication; and mutual understanding and agreement of surgical goals between

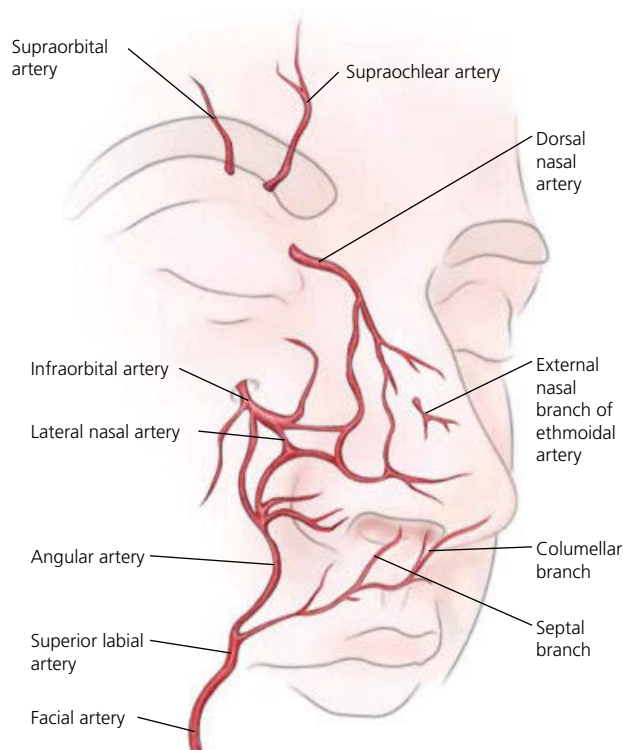


Figure 72.3 Blood supply of the external nose.

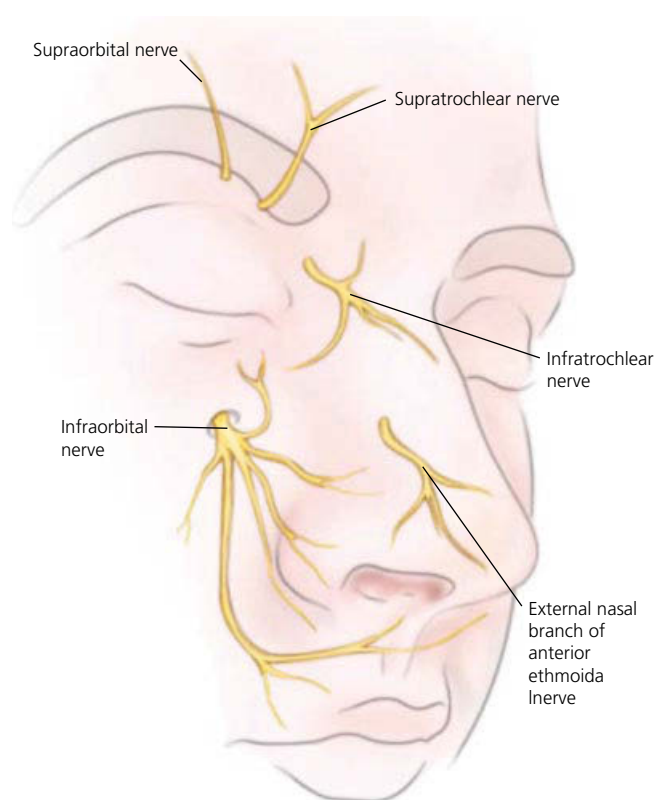


Figure 72.4 Sensory nerve supply of the external nose.

the patient and the surgeon. Meticulous preoperative photo-documentation is compulsory and computer-generated images of potential postoperative goals may be helpful in facilitating patient–surgeon discussion and establishing operative goals.

History

Important components of a complete history include medical, surgical, family and social history as well as thorough review of all prescription and non-prescription medications. Of particular interest is whether there has been any previous nasal trauma or surgery. A history of diabetes, connective tissue disease, anticoagulation or a bleeding or clotting disorder should be noted. Smoking significantly impedes healing and can lead to scarring, skin necrosis and a poor outcome. A psychiatric history may help the surgeon understand a patient's motivation for cosmetic surgery. If a patient's motives are self-driven rather than driven by pressure from peers or a significant other, the patient is more likely to be satisfied with the ultimate surgical outcome. In addition to motivation, it is important to elicit a clear picture of the patient's desires and expectations from rhinoplasty. This should be considered in the surgeon's nasal analysis and proposal of surgical goals and limitations with thorough counselling and establishment of reasonable goals.

Physical examination

Physical exam should include a thorough facial analysis characterizing the nasal structures themselves and their relationship to the face as a whole. A careful intranasal exam should also be performed, including inspection of the septum, turbinates, nasal valves and an assessment of nasal breathing. Finger palpation is an essential component of the exam.

Only in idealized drawings is the face completely symmetric, and strict symmetry of the nose and the face should not be anticipated. The nose should occupy the middle third of the face horizontally and the middle fifth of the face vertically. The nose itself may be divided into thirds vertically. Generally, the upper third comprises the nasal bones, the middle third comprises the upper lateral cartilages and the lower third comprises the tip and the LLCs. Deviations from the ideal should be accurately localized and surgical correction of these variations should address the appropriate structures.

An important feature is the smooth, unbroken and symmetric brow-tip aesthetic line, which is described as a 'gentle sweeping line from the medial brow to the lateral nasal wall to the tip-defining point'; the ideal shape of this aesthetic line in a female nose is that of an hourglass, narrowing in the middle and flaring at the top and bottom.¹

Numerous proportions and relationships have been described to characterize the ideal nose (Table 72.1). In the frontal view, the ideal nasal length is two thirds that of the midface, measured from the glabella to the alar base, and equal to the vertical length of the chin, measured from the stomion to the menton.¹⁰ The width of the alar base should approximate the intercanthal distance and may be up to but not more than 2 mm wider.¹

Table 72.1 Nasal analysis

Frontal view	Brow tip aesthetic line Nasal length = chin vertical = 2/3 midface Alar base width = intercanthal distance + 2 mm Dorsal width = 2/3 alar base width
Profile	Nasofrontal angle 115–130° Nasolabial angle 90° male, 100° female Dorsal straight line until supratip break especially in females Columella and ala should be 1 mm from long axis of nostril
Base view	Equilateral triangle Tear-shaped nostrils Lateral and medial crura should meet at 30°

The radix should be at the level of the supratarsal crease. The width of the bony dorsum should be approximately two thirds of the alar base width.

In the lateral view, the nasofrontal angle should be approximately 115–130°⁴ and the radix should be 4–6 mm deep. In females, the dorsum should extend as a straight line until reaching the supratip break, which aesthetically separates the dorsum from the tip unit. Ideally, the nasolabial angle should be 93–98° for men and 95–100° for women, although this angle varies somewhat based on the height of the patient. In general, shorter individuals may have a more obtuse angle whereas taller individuals may have a more acute angle. In the lateral view, the alar base should be 2 mm above the line between the lower and middle third of the nose.¹¹ The lateral tip projection can be measured from the alar–cheek junction to the tip-defining point.¹⁰

The alar–columellar relationship should be carefully evaluated from the lateral view. When viewed from this aspect, the upper border of the nostril is formed by the alar rim and the lower border is formed by the columellar rim. Gunter described a method of analysing this relationship. A line is drawn which approximates the long axis of the nostril: the ala should be 1–2 mm above this line and the columella 1–2 mm below this line. If the columellar rim is less than 1–2 mm from this line, the columella is considered to be retracted; conversely, if the distance is greater than 1–2 mm, the columella is considered to be ‘hanging’. If the ala is less than 1–2 mm from this axis, it is considered to be a hanging ala; if it is greater than 1–2 mm from this axis it is considered a retracted ala.^{12,13}

The alar–columellar relationship and alar rim deformities should also include analysis from the alar base view. The ideal base view is an equilateral triangle with each ala forming the sides of the triangle and the alar base width forming the remaining side. A concave ala is present when the lateral border of the ala is medial to the line of the equilateral triangle, whereas when it extends lateral to the line of the triangle, it should be considered convex.¹⁴

The ideal nostril is oval shaped, wider than the columella, and should be almost parallel to the vertical axis of the columella in the nose of a white person. The length of the columella should be twice the height of the infratip lobule. The alae should insert

at a soft angle to the cheek with a slight curve medially. Disharmonies of the alar base can be in either the vertical or horizontal plane, and careful analysis is critical, as surgical correction of these deformities differ depending on the nature of the deformity.¹¹

The relationship of the nose to surrounding facial structures affects the perception of nasal features. A sloping forehead may give the illusion of overprojection of the nasal tip whereas a flattened forehead may give the illusion of an underprojected nasal tip. Similarly, an underprojected chin may make the nose appear relatively overprojected and vice versa.

The aesthetics of the nasal tip may be defined by a number of relationships, many of which are quite subtle. The angle at which the lateral and medial crura of the LLCs meet should be about 30° on either side. The ideal nasal tip should be well defined. Subtle variations in anatomy may make it appear boxy or bulbous. Wide lateral crura may blunt dome definition resulting in a boxy tip. A greater than 30° angle between the lateral and medial crura or divergent domes can result in a bulbous appearing tip.¹

Physical examination should include palpation of the bony and cartilaginous components of the nasal dorsum to understand the relationships between them. The tip recoil test is performed by placing gentle pressure on the tip to assess the strength of the LLCs. An intranasal exam should be performed to identify deviations of the nasal septum, turbinate hypertrophy and any internal causes of nasal obstruction. The Cottle or modified Cottle manoeuvre is performed to assess nasal valve collapse.

Although the most thoroughly studied and well-described ideal nasal aesthetics pertain to the nose of patients from the white population, much individual variety exists and these ideals cannot be indiscriminately applied to all noses, particularly in different ethnicities such as the African, Asian, Middle Eastern or Hispanic noses.¹⁵

The African nose has a wide base and low, flattened appearing dorsum. The ascending maxillary process often lacks vertical projection, which contributes to the low dorsum. Increased distance at alar insertion and alar flaring both contribute to the increased interalar distance.¹⁶ There also tends to be increased fibro-fatty tissue and thicker sebaceous skin at the tip, both of which contribute to lack of tip and soft tissue triangle definition.¹⁵

The Asian nose has typical characteristics somewhere in between the white and African nose. There is typically a low dorsum that begins more caudally. Again, the skin at the tip and supratip region is thicker than the white nose but not quite to the extent of the African nose. The alar width is slightly wider than the white nose. Characteristically, in the Asian nose, there is paucity of septal cartilage leading to a less projected tip and occasionally retracted-appearing columella. This should be anticipated in Asian rhinoplasty and consideration of other sources of cartilage grafts should be explored and discussed. There also tends to be weaker LLC.¹⁷

The Middle Eastern and Indian noses share some characteristics with the nose in white populations with similar height of

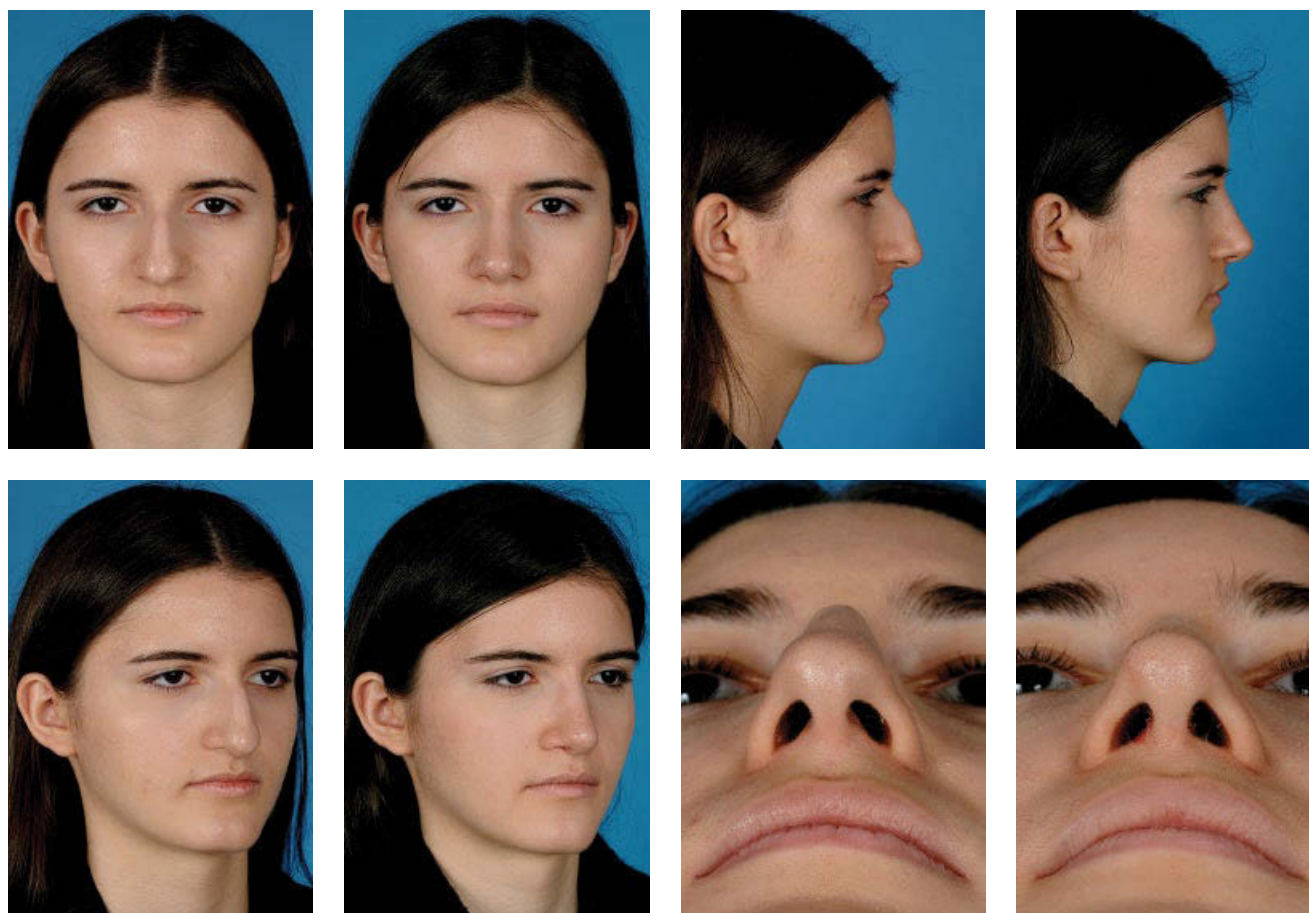


Figure 72.5 Photographic standardization assists surgical planning and allows accurate pre- and post-op comparison, thereby facilitating the surgeon's self-assessment and critique allowing continual refinement of surgical technique.

the dorsum.¹⁵ However they also frequently have overprojection of the dorsum resulting in a high dorsal hump. There is mild alar flaring and thick skin around the tip region. There is variable skin thickness of tip and alar flaring. Dorsal height also varies between the norms of white and Asian noses.¹⁸

Photography

The standard approach to photography for rhinoplasty is to obtain realistic preoperative photographs using a digital single lens reflex (SLR) camera. Photos should be taken with a standard blue background with appropriate lighting. Many surgeons will have a dedicated space for photography in their office. Standardization of patient positioning, lighting and angles should be maintained. This will facilitate the surgeon's study of the patient's nasal anatomy and facial harmony for surgical planning. Moreover, it permits accurate pre- and postoperative comparison, thereby facilitating the surgeon's self-assessment and critique allowing continual refinement of surgical technique (Figure 72.5).

An important aspect of successful photography is the lighting and positioning for rhinoplasty portraits. The simplest lighting setup is a single mounted flash on top of the

camera itself; however, this can result in harsh and uneven shadows. There are several established, sophisticated lighting arrangements that have been described.¹⁹ The key light system, developed for portrait photography, positions the main light at a 45° angle from the patient, casting a shadow from the nasal tip to the oral commissure. A second fill light is placed at nearly the same angle on the opposite side in a position that it fills the shadow cast from the main light. Then, two backlights are placed behind the patient to separate him/her from the background. The quarter system places two lights equally at 45° angles from the patient.

The traditional six standard photographic views taken for rhinoplasty are the frontal anteroposterior, right and left lateral, right and left oblique and base views.^{20,21} A top view is a useful addition that can be used to better characterize and understand deviations in the crooked nose, especially for patients with dorsal deviation.⁵ Some surgeons routinely obtain a smiling view. Proper positioning in both frontal and lateral views requires alignment along the Frankfort horizontal plane, defined by a line that connects the top of the tragus to the infra-orbital rim; this line should be truly horizontal and parallel to the ground.²⁰

Table 72.2 Rhinoplasty dynamics

Facial dynamic dimension	Procedures that increase	Procedures that decrease
Intercanthal distance	Deepening radix	Osteotomies Augmentation of the radix
Nasal length	Deepening or decreasing height at the radix	Decreasing nasal height at medial canthal level
Width of dorsum	Reduction of dorsum	Osteotomy Augmenting dorsum
Height of dorsum		
Tip projection	Nasal spine graft Maxillary advancement Transdomal suture Columellar strut Anchoring medial crura of LLC to nasal spine Footplate approximation	Dorsum reduction Alar base narrowing Nasal spine reduction
Tip rotation	Cephalic rotation: Reduction of dorsum Reduction of caudal septum Reduction of cephalic margin of LLC Columellar strut placement Footplate approximation Nasal spine augmentation	Caudal rotation
Width of nasal tip	Interdomal or subdomal grafts Onlay graft Subdomal graft	Interdomal stitch Transdomal stitch Lateral crural spanning stitch Tip graft
Width of nasal base		
Upper lip length	Nasal spine augmentation	Nasal spine reduction

Rhinoplasty dynamics

The nose is a three-dimensional centrepiece on the face with critical structural and functional roles. There are many variables and dimensions that can be adjusted to alter the aesthetic appearance, structural components and functional role of the nose. There are many tools and manoeuvres available to the rhinoplasty surgeon to adjust these numerous variables. Although each adjustment may have a primary anticipated effect, such as increasing tip projection or reducing a dorsal hump, it will invariably have consequences, desirable or undesirable, to another variable nasal dimension (Table 72.2).²²

Adjusting the height at the radix or the caudal aspect of the dorsum will have different effects on a number of facial dimensions. Lowering the radix will frequently result in the appearance of increased intercanthal distance and increased nasal length. Augmenting the dorsum at the radix will appear to reduce the intercanthal distance and shorten the nose. In contrast, reducing the height of the dorsum more caudally can have the opposite effect, giving the nose a shorter appearance and the illusion of greater cephalic rotation of the tip. Similarly, reduction of the caudal dorsum will cause the intercanthal distance to appear wider, while augmenting the dorsum will cause the intercanthal distance to appear more narrow. Reducing the caudal dorsum may also reduce the tip projection as the caudal septum has an important role in supporting tip projection. Osteotomies can result in a narrower appearing nose and intercanthal distance.²²

Numerous procedures may be used to adjust tip projection and width; however, not every procedure is appropriate for

every patient. For example, an overprojected tip can be reduced with a cephalic trim of the LLCs only if the cephalic portions of the LLCs are actually the highest projecting part of the caudal tip. If this is not the case, securing the footplate of the medial crura to the caudal septum can decrease or increase tip projection depending on the location on the septum to which they are secured. Reducing tip projection may result in the appearance of a relatively widened alar base and bowed columella. A number of manoeuvres are frequently used to both increase tip projection and increase the angle of cephalic tip rotation. Placement of a columellar strut, anchoring the medial crura to the caudal septum, approximating the medial crura foot plates and advancing the nasal spine can all result in the appearance of increased cephalic rotation and projection. Advancing the nasal spine may also shorten the upper lip. Care must be taken to choose the appropriate procedure for the anatomical constraints presented by each patient.²²

The astute nasal surgeon will be aware of these effects in planning for rhinoplasty to anticipate the most appropriate and effective manoeuvres for a specific patient's anatomy, during surgery make adjustments when needed and postoperatively to assess the results of their adjustments.

Operative approach

For several decades, surgeons have debated the merits of 'open' or the 'external approach' versus endonasal rhinoplasty. In the United States, most rhinoplasty was performed endonasally in

the latter decades of the twentieth century, whereas today, external approach rhinoplasty is the technique of choice for most patients by the majority of rhinoplasty surgeons, including the authors, who reserve the endonasal approach for certain patients needing only minor revisions. One cannot definitively say that one approach is better than the other for all surgeons, as good results can be achieved by some surgeons using one method whereas equally good results can be achieved by others using the other technique. Some criticize external approach rhinoplasty with the assertion that by opening the skin–soft tissue envelope, some of the strength and support of the tip are lost when the soft tissue attachments are separated from the cartilaginous support. Proponents of the external approach feel there is better visualization and understanding of the anatomy, especially for the novice rhinoplasty surgeon and in the teaching setting. Cartilages are observed in their natural anatomic position and can thus be manipulated *'in situ'* as opposed to delivering them to make changes. Endonasal rhinoplasty may be more suitable for noses that may require a limited cephalic trim but otherwise have a well-defined tip and symmetric base requiring minimal, if any, intervention in these areas.²³

Although the primary difference between the two approaches appears to be the mid-columellar incision, which connects the bilateral marginal incisions also used in the endonasal approach, we believe that the more important difference is the ability to directly visualize the underlying structures and their relationships to one another in their resting position. Indeed, most of the trimming, suture and grafting techniques performed are more easily performed with the exposure provided by the external approach.

Endonasal rhinoplasty

A very brief discussion of endonasal rhinoplasty is warranted, although not our preferred approach. Endonasal rhinoplasty is performed using one of several incisions: a marginal incision (at the lower border of the LLC), an intercartilaginous incision (between the upper and LLCs), a transcartilaginous incision, or a rim incision (largely abandoned because of predictable alar notching). To modify the nasal tip with the endonasal technique, the LLCs are typically brought out of the nostrils, or 'delivered'. The bilateral marginal and intercartilaginous incisions allow the development of bipediced chondromucosal flaps consisting of the LLCs and the underlying mucosa. The cartilages are delivered through the nostril from which they came or can be delivered through a single nostril and then modified as required. The main drawback of cartilage delivery with the endonasal approach is that it requires retraction of the LLCs from their normal anatomic position; adjustments are then made with the cartilages out of their normal resting positions.

The plane of dissection is similar to that of the external approach: above the perichondrium and periosteum of the nasal cartilages and bones. Concurrent septoplasty is well described and routinely performed with good outcome, although it is performed through a hemitransfixion incision or a Killian incision

as opposed to from above. Dorsal hump reduction can be achieved using an osteotome or rasps, albeit with less direct visual control to make fine adjustments compared to the external approach.⁷

Many surgeons feel that a primary limitation of the endonasal technique when combined with septoplasty is if there is deviation of the L-strut requiring stabilization with spreader grafts, as this can be difficult to accomplish. Many surgeons also feel that the endonasal technique is more limited in its ability to allow adjustments of the alar rim, base and tip; however, the cartilage delivery technique of endonasal rhinoplasty can provide access to perform cephalic trim of the lower lateral crura as well as a variety of dome sutures.

External approach (open) rhinoplasty

Although every rhinoplasty operation should be individualized to each patient's particular anatomic indications, a systematic order and algorithm may be helpful in operative planning as well as establishing a logical progression of steps and maintaining stability. Local anaesthesia using lidocaine (lignocaine) with adrenaline (epinephrine) should be carefully but judiciously infiltrated into all of the areas of the nose to be dissected, but not adding so much volume that the features and subtle nuances of anatomy are masked.

Numerous variations of the columellar incision exist. The authors use a mid-columellar stair-step incision, whereas many surgeons prefer an inverted V or 5-cornered incision which is connected with bilateral marginal incisions placed at the caudal border of the LLCs. Care is taken not to violate the soft tissue triangles. Dissection of the skin–soft tissue envelope is performed in the subperichondrial avascular plane directly over the cartilage of the LLCs, then the upper lateral cartilages; keeping the dissection in this plane will allow the surgeon to avoid the neurovascular structures near the nasal tip.⁹ The dissection is then continued cephalically to the caudal aspect of the nasal bones, at which point a periosteal elevator is used to elevate the periosteum of the nasal bones in the radix region.

As previously noted, each operation should be individualized to address each patient's anatomy. As required, the radix is addressed first, either by reducing it with a guarded burr if it is overprojected or by placing radix grafts if it is underprojected.

Next, the bony and cartilaginous dorsum is addressed. Preoperative evaluation of the dorsum should have identified whether there is an excess or deficiency of the dorsum. The skin is thinnest at the rhinion or bony–cartilaginous junction and becomes thicker cephalically towards the radix and caudally towards the nasal tip. Therefore, a slight prominence should exist at the upper portion of the upper lateral cartilage in order to compensate for the thinner skin to create a straight profile. Any cartilage or bony hump in excess of this should be removed.³

The upper lateral cartilages are released from the septum, preserving the underlying mucoperichondrium thereby minimizing chances of postoperative internal valve narrowing. Any significant prominence of the cartilaginous and bony dorsum is

addressed together, using sharp dissection and an osteotome or a rasp. For a larger dorsal hump, a Rubin osteotome may be used to reduce the bony hump en bloc.^{23,24} The bony dorsum can be further reduced or smoothed with rasping.²³ Precise cartilage reduction can be achieved by removing consecutive thin slices with a 15 blade until reaching the desired profile. After dorsal hump reduction, the patient may be left with an 'open roof deformity' where the lateral nasal bones and upper lateral cartilages are no longer touching the septum. This deformity is corrected later in the procedure with osteotomies and spreader grafts. When the dorsum is deficient, grafts can be placed to create a pleasing aesthetic.

If there is significant septal deviation, the septum is addressed at this time either from the dorsal approach or intranasally through a hemitransfixion or Killian incision. Also at this time, if cartilage grafting is anticipated, septal cartilage can be harvested. As the old adage wisely professes: 'Where the septum goes so goes the nose'. Several indications exist for septoplasty in septorhinoplasty: obstructed nasal breathing associated with septal deviation, dorsal deviation associated with deviation of the anterior septum and for harvest of graft material for other aspects of the rhinoplasty.

When septoplasty is performed from the dorsal approach, the anterior septal angle is identified: the LLCs are retracted laterally and downward and the septum is identified caudal to its attachment to the upper lateral cartilage. The perichondrial envelope is dissected from the septal cartilage in the subperichondrial plane and, if not already performed or as dissection proceeds, the upper lateral cartilages are released from the septum to provide adequate visualization of the more posterior septum. Preservation of the mucoperichondrium provides support and ultimately reduces internal valve collapse.

Once the septal cartilage and bone is exposed, it is assessed to determine which portion of the septal cartilage must be resected or weakened in order to relieve the deviation. One should also consider how much cartilage grafting material should be harvested. An important principle guiding septoplasty or septorhinoplasty is to maintain the structurally supportive 'L-strut' composed of dorsal and caudal septum. We recommend leaving at least a 1.5 cm, preferably 2–2.5 cm, dorsal strut to maximize the degree of support. If significant deviation exists in the L-strut, this may be corrected and stabilized with unilateral or bilateral spreader grafts.⁵ An effectively placed horizontal mattress suture (e.g. 4-0 PDS®) may correct mild deviations of the L-strut. Gruber *et al.* demonstrated that a 10 mm wide, 0.5 mm thick piece of cartilage with a curvature may be adequately straightened with a horizontal mattress suture placed with an 8 mm longitudinal spacing with the knot on the convex side.²⁵

If there is a spur or deviation involving the bony septum, either superiorly in the perpendicular plate of the ethmoid or posteriorly in the vomer, these may be addressed by resecting the deviated portions. Caution should be exercised during resection of the perpendicular plate; avoiding rocking motions, as CSF leak may occur as a rare complication of septorhinoplasty.

If the inferior edge of the caudal L-strut has been released from the maxillary crest, it can be repositioned in the midline and sutured to the maxillary crest periosteum to secure it. Alternatively a piece of cartilage may be secured with a permanent suture as bridge between the caudal septum and the nasal spine if there is not enough length for adequate projection.⁵

If the septorhinoplasty is being performed to improve nasal airway obstruction, inferior turbinate hypertrophy should be addressed with submucosal resection, inferior turbinate outfracture and/or conservative resection of the hypertrophic portions of the turbinate, being careful not to be overzealous, possibly causing atrophic rhinitis or empty nose syndrome.

Once satisfactory dorsal projection is achieved, osteotomies are planned and executed as necessary. Osteotomies are performed to narrow the bony dorsum, to close an open roof deformity created by dorsal hump reduction or to straighten a crooked bony dorsum. Recalling anatomic considerations, the ideal width of the bony dorsum is about two thirds that of the alar base width. The nasal bones and ascending maxilla tend to be thicker cephalically; therefore, planning osteotomies higher may help to minimize the narrowing effect on the internal nasal valve and nasal breathing. However, if the osteotomy is carried too high or if the medial and lateral osteotomies do not meet, a rocker deformity may result. Another anatomic consideration in osteotomy is the patient age: younger patients tend to have more flexible bone whereas older patients have more brittle and easily fractured bone. Interrupted 'postage stamp style' osteotomies in younger patients may be less effective if they result in greenstick fracture and their nasal bones spring back into place.⁴

It is important to perform effective injection of local anesthetic containing adrenaline prior to the osteotomies in order to minimize bleeding and postoperative swelling. The periosteum of the nasal bones is elevated medially while the lateral periosteum is left attached to bone in order to provide stability to the segment of bone after lateral osteotomy during the healing period. If significant hump reduction has been performed, a formal medial osteotomy may be unnecessary.

In general, medial osteotomies should be performed first, followed by intermediate osteotomies (if indicated), then the lateral osteotomies, in order to maintain stability of the remaining lateral portions of the nasal bones for each succeeding osteotomy. Medial osteotomies are usually performed intranasally by seating the osteotome between the septum and the upper lateral cartilage at the caudal edge of the nasal bone. Since the upper lateral cartilages obtain their structural support from their connection to the underside of the nasal bones, this relationship must not be interrupted.⁴ Medial osteotomies are usually and ideally performed in a laterally fading or oblique direction. If no open roof deformity is present, some surgeons create medial osteotomies that are directed linearly upwards towards the nasal root, but this can result in a rocker deformity. A straight medial osteotomy may be more appropriate in a nose with a deviated

dorsum that may require intermediate osteotomy or if the bony septum needs to be repositioned. If a straight osteotomy is performed, a transverse (anteroposterior) osteotomy may be necessary at the cephalic limit at the level of the medial canthal ligament to connect with the lateral osteotomy and mobilize the bony segment.² Transverse osteotomies should be performed below the level of the canthus to minimize risk of lacrimal system injury, and if performed correctly can provide more precise control of nasal bone positioning.²²

Intermediate osteotomies may be performed for a deviated nasal dorsum. They should be designed for placement at the apex of the deformity, on the side of deviation.

The lateral osteotomy may be performed in a continuous fashion in a caudal to cephalic direction or through percutaneous stab incisions. With either approach, the inferior limit of the lateral osteotomy should be just above the level of the inferior turbinate insertion in the ascending process of the maxilla and care must be taken to preserve the bony triangle at the level of the piriform aperture at the caudal aspect of the maxillary bone. Staying above the inferior turbinate, as in a high-to-low osteotomy, will minimize risk of nasal airway compromise and address a wide bony base at the same time. If only a small open roof deformity exists, low-to-low osteotomy may be used to correct this.²² For correction of the anteriorly wide nose it might be necessary to remove a wedge of bone between the nasal bone and the septum to allow medial repositioning of the lateral nasal bone.

If a continuous osteotomy is performed, a subperosteal tunnel is dissected with a Joseph elevator. A guarded 2–2.5 mm osteotome is used to minimize mucosal tears. The lateral osteotomy connects with the medial osteotomy cephalically. If performing transcutaneous perforating osteotomies, one or two well-placed 2 mm stab incisions through skin are all that is necessary to obtain a series of percutaneously directed ‘postage stamp style’ osteotomies using a 2 mm osteotome. The most inferior punch should be directed slightly higher in order to minimize collapse of piriform aperture. The more superior osteotomies should be placed high in order to maximize narrowing of dorsum and avoid rocker deformity.

Tip and lower lateral cartilages

The ideal nasal tip should be well projected, supported, not over- or under-rotated, and well defined without being bulbous or have skin that is so tightly pulled over the cartilage that the tip appears bifid. Appropriate tip projection may vary in different ethnicities. This should be considered in ethnic rhinoplasty, as overprojection may result in racial incongruity^{15,17} and disharmony with the rest of the patient’s face.

Various cartilaginous trimming, grafting and suture techniques may achieve nasal tip definition and correct mild tip deviations. Conservative trimming of the lateral crus may be performed to even out asymmetries. It is imperative to leave a minimum of 6 mm strip of the lateral crus to preserve enough strength to prevent buckling.²⁵

A lateral crural mattress suture may be used to strengthen the lateral crus or to straighten convexities of the ala, either after over-resection or in the setting of weak cartilage. For the LLC, typically about 6 mm wide and 0.5 mm thick, a mattress suture may be placed with 6 mm spacing between the two loops of the stitch. Multiple stitches may be placed along the length of the LLC for added strength.²⁵ The same concept may be used to provide a subtle increase in strength for the columella. A columellar–septal suture strengthens the columella when only a mild weakness is present that does not require grafting.²⁵

Dome stitches may be used to not only create more definition of the nasal tip but also to correct mild deviations or asymmetries by bringing together divergent intermediate crura.⁵ If there is any chance of knots protruding into the skin, absorbable monofilament PDS® is preferred. If sutures are placed deep in a location with no risk of extrusion or visibility, nylon is also a viable option.²⁵ A hemi-transdomal stitch is placed through the vestibular skin and traverses the most cephalic end of the dome and attempts to evert the lateral crus. A transdomal suture is a horizontal mattress suture that brings together the medial and lateral crura of one of the LLC.²⁶ When placing this stitch, care must be taken to avoid causing the lateral crus to invert as it may result in concavity of the ala. Also, if the lateral bite of the stitch is not placed laterally enough, it may result in counter-rotation and decrease in projection. A transdomal stitch placed appropriately laterally should increase tip rotation, projection and definition.²³ An interdomal stitch approximates the medial crura together and is set back from the tip several millimetres.²⁵

As previously described, in the ideal profile view of the nose, the ala sits 1–2 mm above the long axis of the nostril and the columella 1–2 mm below it. It is vital to identify the correct anatomic problem: when a hanging ala is present, this should be correctly distinguished from retracted columella so that the appropriate problem is addressed.¹³ If a mild alar retraction exists (<2 mm from the long axis of the nostril), an alar rim graft will not only correct this deformity; it will also correct the concavity commonly associated with the retraction. On noses with wide bases and mild alar retraction, a small wedge of alar skin may be resected and the remaining ala repositioned inferiorly. If greater than 2 mm of retraction exists, a V-Y advancement, a composite graft, or an intercartilagenous graft may be required between the upper and lower lateral cartilages on that side.²² These manoeuvres can lower the ala 4–8 mm. A hanging ala may be corrected by excising a small wedge of vestibular skin intranasally or by resecting a strip of cartilage at the caudal edge of the lateral crus of the LLC. A hanging columella can be corrected by resecting a thin strip of the caudal cartilaginous septum or by securing the medial crura to the caudal septum in a tongue-in-groove configuration.²⁷ Columellar retraction can be addressed by columellar strut or extension grafting.¹³

The base view of the nose should approximate an equilateral triangle. If there is convex ala protruding from the triangle, debulking of soft tissue, trimming lateral crura or placing dome spanning sutures may correct this. Concave ala may be corrected

by alar rim or strut grafting with cartilaginous grafts on top of or deep to the lateral crus respectively depending on the severity of the concavity.¹⁴

Grafts

Autologous graft material is preferable to alloplastic implants, as the latter is significantly more prone to infection and extrusion. Common choices of graft material include septal cartilage, conchal cartilage, rib cartilage and temporalis fascia. Cadaveric irradiated cartilage and alloplastic materials such as Medpore®, silicone, and Gortex® are used by some surgeons. For most primary rhinoplasties, septal cartilage is preferred and has several advantages: it is readily accessible and in the same operative field, abundant (in patients who have not undergone septoplasty), and strong, which is ideal for supportive grafts. Auricular conchal cartilage can be harvested without additional prep if its use is anticipated and the ear is prepped into the operative field. Some surgeons prefer a preauricular incision, whereas others prefer a postauricular incision for graft harvest. A significant amount of cartilage may be harvested without compromising the structural or aesthetic appearance of the ear as long as a few millimetres of rim are left in place along the conchal wall. Conchal cartilage is curved and significantly weaker than septal cartilage thus making it less suited for supportive strut grafts and better for alar reconstruction. Potential donor site morbidity includes auricular haematoma or perichondritis, although their risk can be minimized by the use of antibiotics and a bolster dressing.

Rib cartilage may be necessary for significant nasal reconstructive procedures. The rib provides an abundant source of graft material but it carries the minor disadvantage of requiring a second harvest site and potentially causing a pneumothorax. The most common source of costal cartilage for grafting in females is the 6th and 7th rib cartilages, as the incision can be hidden in the inframammary crease. Costal cartilage carries the risk of warping once it is in place. The effect of warping can be minimized by keeping the cartilage in saline for 30–60 min after harvest, determining in which direction twist will occur, and anticipating warp when carving the cartilage. In some cases, securing the rib cartilage in place with a K-wire is a useful manoeuvre to minimize warping. Perichondrium, native fascia or temporal-parietal fascia harvested from the patient can be laid over cartilage grafts to camouflage irregularities. Multiple layers of fascia can be laid on top of each other with each layer of fascia resulting in 0.5 mm of augmentation²⁸ although additional layers of fascia may prolong postoperative swelling. For bulk, any of the above cartilages can be diced and wrapped in fascia. These fascia-wrapped cartilage grafts can add volume in areas such as the dorsum but have little utility as supportive grafts.²⁸ We prefer placement of diced cartilage using a 1 mL insulin syringe in an ideally prepared pocket without fascia. Alloplastic material such as Medpore®, a high-density polyethylene, is theoretically biocompatible and intentionally placed pores allow fibrovascular ingrowth which serves to prevent graft migration. However, the risks of extrusion and infection and the

fact that it is often difficult to remove because of the fibrovascular ingrowth make it less ideal than autologous graft materials.²⁹

Spreader grafts

Spreader grafts may be indicated when there is mid-vault nasal obstruction to widen the internal nasal valve, to augment an overly narrow dorsum, to support a deviated L-strut, to fill a defect from dorsal hump reduction or when there is an inverted-V deformity. We believe that any time a large enough hump is removed to create an open roof, the use of spreader grafts becomes mandatory, otherwise an inverted-V deformity will ensue months or years later. The ideal material for a spreader graft is septal cartilage. Costal cartilage, layered or folded conchal cartilage, or the medial edge of the upper lateral cartilages (autospreaders) may also be used. Typically the cartilage is cut into strips approximately 5 mm high and 30 mm long, spanning the cartilaginous dorsum. If there is a curve in the graft cartilage, it can be scored and oriented so that the two pieces counteract the other's curve. The graft is secured with at least two mattress sutures. The mattress sutures each traverse five layers, securing both pieces to the septum medially and the upper lateral cartilages laterally.⁵

Supportive grafts

The columellar strut graft is the most commonly used graft. The ideal material for a columellar strut is septal cartilage. It can be placed to help support the tip and overcome ptosis and over-rotation of the tip. The medial crura of the LLC can be secured to the graft to prevent hanging columella.¹⁰

For dorsal augmentation and increased tip projection with adequate support, septal extension grafting may be performed. Adequate healthy skin must be present at the tip, as this graft may stretch the skin increasing the risk of skin necrosis. The ideal graft material is either septal cartilage or a straight segment of rib. This graft may be secured to extended spreader grafts that extend beyond the caudal septal angle of the native septal cartilage using sutures.¹⁰

A lateral crural strut graft can be placed to correct a prior concavity of the base view of the ala or to counteract the effect of dome sutures that may lead to concavity. A pocket is dissected on the deep surface of the lateral crus of the LLC beneath the vestibular skin intranasally. The graft cartilage is placed in the pocket and secured to the lateral crura using mattress sutures.

Alar rim or batten grafts, fashioned from septal or conchal cartilage, may be used to support the nasal valve. These grafts can span between upper lateral cartilages and LLCs at various positions. Preoperative assessment should pinpoint the location of nasal valve collapse in order to direct placement of the graft. Cephalic placement on the LLC will stent open the internal nasal valve, whereas caudal placement on the LLC will stent open the external nasal valve.²⁹ Once the appropriate position is decided, the graft is secured with sutures.

The ideal alar rim smoothly transitions from the tip-defining point to the nasofacial insertion, the contour and strength of

which are determined by the lateral crus of the LLC. The LLC is in a position close to the rim more medially but laterally is in a more cephalic position as it moves away from the alar margin: this lack of rigid support can lead to scarring, retraction and/or unpredictable asymmetry. The alar rim graft, placed along the alar margin, is a powerful graft capable of directly altering the contour and strength of the alar margin. Indications include correction of alar flare, cephalic malposition and inadequate alar support, and correction of dynamic margin collapse.³⁰ On patients with cephalically oriented LLCs, the lateral crura are dissected and transposed caudally.

Several types of tip grafts can be used to create a more defined-appearing tip and/or to achieve slight increase in projection of the nasal tip. A shield or onlay graft is cut or prepared using the tip punch device designed by our group and placed as an onlay graft on top of the LLCs or as a shield graft.³¹ A cap graft is created from a horizontal piece of cartilage that crosses both domes with bevelled edges laterally. The cap graft can be used to correct minor tip asymmetries. An infralobular graft can be attached to the septal cartilage in order to increase columellar show in noses with a retracted columella. An umbrella graft can also increase projection and support. After placement of a columellar strut, an onlay to the nasal tip is supported by the columellar strut and overlies both domes. A dome spreader graft can be used to correct a pinched dome appearance. The dome spreader graft is placed after dissecting a deep pocket between the medial crura, placing a small piece of cartilage in the pocket and securing it with monofilament suture.¹⁰ A subdomal graft is used to control the position and the distance between the domes.³²

Alar base adjustments

Alar adjustments should be reserved for the last step after all the tip refinement and supportive grafts have been completed. An overprojected nasal tip or hypoplastic maxilla may give the illusion of narrowed alar width. Similarly an underprojected tip or protruding maxilla may give the illusion of widened alar width. These issues must be ruled out before considering adjusting the alar base positions.¹¹ Alae that are displaced cephalically can be repositioned by resecting an ellipse of skin around the alar base and repositioning the ala medially; this will bring it to a more medial and caudal position. Wedge excisions according to the appropriate abnormality can remove part of nasal sill, ala or both. V-Y advancement and inverted-T excision for wide and thick alae are other manoeuvres that can be useful in certain patients.¹¹

Final steps

Typically, if septoplasty has been performed and there is a mucosal incision, it is closed with interrupted 4-0 chromic sutures and intranasal Silastic® or Doyle® splints are placed. Some surgeons prefer quilting sutures to reapproximate the apposing mucoperichondrial flaps in the absence of the removed septal cartilage. The columellar incision is closed with

interrupted 6-0 fast absorbing catgut sutures. Marginal incisions are closed with interrupted 6-0 fast absorbing catgut sutures.

The dorsum is taped with Steri-strips®. If osteotomies were performed, the dorsum is splinted with a Denver Splint® or a thermoplastic splint. If a rib graft has been used for dorsal grafts, it may have been secured in place with K-wires: these are pulled through the splint and secured in place until they are removed 2-3 weeks postoperatively.

Postoperative care

Postoperative care varies among surgeons. Patients are counselled about the potential for postoperative bleeding, which can be self-limited and treated with oxymetazoline spray. In the immediate postoperative period (24-48 h), patients should keep their head elevated to minimize swelling. Patients should avoid exercise or strenuous activity for 2-4 weeks postoperatively. Postoperative antibiotic prophylaxis may be considered if intranasal splints have been placed or an ear bolster is used after conchal cartilage harvest. Sutures and splints are removed one week postoperatively. Although it is not always necessary or indicated, postoperative steroids have been observed to be effective in decreasing swelling.³³ Perioperative steroids have been shown to decrease periorbital oedema in the immediate postoperative period.³⁴

Complications

After rhinoplasty, the normal postoperative course has some expected inconveniences that the patient should be made aware of preoperatively and should not be considered as complications. Pain, bruising and postoperative swelling are to be expected. If the swelling is excessive or prolonged, postoperative oral steroids may help to speed resolution. Taping the nose may be helpful in decreasing swelling and may be continued by the patient on a nightly basis even weeks after surgery.¹⁵ Gryskiewicz *et al.* feel that after 14 days, Kenalog® injections may be considered every 2-4 weeks for up to six injections.³⁵

Intraoperative complications should be identified and addressed prior to completion of surgery. These include excessive bleeding, iatrogenic injury to the nasal skin or underlying cartilages, lack of dorsal support as a result of fracture of the L-strut, septal fracture; inward displacement or collapse of the nasal bones; 'rocker deformity'; and cribriform plate fracture. The risk of bleeding can be minimized by counselling the patient to avoid not only anticoagulants and non-steroidal anti-inflammatory drugs (NSAIDs), but also supplements that may have anticoagulative effects. Bleeding can also be minimized with control of hypertension, topical intranasal vasoconstrictors, adequate preoperative injection of a local anaesthetic combined with adrenaline, dissection in the proper plane, and careful but judicious use of bipolar cautery. Hypertension is the most common reason for intraoperative bleeding and should be appropriately controlled by the anaesthesiologist. Von Willebrand disease is more common than many realize and patients are treated with desmopressin (DDAVP), also useful in patients who have taken NSAIDs. Obviously, iatrogenic

injury to the nasal cartilages or nasal skin should be avoided, but careful repair should be performed if such an injury occurs. The septum can be fractured in about 1% of septorhinoplasty cases. This is more likely to occur if there has been previous septal cartilage harvest or significant dorsal hump reduction, deficient nasal bones or a severely deviated septum. A fractured L-strut can be addressed with suture fixation to spreader grafts and/or by fixation of the strut to the nasal bones. Intraoperative complications associated with osteotomies can be identified immediately after the osteotomies are completed. A collapsed or unstable displaced nasal bone can be suspended from the septum if still attached to the upper lateral cartilages. Sutures, or infrequently, K-wires can be used to stabilize the bones or, one can place a Merocel® pack intranasally for one week to hold the bone(s) in position. A rocker deformity may occur if the lateral osteotomy is carried too far upward and laterally beyond the radix. With a rocker deformity, inward displacement of the nasal bone will result in outward and lateral displacement of the frontal bone segment. This is corrected by an additional osteotomy in the appropriate position.⁴ If a cerebrospinal fluid (CSF) leak is observed, it should be repaired immediately. Nasal packing and lumbar drainage may be necessary.³⁵

In the immediate postoperative period, complications may include bleeding, erythema related to vascular congestion, infection, skin loss or necrosis, and early patient dissatisfaction. Although mild bleeding may be expected after rhinoplasty, excessive postoperative bleeding is rare 0.9%.³⁵ In cases of troublesome or excessive bleeding, hypertension should be controlled, topical vasoconstrictors should be used, and DDAVP may be infused without the need for additional intervention.³⁶ Packing should be avoided. Early postoperative erythema may be a sign of vascular congestion (more common in revision cases), contact dermatitis or early infection. When erythema appears to be of infectious aetiology, early initiation of aggressive oral antibiotic treatment with is indicated. The skin over the nasal tip is delicate and at risk for necrosis, especially if the patient is a smoker. Its vascular supply can be compromised by excessive soft tissue trimming or cauterization over the ala, which may disrupt the lateral nasal artery, or with grafts that exert too much stretch or tension on the skin.

In the intermediate postoperative period (2 weeks to 2 months), additional complications may be noted such as prolonged oedema, delayed healing, patient concern/dissatisfaction, synechiae, osseous overgrowth, septal perforation, anosmia or 'late' presentation of a CSF leak. As the initial postoperative swelling subsides, subtle defects or irregularities may be noticed; more obvious abnormalities such as graft migration may be recognized and will likely need to be addressed surgically.³⁵ Patients may voice concern about the appearance of the nose because they cannot anticipate its final shape. Osseous overgrowths may occur at the site of bony resections or torn perichondrium and can be addressed with conservative rasping in a revision procedure. Septal perforations may occur, more commonly if bilateral septal tears were created and not repaired. Anosmia that

continues after splints are removed and expected postoperative swelling has diminished may be the result of cribriform plate injury. Clear rhinorrhoea that begins in the later postoperative period can be a late presentation of CSF leakage. CSF leaks are rare after rhinoplasty (1.17%) and usually seal on their own; however, patients with suspected leaks should be counselled to be vigilant in watching for signs of meningitis.

Late complications from rhinoplasty include nasal airway obstruction; various irregularities and deformities; implant exposure, extrusion or migration; scarring, silent sinus syndrome; enophthalmos; and mucocoele formation. Nasal obstruction may result from nasal valve collapse due to over-excision of the lateral crura of the LLCs, mid-vault collapse, aggressive medialization of the bony pyramid, or intranasal scarring in the form of synechiae. Saddle nose deformity may result from over-resection of the septum or bony dorsum. Open roof deformity results from failure to fill the midline defect created from dorsal hump resection with osteotomies and spreader grafts. Unsightly scarring may occur at the columellar incision with notching. Scarring may also occur at the site of percutaneous osteotomies with hyperpigmentation or tethering of the skin. Ethnic incongruity may occur by inappropriate planning in ethnic rhinoplasty. Autologous implants may become infected, migrate, extrude or be associated with skin contracture which must be addressed by removal and replacement with an autologous graft, if appropriate.³⁵

Secondary and tertiary rhinoplasty

It is not uncommon for the rhinoplasty surgeon to be presented with a patient that has had a prior rhinoplasty. It is estimated that between 2 and 30% of rhinoplasties result in secondary rhinoplasty.³⁷ The preoperative assessment described above for primary rhinoplasty, for the most part, applies in revision rhinoplasty. Of particular importance is the patient's previous surgical history, the nature of their concerns or dissatisfaction, the patient's social history and psychiatric history and, of course, a thorough objective evaluation of the patient's anatomy.

Evaluation

Operative history

It is important to attempt to obtain the operative record of any and all previous nasal surgeries as part of the preoperative evaluation of a patient presenting for revision rhinoplasty. The most common reasons for revision rhinoplasty are under-resection or over-resection of the structural cartilages. Consequently, eliciting information from the operative record and comparing it with the patient's physical exam may help the surgeon clarify whether the remaining cartilages need to be trimmed further or reinforced with grafts. Also, the extent of previous septoplasty or septal cartilage removed may help the revision rhinoplasty surgeon anticipate the need for various alternative cartilage grafting sources such as conchal or costal cartilage and whether these sources have previously been used.³⁷

Patient-reported issues

The surgeon needs to have a very clear and specific understanding of the source of the patient's dissatisfaction. Perhaps even more than with primary rhinoplasty, realistic expectations must be established. If the patient's expectations are judged to be unrealistic, it should be made clear and documented whether the surgeon believes that their desired results are attainable.³⁷ A number of patient-reported issues may be unique in the setting of prior surgery. Most of these relate to postoperative complications such as an inappropriately placed graft, graft migration, pain, infection or extrusion of implants, or nasal airflow obstruction secondary to nasal valve collapse from excessive cartilage resection.

Objective issues

A full examination and facial analysis should be performed. The height, width and symmetry of the nose should be assessed. The nasofrontal angle location and angulation should be noted. The dorsum should be visually examined and palpated for irregularity. The bony pyramid should be examined, noting its width, any asymmetries and whether there is an inverted-V deformity. The supratip area should be assessed for pollybeak deformity. Adequacy of tip projection and rotation should be assessed. Assessment of the LLCs should include evaluation for symmetry, bossae, alar rim collapse, the tip-defining points, width, position and columellar angle. Intranasal exam can identify valve narrowing or weakness, synechiae, or septal perforation from prior surgery and give the revision surgeon clues as to how much septal cartilage may be available for grafting.³⁷

Skin

Skin examination in the patient who has had a previous rhinoplasty is important as it can be thinned or scarred from previous surgery, perhaps more often with external approach or with extensive dissection. Also, previously placed alloplastic graft material may have resulted in scarring. The presence of scarring may increase the risk of skin necrosis and poor outcome. When the skin is extremely thin and scarring seems highly likely, it may be prudent to stage the revision procedure by placing a dermal graft and allowing it to heal prior to performing revision rhinoplasty so that a healthy skin flap can be elevated and redraped with a good chance of healing at the time of surgery.²⁸

Structural support

The full nasal exam and analysis as described above will provide insight about the structural support remaining in the previously operated nose. Often due to prior cartilage removal, there may be a lack of support to the tip and alae that may require grafting.

Operative principles and approach

One can argue that for revision rhinoplasty cases, the external approach is particularly advantageous, allowing full examination of the anatomy and assessment of what has been done in

the previous surgery unless the revision surgery is minor, which can be done through a closed technique. In revision cases, it is wise to err on the side of minimal resection, as the structural support has already been compromised by prior dissection and resection. Sources of cartilage for grafting should be considered and planned out prior to surgery. When autologous cartilage is not available, some surgeons use alloplastic grafting material such as solid silicone, Medpore or Gortex. If an alloplast is chosen, the patient should be counselled about the additional risks involved,³⁷ although the authors avoid alloplastic grafts and utilize autogenous materials.

When possible, the nose should be entered through the previous incision unless it has been poorly designed. This can avoid leaving the patient with an additional scar and allow the surgeon to resect and revise an unsightly scar. Dissection on the cartilage should proceed with caution, anticipating scarring from previous dissection. If a dorsal onlay graft is deemed necessary, the recipient surface should be flattened with a rasp to minimize irregularity. If costal cartilage is used, securing it with K-wire can limit warping and migration.³⁷ The septum is addressed as described previously. If the L-strut is significantly weakened, it can be reinforced with spreader grafts.

Tip reconstruction is performed, as described for primary rhinoplasty. If supportive grafts are necessary, one should begin by supporting the columella. Grafts can be secured in various ways including by drilling a K-wire into the maxilla just lateral to the maxillary crest and securing the graft to the wire. Occasionally, when there has been excessive resection of the lateral crura, supportive grafts may not be enough and alar replacement is required. Conchal cartilage has a natural curve that mimics the curve of the LLCs and is a good choice for this indication. The soft tissue is dissected, creating a tissue pocket at the pyriform aperture and the graft is placed in this pocket. An anchor graft may be used in revision cases for alar retraction: conchal cartilage is shaped to look like an upside-down anchor with the sides at each LLC and the middle secured to the septum or a columellar graft. Previous rhinoplasty may create a pinched dome appearance. A dome spreader graft may address this by securing a small piece of cartilage between the medial crura.²⁹

Complications

The spectrum of complications that may occur after revision rhinoplasty is essentially the same as that for primary rhinoplasty, although postoperative oedema may take longer to resolve and there may be a higher risk of skin necrosis in previously dissected tissue.

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CHAPTER 73

Genioplasty

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Introduction

The chin is one of the most obvious facial structures. Chin size, shape and position play an important role in facial appearance and balance. Therefore surgery to the chin can play an important part in the correction of facial discrepancies. Surgery is either in isolation where the dental occlusion is accepted or as part of an orthognathic treatment plan, including single or bimaxillary jaw surgery and other soft tissue procedures (Figure 73.1).

An important component of nasal aesthetics and facial balance is the position of the chin. An underprojected, or weak, chin can create the appearance of a larger nose than is actually present. This was noted as early as 1928 when Aufricht described augmentation of the chin with osteo-cartilagenous nasal hump. Advancement genioplasties have been used successfully to treat sleep apnoea in retrognathic patients.¹ In 1942 Hofer first described the osseous horizontal sliding osteotomy for the correction of microgenia via an extraoral approach.² In 1957 Trauner and Obwegeser described osseous genioplasty via an intraoral approach.³ This was when Obwegeser was working in Trauner's unit in Graz. They described the bilateral sagittal split osteotomy in the same year.^{4,5}

The chin, like the face, should be considered in four dimensions: vertical, anterior posterior, transverse and in function.

Minor chin deformities can be camouflaged by the injection of fat or fillers. The use of alloplastic implants allows the correction of mainly anterior posterior deformities. Although now a very common procedure, alloplastic materials have been used to augment the chin since the 1940s.⁶ Commonly used materials include porous polyethylene, hydroxyapatite, silicone and PEEK (polyetheretherketone).

Though it is possible to augment the chin with computer-aided design/manufacture (CAD/CAM) bespoke implants, in the long term there are risks of migration of the implant and resorption of the underlying bone. (Figure 73.2). Osseous genioplasty allows stable three-dimensional correction of chin deformities with minimal morbidity.

The aesthetic goal of any surgery on the chin should be to improve or maintain facial balance to the patient's satisfaction. The surgery should be performed transorally with minimal complications.

Surgical anatomy

Osteotomy cuts are carried out below the lower anterior teeth and need to be placed 4 mm below the tooth apices, noting that the maximum average length of lower incisors is 23.5 mm and canines 27 mm.⁷

The mental nerve carries sensory branches to the chin and lower lip. Care must be taken to identify and protect this nerve during surgery. It is important to note that just before it exits the mental foramen, the mental nerve is 5 mm anterior and inferior to the foramen. If an osseous genioplasty is performed, the incisive branches to the lower incisor teeth are sacrificed and the patient should be warned of the resultant altered sensation in the lower incisor teeth and surrounding gum. Interestingly, even though the nerve is usually divided, normal sensation seems to return after about a year.

The mentalis muscles are paired elevators of the central lower lip that originate from the mandible at the level just beneath the attached gingiva of the central and lateral incisors.⁸ Careful surgery to prevent excessive stripping of soft tissues will decrease the risk of lip ptosis. Accurate planning of incision and careful dissection leaving enough superior bulk of mentalis muscle allows repair of the muscle and a two-layer closure.

Any genioplasty has to ensure bone-on-bone contact between segments to allow adequate bony healing.

Aesthetics and functional assessment

Patient assessment

The patient assessment should start with a discussion to allow the surgeon to have a through understanding of the patient's overall expectations. Unrealistic expectations or multiple

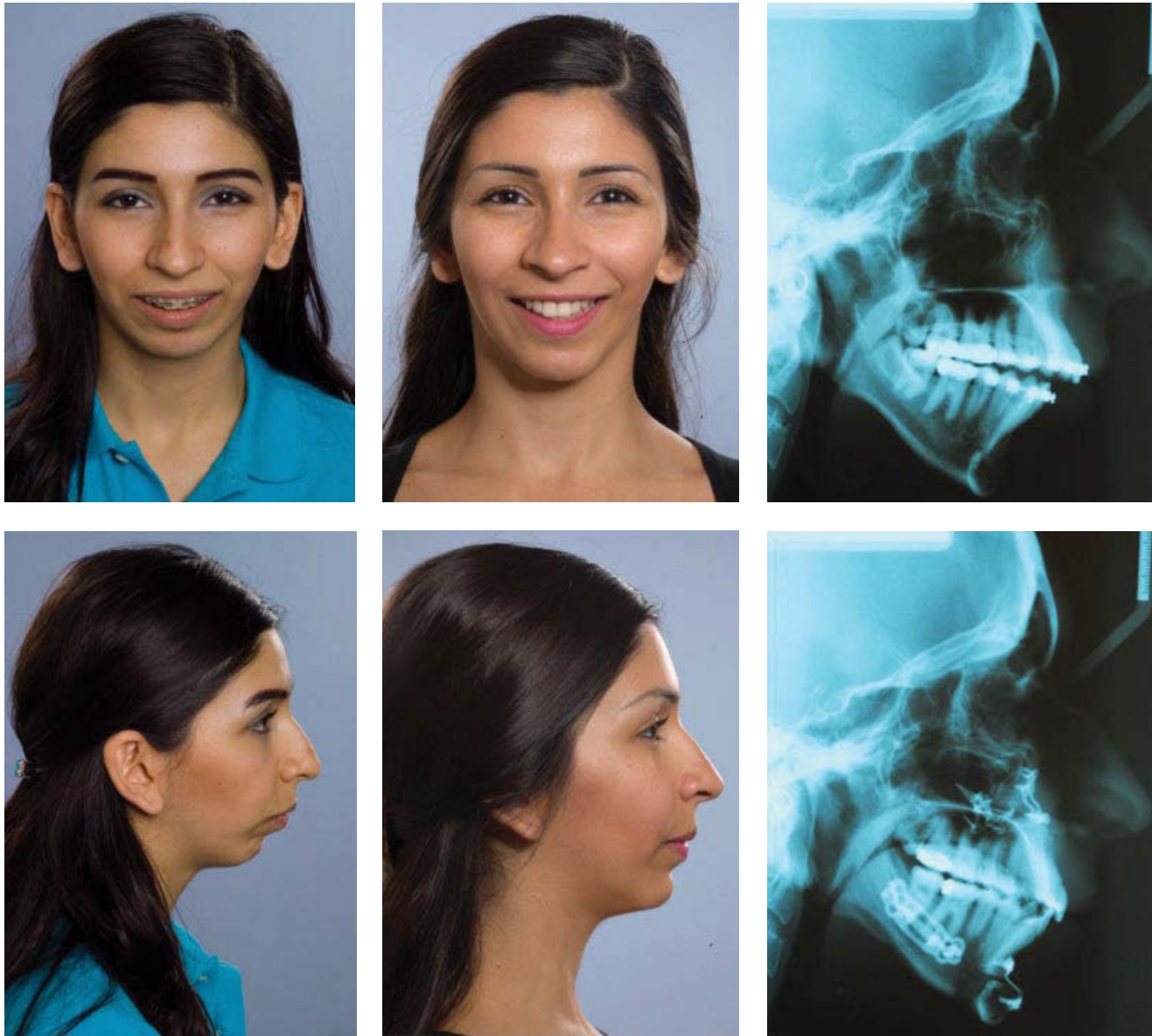


Figure 73.1 Bimaxillary osteotomy and genioplasty.

previous cosmetic procedures may be a sign of body dysmorphic disorder. The medical history should be explored and if the patient is a smoker, smoking cessation advice should be considered. At the very least smoking should be stopped in the immediate postoperative period.

The chin cannot be considered in isolation from the face and has to be examined in all three planes of space. The assessment should include the dental occlusion and the relationship of the maxillary and mandibular skeletal bases. The nose should also be examined and its relationship to the face explored, as any facial discrepancies may benefit from orthognathic surgery or a rhinoplasty, either instead of or in combination with a genioplasty.

The chin plays an important role in the overall facial harmony and balance. A small chin can be described as ‘microgenia’,

whereas a large chin is called ‘macrogenia’. Small or large chins can be deficient in any of the three planes of space but should not be confused with a chin that is normal in size but has a posterior chin point due to a retrognathic mandible or one that is normal in size but more prominent due to excess soft tissue.

From the frontal view the face can be examined for transverse discrepancies, facial asymmetries, the degree of lip competence and the form and depth of the labiomental groove. A deep labiomental groove may be a result of overactivity of the mentalis muscles. This can also cause dimpling of the chin button. Injection of botulinum toxin A can help with these issues.

The relationship of the osseous chin to the overlying soft tissues should be examined. Any osseous genioplasty or alloplastic chin augmentation would be expected to produce only 80–85%



Figure 73.2 Bone resorption from implant.

of the total advancement in soft tissue changes. A chin moved posteriorly or vertically would see soft tissue changes of 90%.

The facial midline should be determined and how this midline relates to the nose, philtrum, upper dental centre line, lower dental centre line and chin point. In complex asymmetry cases there is often no true facial midline as a pan-facial asymmetry is often present.

In the lateral view the anterior posterior relationship and chin point can be assessed. The dental occlusion is examined and its relationships to the mandibular and maxillary skeletal bases. Riedel's line (Figure 73.3) allows a quick facial analysis of the chin point. The line connects the most prominent points of the upper lip, lower lip and soft tissue chin point. If the chin is posterior to the line it suggests microgenia in an anterior posterior direction which can be corrected with an advancement osseous genioplasty or implant. If the chin extends anterior to this line then it suggests anterior posterior macrogenia, which may require a reduction or a clockwise rotation of the chin point.

In examining the whole face a 'zero meridian line' described by Gonzalez⁹ (Figure 73.3) can be applied. Here a line perpendicular to the Frankfort horizontal, passing through soft tissue nasion to measure the position of the chin. The chin should be on this line or just posterior of the 'zero meridian line' for good facial balance. Individual preferences should be taken into account; men, for example, often associate masculinity with a strong chin, whereas women have a preference for either a soft or stronger chin, depending perhaps on cultural differences.

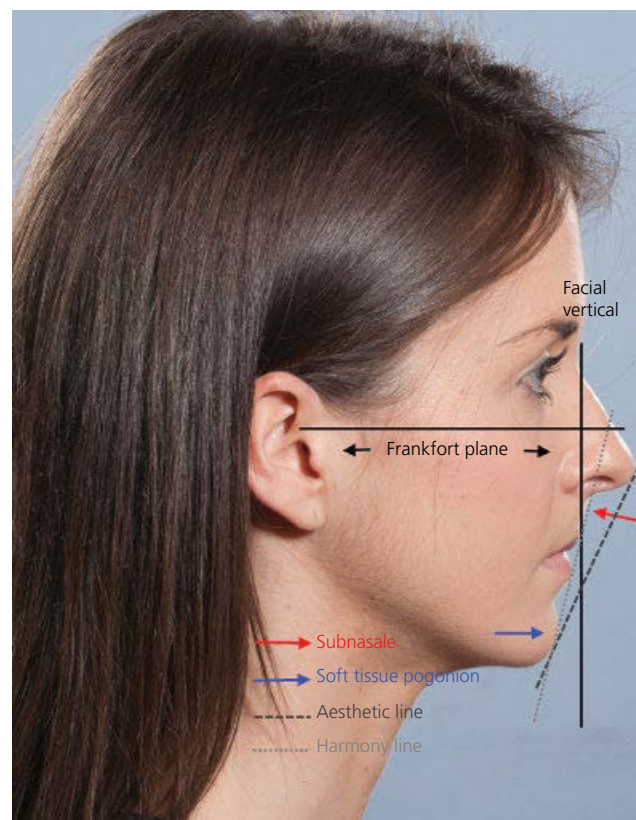


Figure 73.3 Dotted line is Riedel's (harmony) line. The distance from the soft tissue chin to a line drawn perpendicular to Frankfort plane should be within the range 0 ± 2 mm.

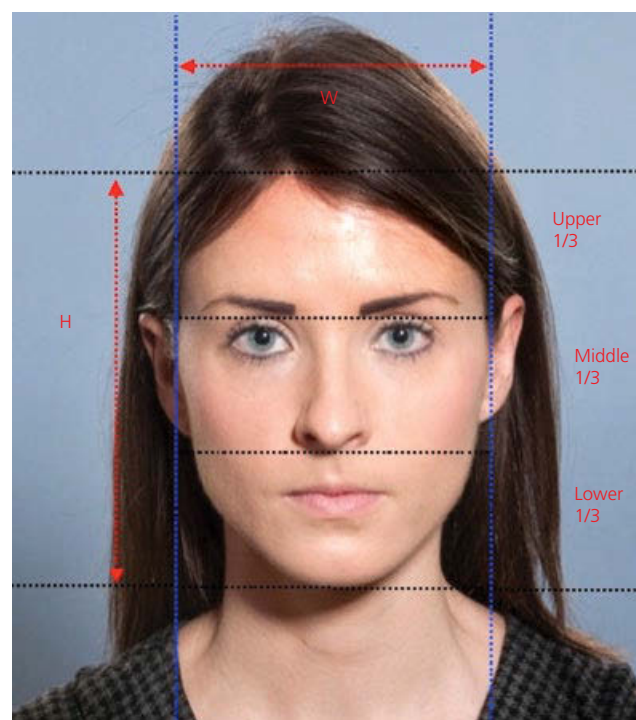


Figure 73.4 Facial proportions, frontal view.

Facial thirds (Figure 73.4) are used as a guide to the overall facial harmony and any deficiency or excess in lower anterior facial height.

The following medical photographs should be taken as part of the clinical examination:

- Full frontal view at rest
- Full frontal view in smile
- Right lateral view

- Left lateral view
- Right three quarters view
- Left three quarters view
- Worm's eye
- Occlusal views if required.

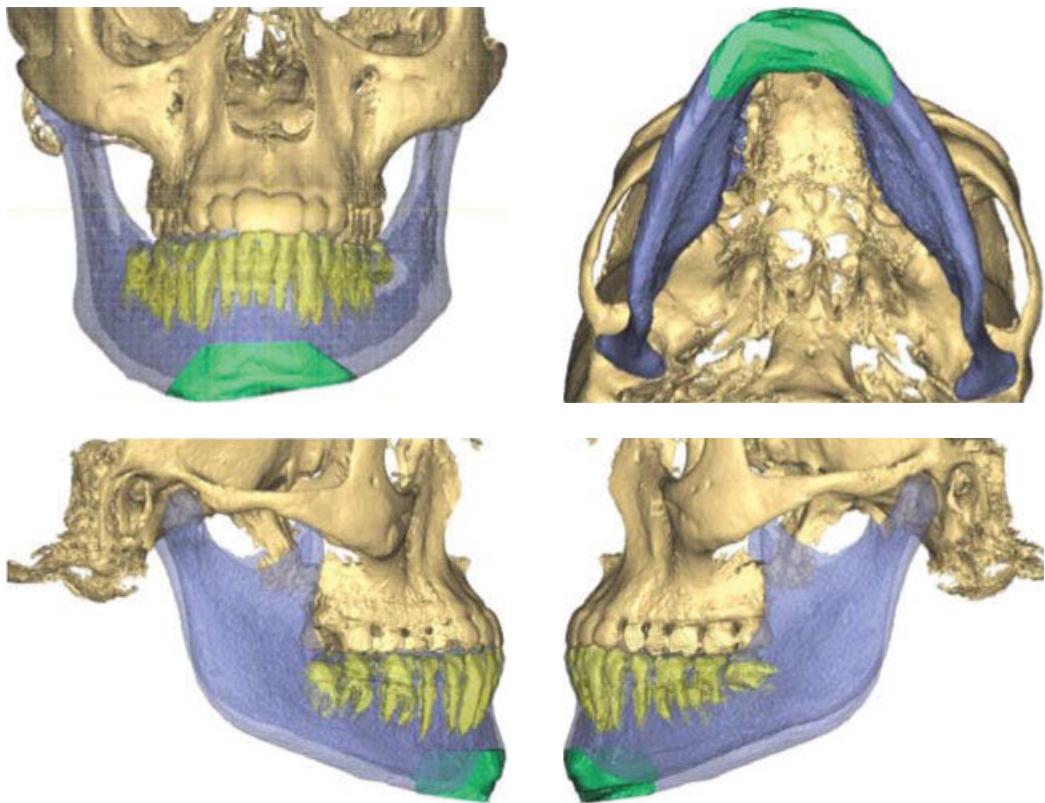
Radiographs should be obtained either as a two-dimensional set or a cone beam computed tomography (CBCT):

- *Two-dimensional:*

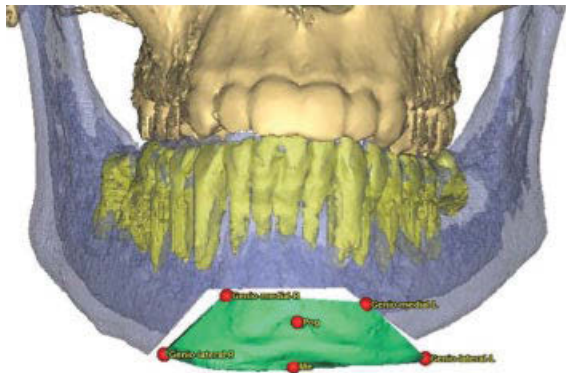


Case ID: ME13-ITE-IJI
Case report version: 2.0
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Surgical plan: Planned genioplasty osteotomy



Surgical plan: Movements analysis



Name	Left/Right (mm)	Ant/Post (mm)	Up/Down (mm)
Me	3.5 L	0,0 Post	1.7 Down
Pog	4.6 L	0,1 Post	1,7 Down
Genio-medial-L	4.6 L	1.2 Post	2,8 Down
Genio-lateral-L	2.5 L	2.2 Post	3,9 Down
Genio-lateral-R	2.6 L	2.0 Post	0,2 Up
Genio-medial-R	4.5 L	1.0 Post	0,7 Down

Figure 73.5 CAD planning.

Guide design: Mandible guide

- Flanges
- Fixation hole diameter: 1.7 mm (suitable for 1.1 mm drill, 1.5 mm screws)
- Fixation holes are intended for temporary fixation of the guide

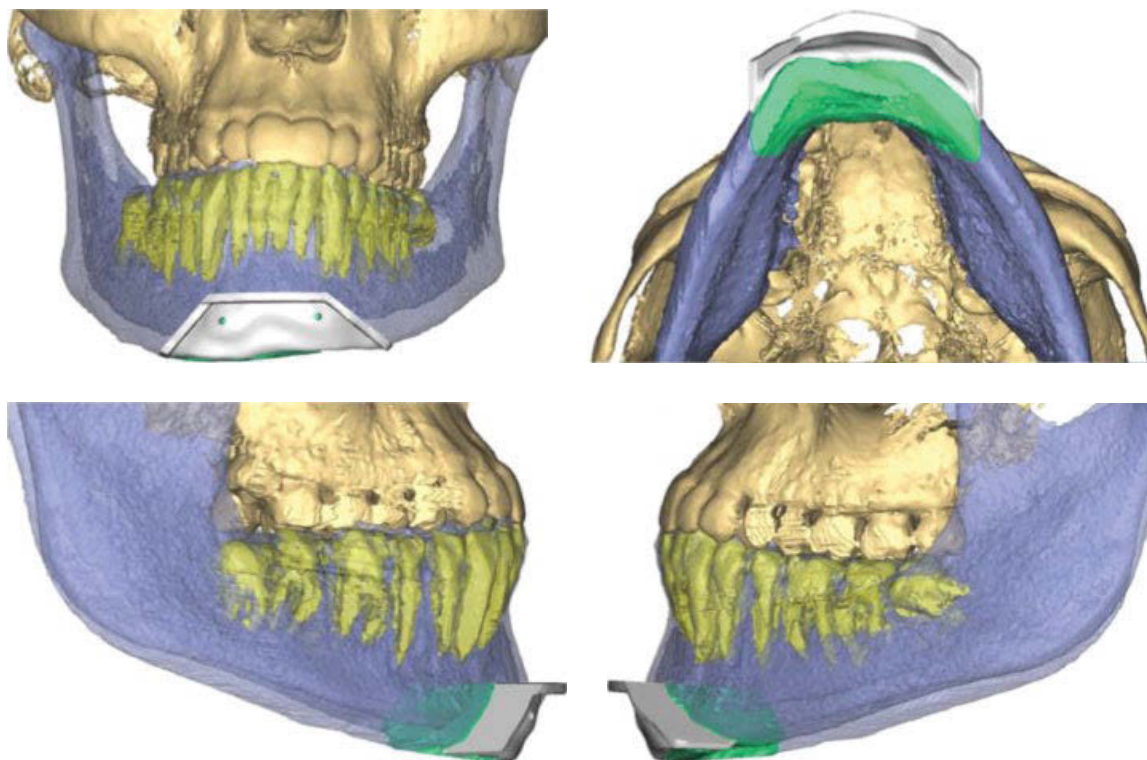


Figure 73.5 (Continued)

- Orthopantomogram to assess the bony height, position of the mental nerve and possible tooth pathology.
- Lateral cephalogram for 2D cephalometric analysis.
- **CBCT** (Figure 73.5): This allows CT of the bony facial skeleton at a lower radiation dose than a conventional spiral CT scan. The skull can be visualized in three dimensions allowing a true assessment of the facial discrepancy. Three-dimensional cephalometric analysis can be carried out. Once the virtual skull has been segmented then virtual surgery can be carried out. With CAD software, surgical guides, models and custom implants can be fabricated. If needed, 3D models of the skull allow patient-specific implants to be made in the laboratory. Orthognathic planning software as well as allowing the design and planning of the osteotomy also allows the surgeon to visualize the inferior dental nerve and tooth roots. Soft tissue algorithms allow some indication of the soft tissue changes expected.

Operative principles and approach

Osseous genioplasty

The surgery is carried out under general anaesthesia with a nasal endotracheal tube to allow good visualization of the face.

If full-face exposure is needed in complex facial asymmetry cases where the genioplasty is being carried out with other facial procedures then a submental intubation can also be considered.¹⁰

If combined with another orthognathic procedure then the genioplasty should be carried out after any mandibular osteotomies with the patient in intermaxillary fixation while the genioplasty is being carried out.

The oral cavity is rinsed with chlorhexidine digluconate mouthwash. The surgical field is infiltrated with local anaesthesia with a vasoconstrictor (2% lidocaine (lignocaine) with 1:80 000 adrenaline) or approximately 40 mL of tumescent solution. Tumescent solution is mixed in a 250 mL bag of normal saline and contains 1 mL of 1:1000 adrenaline, 1 vial (40 mg) triamcinolone acetate, 20 mL of 5% Marcaine and 1 vial hyaluronidase. It has been shown to improve the surgical field and postoperative swelling in craniofacial surgery and is used routinely in our unit.¹¹ Low-power cutting diathermy or a blade is used to make an incision just through the mucosa at least 5 mm below the attached mucosa from lower canine to lower canine (Figure 73.6a). The submucosal incision is deepened in the midline through periosteum to bone at a 45° angle, making sure that both mentalis muscles are divided, allowing a satisfactory

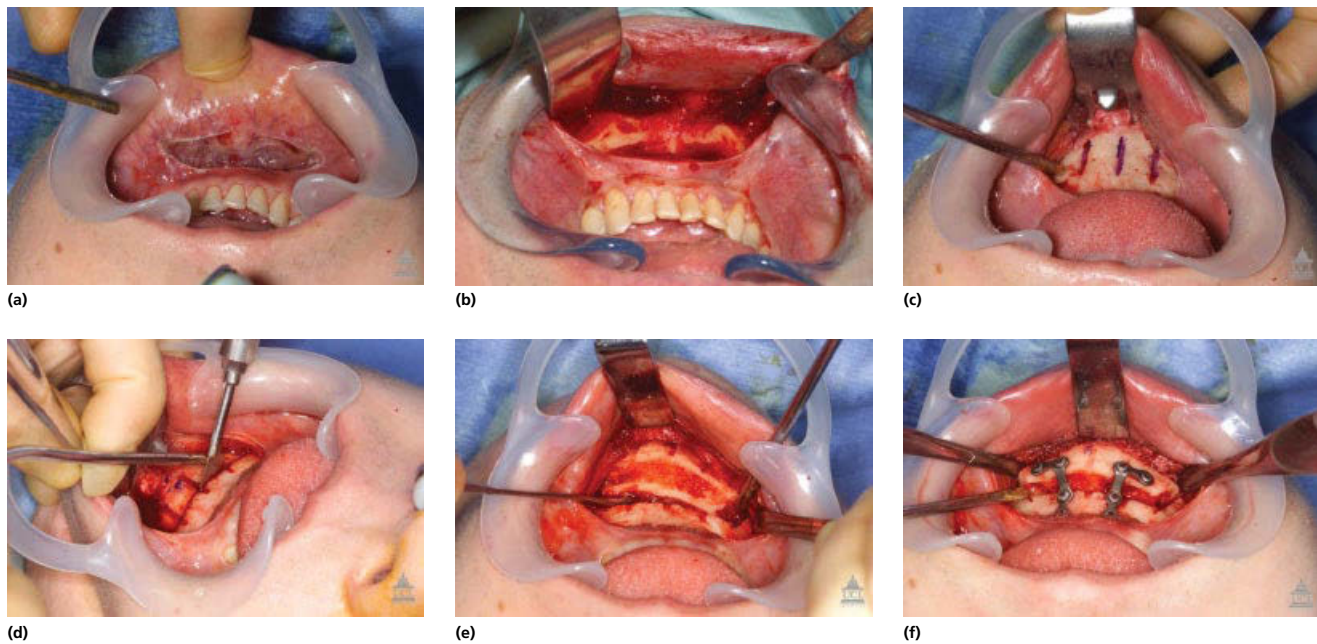


Figure 73.6 Advancement genioplasty surgical stages.

muscle repair on closure (Figure 73.6b). The superior edge of the flap is elevated to allow an easier mucosal closure.

A subperiosteal dissection is carried out, starting at the midline and working out laterally, identifying the neurovascular bundle exiting the mental foramen. The mental nerve is then identified and protected. The dissection does not need to include the whole chin, just enough to visualize and perform the osteotomy cuts and to give access for the fixation while preserving vascular supply.

Three vertical reference marks are made with a water-cooled fissure bur to allow accurate horizontal repositioning of the chin post osteotomy (Figure 73.6c).

The osteotomy should be performed 5 mm below the apices of the roots of the lower incisors and 5 mm below the mental foramen.

Osteotomy cuts are carried out with a water-cooled reciprocating saw blade (Figure 73.6d) or a piezoelectric saw if available. The angulation is important, as subtle changes will have profound changes in the vertical chin point position and anterior mandibular height. Care must be made to ensure that both the anterior and posterior cortices are osteotomized to avoid an unfavourable split. The final completion of the osteotomy is carried out with an osteotome if necessary and separation by the placement of a Howarth's periosteal elevator in the osteotomy cut and turning 90° (Figure 73.6e).

The mobile chin is then mobilized into the planned position while maintaining its lingual soft tissue and muscle attachments. If an advancement is planned, the surrounding soft tissues including the muscles of genioglossus and geniohyoid are stretched. Fixation is completed with either plates bent at the time of surgery or preformed 'Lindorf'-type plates (Figure 73.6f).



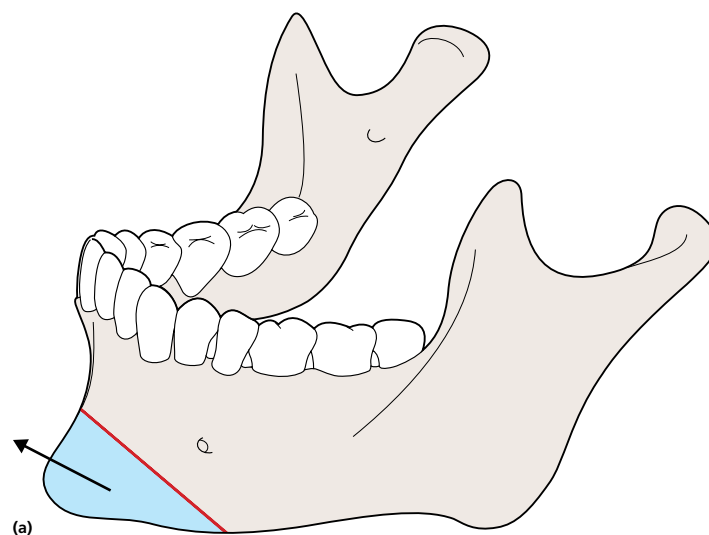
Figure 73.7 Postoperative strapping.

The osteotomy can also be stabilized by bicortical screws through both segments. If screws are used then it is helpful to countersink them into the most anterior plate of bone and for there to be at least two screws to prevent rotation. Any bony steps or spurs are then removed and smoothed.

Closure is carried out in layers with particular attention to the repair of mentalis muscle. At the end of surgery chin strapping is applied to prevent lip ptosis and postoperative swelling (Figure 73.7).

Sliding genioplasty

The sliding genioplasty and variations of the theme is our most commonly performed osseous genioplasty (Figure 73.8a–c).



(b)



(c)

Figure 73.8 (a) Sliding advancement genioplasty. (b) Preoperative. (c) Postoperative.

Anterior posterior advancements or reductions of the chin can be performed. Care has to be made with the angulations of the osteotomy cut relative to the occlusal plane as the chin point will move superiorly, with a reduction of the lower face height with an increasingly more steep osteotomy cut (Figure 73.9). Conversely, with a sliding anteroposterior reduction genioplasty there will be an increase in lower face height. This procedure also results in irregularities in the

lower border of the mandible, notching in advancement and a spur in an anteroposterior reduction. For an increase in the vertical height of the anterior mandible with or without an advancement, the mobile osteotomized segment can be repositioned inferiorly, creating a gap. If large, the gap is grafted with either autologous bone or bone substitutes. To reduce the vertical height of the anterior mandible a double osteotomy cut can be performed and the middle segment removed.

Double osteotomy cuts also allow greater advancement by a creating a double step appearance (Figure 73.10). When performing a double osteotomy it is important to make sure that all cuts are complete and parallel prior to separation and that the most inferior segment is separated first. Failure to do so will result in the surgeon trying to make cuts on a mobile segment.

When the chin is set back, a small ledge on the lingual aspect is created that needs to be reduced to prevent the patient noticing a small palpable step on the border of the mandible (Figure 73.11).

A reduction genioplasty moving the chin posteriorly can produce unfavourable submental and cervical changes. The authors prefer the inferior posterior chin point repositioning osteotomy to prevent these changes.

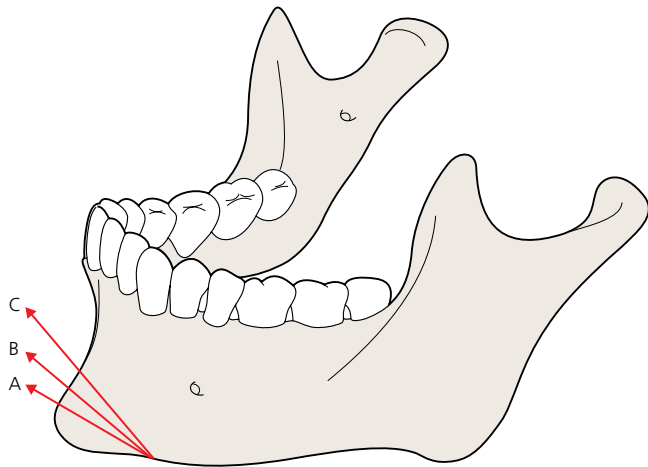
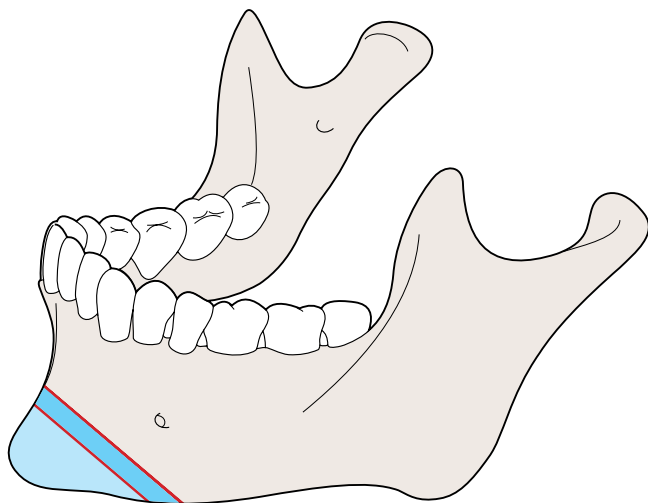


Figure 73.9 Sliding advancement genioplasty.



Michelet genioplasty

The Michelet genioplasty¹¹ is a useful osteotomy in patients in need of maximum chin point advancement while still maintaining bone-to-bone contact (Figure 73.12a–c). The osteotomy cuts are performed with the aim of keeping a cortical square of anterior labial bone attached to the proximal mandible and a posterior lingual cortical square attached to the mobile osteotomized segment. This is then mobilized and the inferior mobile segment is placed in front but in contact with the superior anterior labial cortical square. The bony cortical squares are now reversed from their normal anatomical position (Figure 73.13). The cortical squares are secured with a titanium bicortical screw, giving maximum chin point advancement with minimal change in the height of the lower facial third. If a reduction of the lower facial third is required, lateral segments can be removed.¹²

Jumping genioplasty

The inferior mobile segment is advanced anteriorly and placed in front of the mandible, moving the chin in an anterior position but decreasing lower facial height (Figure 73.14).

Centralizing genioplasty

In facial asymmetries a centralizing genioplasty can be used to centralize the chin point to the midline in a transverse dimension (Figure 73.15). Wedges can be removed asymmetrically to correct the asymmetry if there is a vertical component.

Box genioplasty

A box genioplasty can be used for simple advancements and to correct a transverse discrepancy (Figure 73.16). For the correction of a transverse discrepancy a box osteotomy is cut with a rectangular wedge removed to allow the mobilized

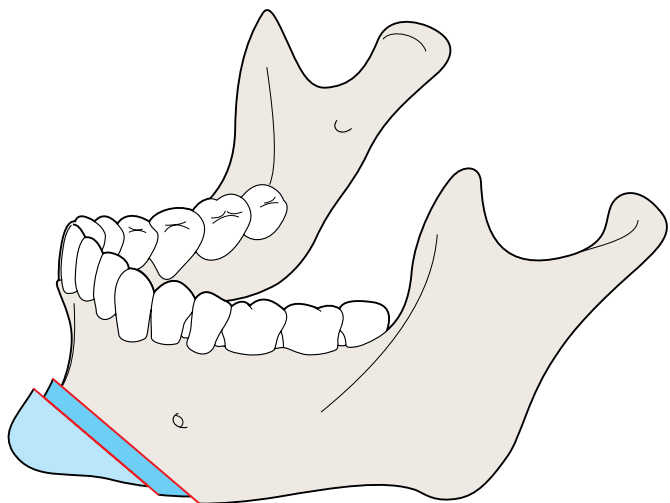


Figure 73.10 Double sliding advancement genioplasty.

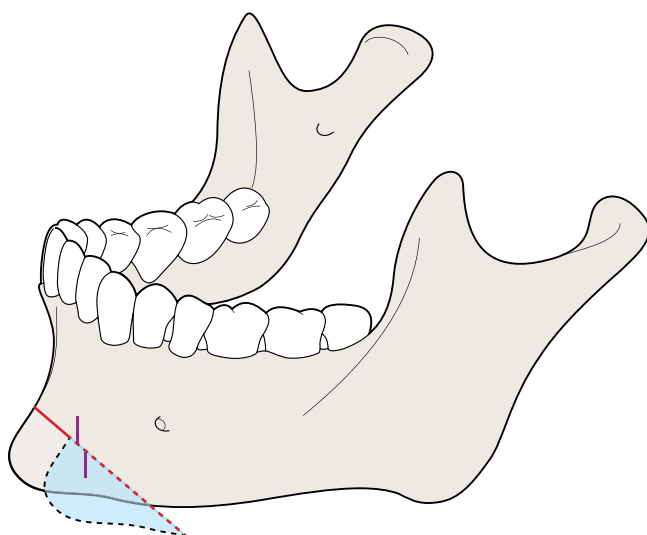


Figure 73.11 Sliding reduction genioplasty.

anterior segment to be centralized, bringing the chin midpoint to the midline. The wedge is then used as a free bone graft to the resulting gap. All is held with titanium plates and screws and any bony spurs reduced. With the box osteotomy it is easier to make your horizontal bone cut parallel to the occlusal plan and you have more control of the vertical position of the chin point. Additionally, your posterior vertical bone cuts are made in a sagittal plane, preserving the continuity of the lower border and avoiding the irregularities that occur with a conventional sliding genioplasty. This is the preferred method of the authors for a simple advancement.

Inferior posterior chin point repositioning osteotomy

This osteotomy allows the chin point to be repositioned posteriorly, giving the illusion that the mobile osteotomized segment has been reduced without any soft tissue changes to the submental and cervical region. A segment of anterior mandible is removed with the posterior base greater than the anterior apex. Increasing the width of the segment removed will also have an effect on lower face height (Figure 73.17).

The T genioplasty

The T genioplasty is useful in dealing with transverse discrepancies, the broad or narrow chin and asymmetries.¹³ The chin is divided in the sagittal plane with either two parasagittal cuts when the chin is to be narrowed or a single midline sagittal cut with an interpositional bone graft to widen the chin point (Figure 73.18). This technique is useful in cases of gender reassignment surgery.

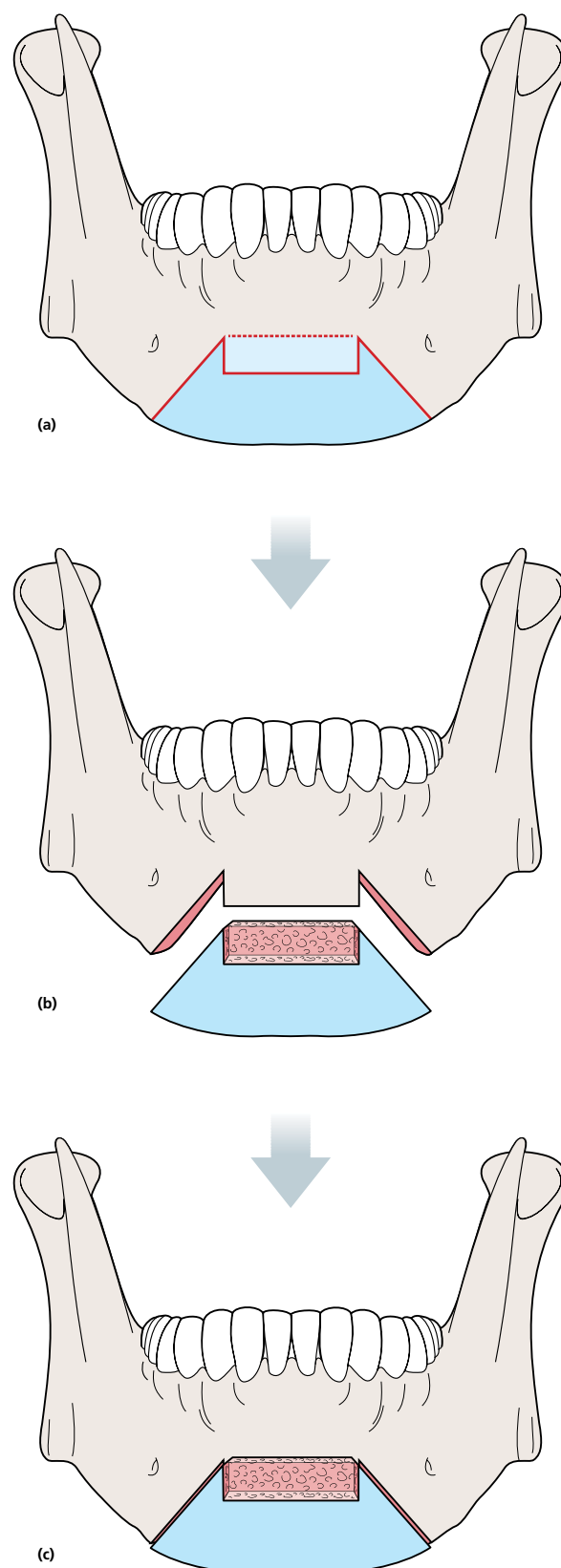


Figure 73.12 Michelet genioplasty.

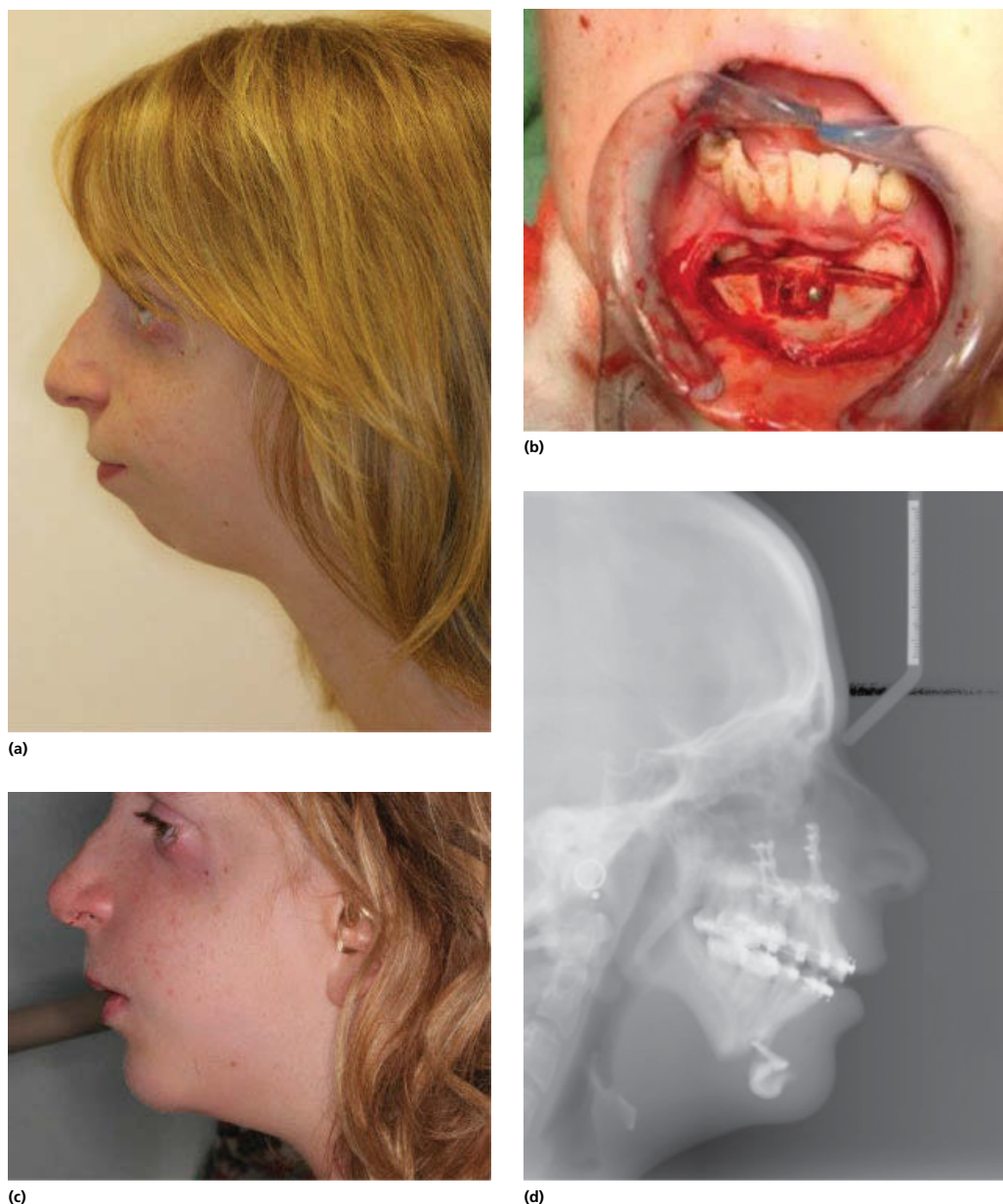


Figure 73.13 (a) Preoperative Michelet genioplasty. (b) Bicortical screw fixation for Michelet genioplasty. (c) Postoperative Michelet genioplasty. (d) Postoperative lateral cephalogram. Michelet genioplasty.

Extended or wing genioplasty

This is a lower border osteotomy recently described by Triaca.¹⁴ The osteotomy runs the complete length of the lower border of the mandible, with the buccal cut higher than the lingual. This allows an increase in vertical height of the mandible. (Figure 73.19).

The shield genioplasty

Described by Triaca, this procedure is designed to give the lower lip more support by making the buccal bone higher than the

lingual cut.^{15,16} This avoids creating a deep labiomental fold when advancing the chin point.

Combination procedures

In complex chin deformities seen in patients with severe facial asymmetries or craniofacial conditions, a combination of different osseous genioplasty osteotomies may be required to improve the facial profile.

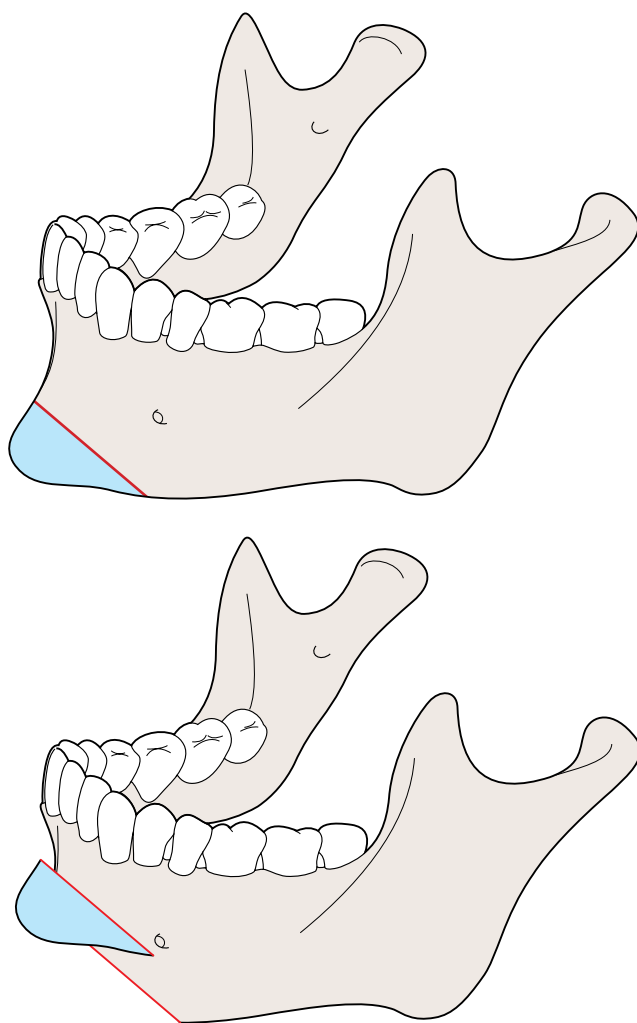


Figure 73.14 Jumping genioplasty.

Alloplastic augmentation

The majority of alloplastic implants are preshaped in a variety of sizes designed to fit over the chin. An alloplastic implant is most suitable for microgenia, where there is an anterior posterior deficiency, but generally less than 5 mm. An implant larger than this is at risk of symphyseal erosions over time.¹⁷

If appropriate, the implant can be custom made. This requires a CT scan – either conventional spiral or CBCT of the jaw. The implant is then either made on an acrylic model produced from the DICOM data (Digital Imaging and Communications in Medicine) or on a virtual 3D model using specialized CAD software.

Silicone is a commonly placed implant, but solid silicone does not allow tissue in-growth so the implant is easily removed.

Porous polyethylene (Medpor) implants are commonly used in craniofacial reconstruction and augmentation. The implants

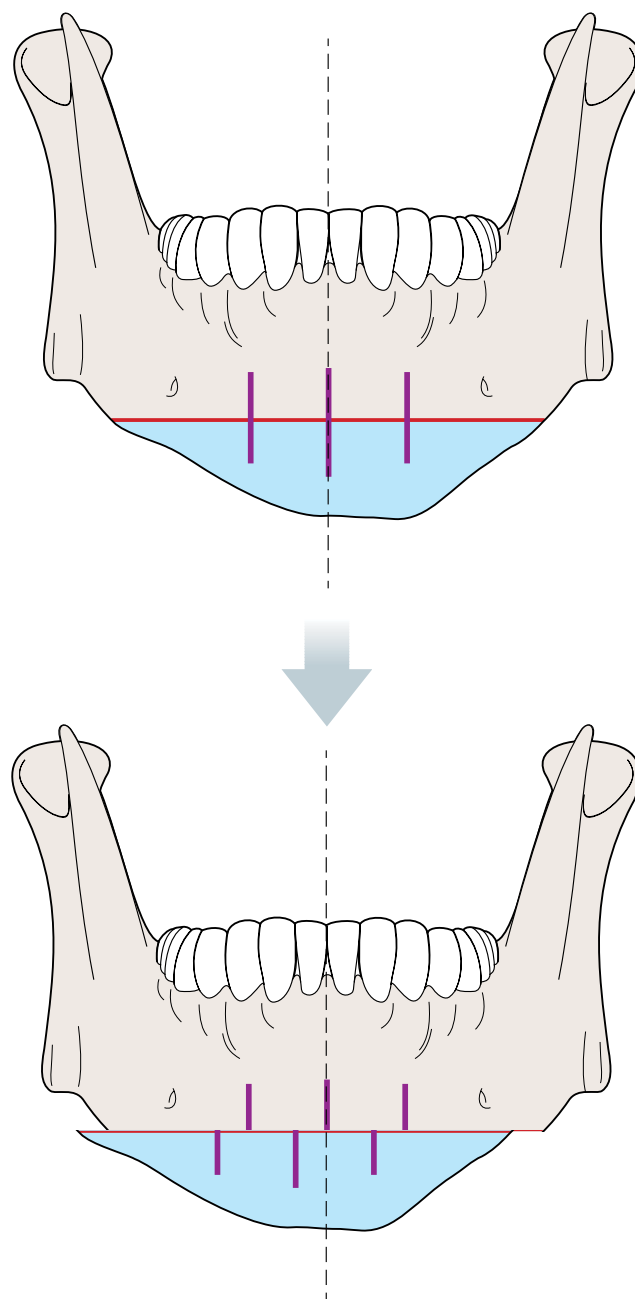


Figure 73.15 Centralizing genioplasty.

have a pore structure allowing fibrovascular in-growth and integration of the patient's tissue.

PEEK (Polyetheretherketone) is an implant material used in the construction of custom-made patient-specific implants with qualities similar to bone. The alloplastic implant can be placed via either an intraoral or an extraoral approach. The intraoral approach is as the operative technique for the osseous genioplasty with the aim of creating a wide subperiosteal pocket. The soft tissue and musculature needs to be adequately released to allow the alloplastic implant to seat well, especially along the lower border of the mandible. The implant is held with titanium

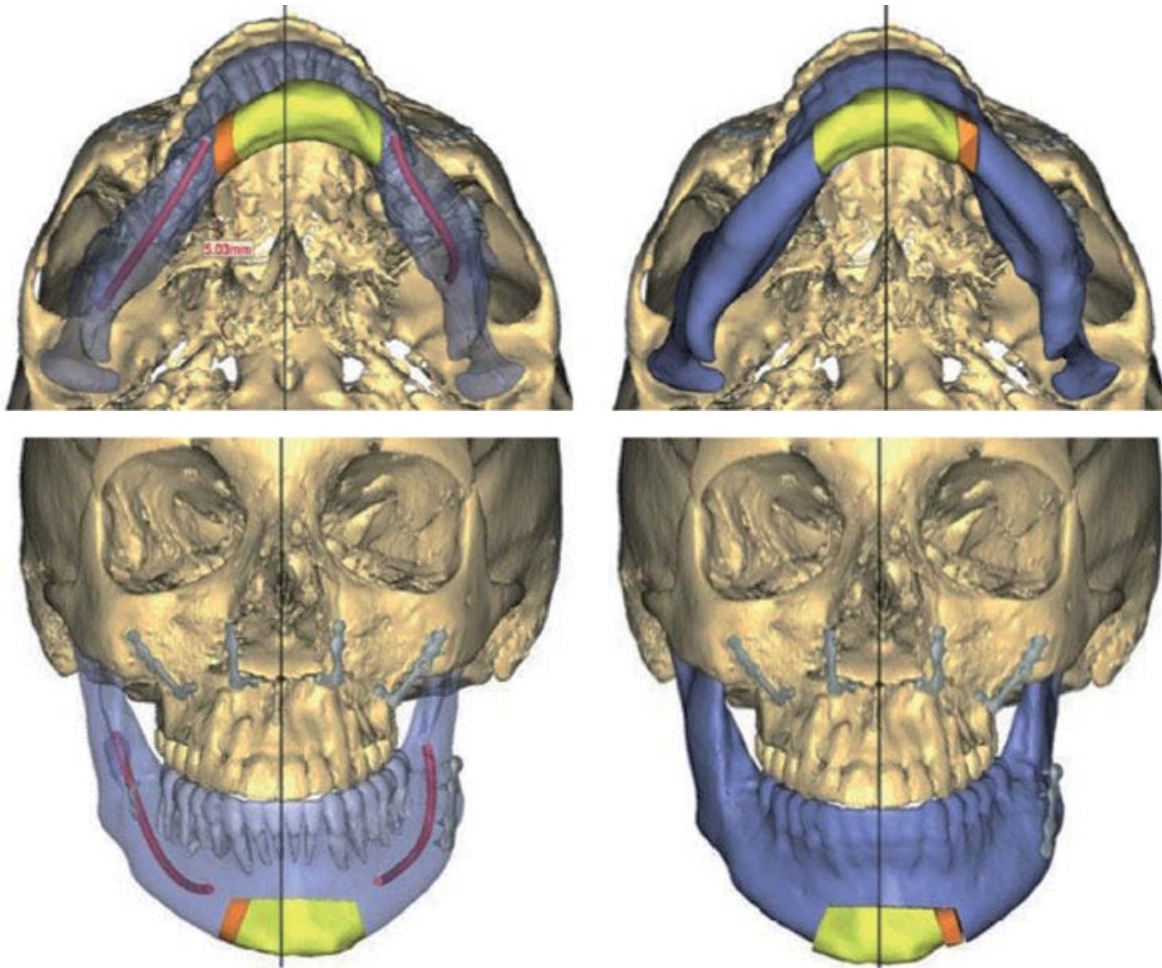


Figure 73.16 Box genioplasty.

screws. Inadequate fixation may lead to displacement, wound dehiscence and extrusion.

The submental approach is normally performed under general anaesthesia with a nasal endotracheal tube to allow visualization of the face. The incision site is infiltrated with 2% lidocaine with 1:80 000 adrenaline. The incision is made just inferior to a natural submental crease. The dissection is developed superiorly to the inferior surface of the chin. The periosteum is incised and a subperiosteal pocket developed large enough to fit the implant. The mental nerve is identified and avoided. The implant is placed inferior to the mental nerve. Once haemostasis is obtained the sterile implant is placed into the pocket with minimal handling. Titanium screws are used to fix the implant in place and the wound is closed in layers.

Autologous augmentation

An onlay graft of autologous bone or costal cartilage may be required when a conventional osseous genioplasty or alloplastic implant has failed. The stability of onlay bone and cartilage grafts is questionable.

Other procedures that change the chin

Changing the vertical position of the maxilla will result in auto-rotation of the mandible. The chin point will come forward with impaction of the maxilla and go back with inferior repositioning of the maxilla (Figure 73.20).

In a patient with an increased overjet and a chin point in the correct position, a total subapical osteotomy can bring about favourable changes to the appearance of the chin (Figure 73.21).

Complications

Complications following alloplastic augmentation and the osseous genioplasty include:

- the unhappy patient,
- sublingual haematoma resulting in airway embarrassment,
- poor aesthetics,
- paraesthesia of the lower lip and chin,
- lower lip ptosis,

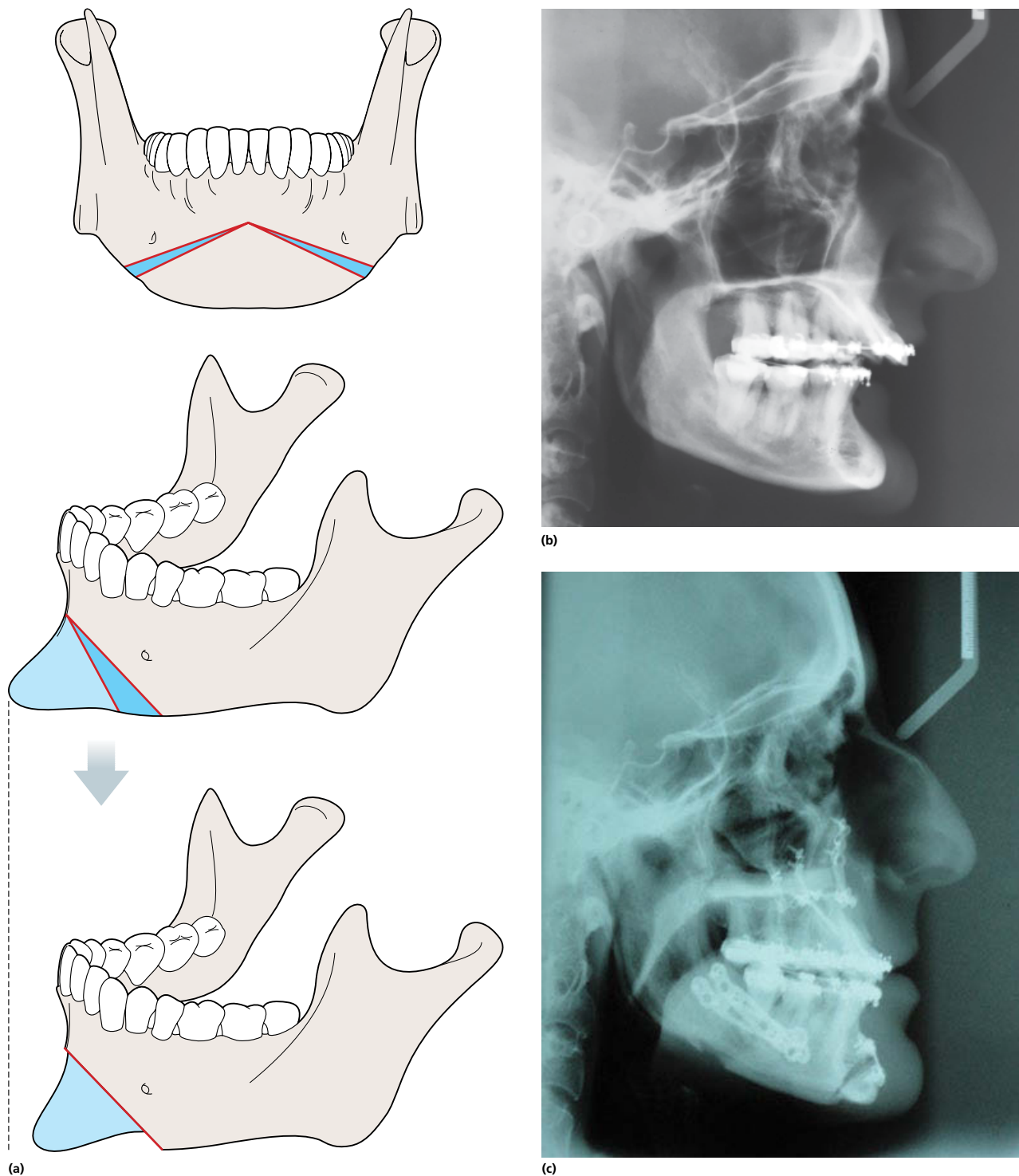


Figure 73.17 Inferior posterior chin point repositioning osteotomy with a rotating reduction genioplasty. (a) Diagrams. (b) Pre-operative lateral cephalogram. (c) Post-operative lateral cephalogram.

- infection,
- bone resorption,
- loss of the osteomized segment, and
- wound dehiscence and extrusion with implants.

Understanding the patient's expectations, the ability to view in three dimensions and a surgical plan will hopefully prevent the unhappy patient. The incidence of neurosensory deficiency in the distribution of the mental nerve following an isolated

genioplasty is in the region of 10–12%, but can be as high as 70% when performed in combination with a bilateral sagittal split osteotomy.^{18,19} Direct transection of the nerve requires a microsurgical repair.

Bone resorption may be seen in large alloplastic implants or if excessive stripping has been carried out of the mobile osteomized chin segment, compromising the soft tissue pedicle.

An infection of the surgical site should be treated aggressively but may result in the removal of the implant, bone or mode of fixation resulting in a later reconstruction.

Wound dehiscence can be secondary to infection but may also occur if the pocket created for the alloplastic implant is too small

and again will result in removal of the implant. Extrusion may follow dehiscence. Excessive stripping of the soft tissues compromising the vascular pedicle may result in avascular necrosis and may require a future alloplastic implant or free bone graft.

Lip ptosis is related to inadequate repositioning and repair of the mentalis muscles prior to soft tissues closure. Correction of the lip ptosis requires soft tissue and mentalis muscle resuspension with bony tunnels or anchors.

The implant or mobilized osteotomized segment can become dislodged due to trauma or inadequate retention. This may

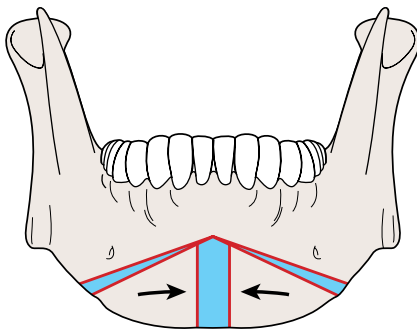


Figure 73.18 T genioplasty.

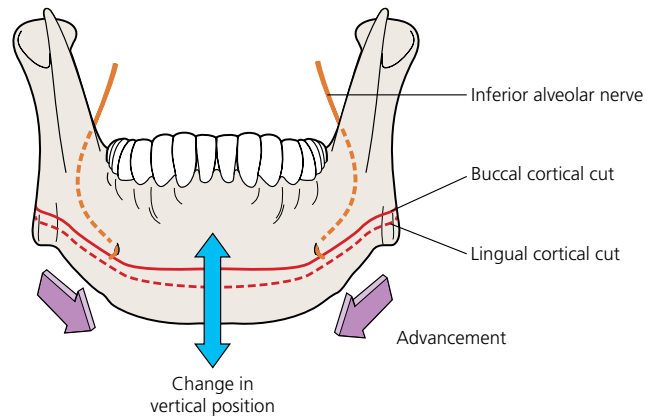


Figure 73.19 Shield genioplasty.



Figure 73.20 Maxillary Le Fort I inferior repositioning.

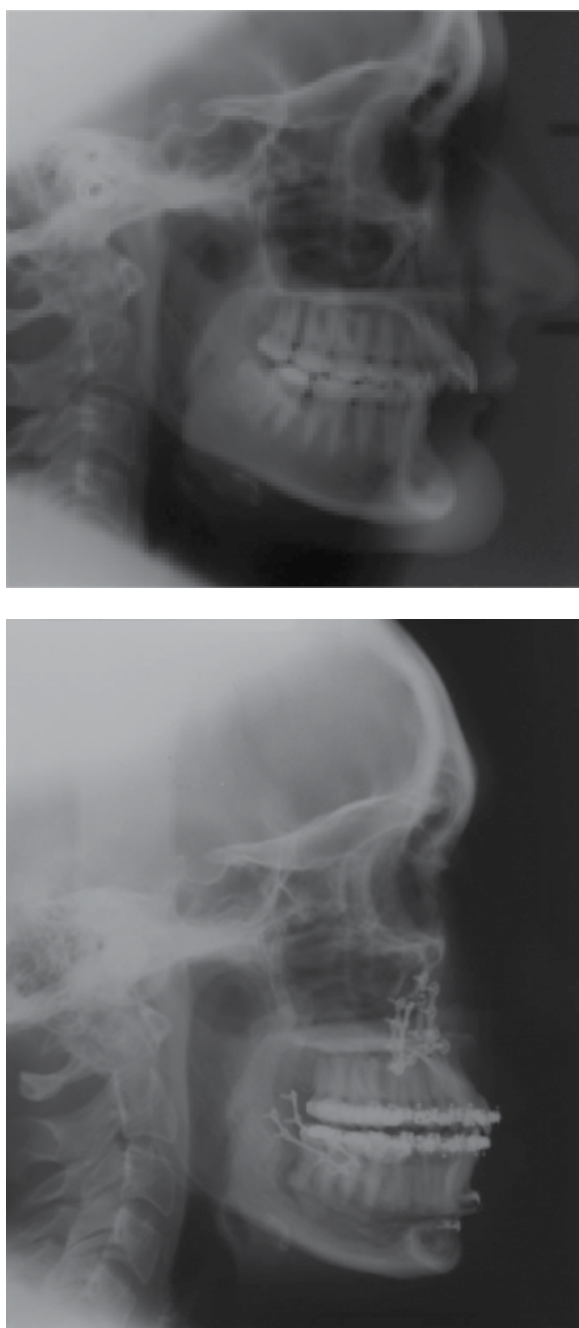


Figure 73.21 Total subapical osteotomy.

necessitate the need for a second procedure to realign the implant or mobile segment.

Reduction genioplasties could reduce support for the overlying soft tissues. This results in unfavourable changes to the cervico-mental angle, producing a double chin appearance. Plication of the platysma muscles has been suggested as a method to treat this problem.²⁰ The authors, however, favour submental liposuction or, in the more severe cases, a lower facelift procedure.

Summary

The chin is one of the most obvious facial structures and genioplasty surgery can play an important part in the correction of facial discrepancies. A clear understanding of assessment, planning and surgical technique are imperative to achieve a pleasing result and happy patient.

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CHAPTER 74

Blepharoptosis

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Definition

Blepharoptosis is defined as a lower than normal position of the upper eyelid in relation to the globe and pupil. The term is often abbreviated to ptosis.

The upper eyelid normally covers the upper 1–2 mm of the cornea, but this tends to increase slightly in old age. As the cornea has a diameter of 11 mm, the distance from the upper eyelid margin to the inferior corneo-scleral limbus is therefore normally 9–10 mm. This distance can be used as a means of measuring the amount of ptosis, but conventionally, the distance from the upper eyelid margin to a reflection of a point source of light shone directly at the patient is used. This distance is called the margin reflex distance 1 or MRD1. This is normally 3–4 mm. (The MRD2 is the distance from the light reflex to the lower lid margin.) The disadvantage of using the MRD1 over a simple measure from the lid margin to the inferior limbus is that when the upper eyelid margin is lower than the position of the light reflex on the cornea, the lid must be lifted up to then get an indirect measure of how far below the light reflex the lid margin sits. If it is lower than the light reflex it is expressed as a negative value in millimetres.

Anatomy

Knowledge of the anatomy relevant to ptosis is vital in understanding the aetiology and mechanisms of ptosis, and the clinical signs and surgery of ptosis.

The main surface anatomy marking of relevance is the upper eyelid crease or eyelid fold. In white populations this lies about 6–10 mm above the eyelash line. In Asian eyelids, there is much more variability, with the crease being absent or often much lower than in white eyelids, with an associated fullness of the upper eyelid. This is because the orbital septum attaches much lower in most Asian eyelids with the pre-aponeurotic fat pad extending more inferiorly, giving the eyelid a fuller appearance.

The upper eyelid retractor, the levator palpebrae superioris (LPS), is innervated by the superior division of the third cranial nerve (oculomotor nerve). The muscle belly arises from the apex of the orbit on the lesser wing of the sphenoid, just above the optic canal. It travels anteriorly in the orbit below the orbital roof, partly overlapping the superior rectus muscle, which lies immediately below it. As it reaches the anterior orbit, it turns more inferiorly towards the tarsal plate of the eyelid. About 8–10 mm above the superior border of the tarsus, it broadens out into a tendinous aponeurosis. This aponeurosis attaches onto the anterior surface of the tarsal plate, and also sends fibres to attach between bundles of the orbicularis oculi muscle just under the eyelid skin to form the upper eyelid skin crease (Figure 74.1).

The levator aponeurosis expands medially and laterally as its medial and lateral horns. The lateral horn is much stronger and attaches on to Whitnall's tubercle just inside the lateral orbital rim. The lateral horn also partially divides the lacrimal gland into orbital and palpebral lobes, with the two lobes continuous behind the posterior edge of the lateral horn. The medial horn is less well defined and becomes weaker with age. It attaches to the superior border of the medial canthal tendon.¹

There is a smooth muscle component of the levator apparatus, called Müller's muscle, which is innervated by sympathetic nerve fibres. This is a sheet of smooth muscle fibres with a rich blood supply that arises from the undersurface of the levator muscle and extends downwards under the levator aponeurosis to attach to the superior border of the tarsal plate. It is about 10 mm in length.

There is a strong transverse band of connective tissue running from the region of the trochlea to the lacrimal gland, which runs over the superior surface of the levator muscle near the point where it turns more inferiorly towards the tarsal plate. This is called the superior transverse ligament or Whitnall's ligament. There is a less well-defined portion of this structure which passes under the levator muscle, called the transverse superior fascial expansion.²

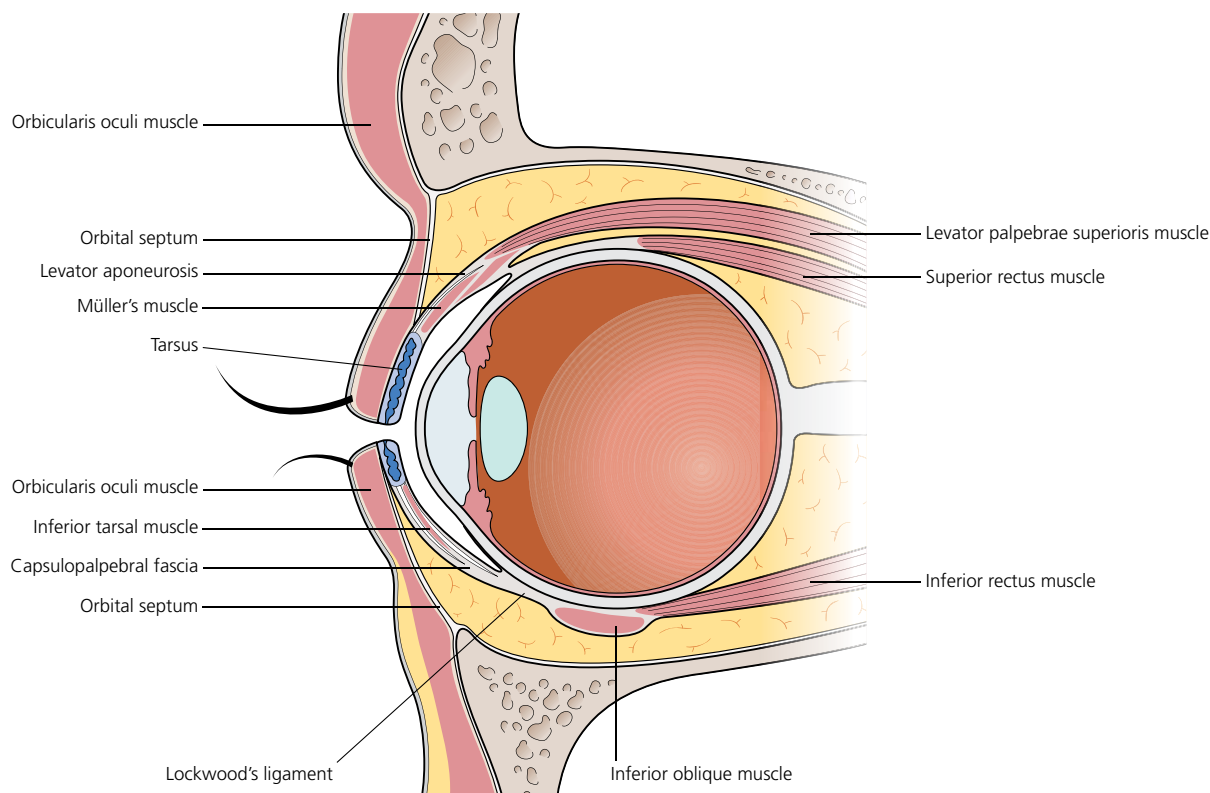


Figure 74.1 A sagittal section through the eyelids to show the relationship between the levator apparatus, tarsal plate and orbital septum.

The antagonist muscle of the LPS is the orbicularis oculi muscle, innervated by branches of the facial nerve. It runs circumferentially around the eyelids, divided into parts called pre-tarsal, preseptal and orbital. The orbicularis oculi muscle lies immediately below the skin of the upper eyelid, which is extremely thin.

The orbital septum in the upper eyelid extends from the superior orbital rim downwards to attach to the anterior surface of the levator aponeurosis. Behind the orbital septum and lying over the levator aponeurosis and anterior portion of the levator muscle belly is the pre-aponeurotic fat pad, a vital landmark in levator aponeurosis and muscle surgery. Laterally, in the same retroseptal space, the orbital lobe of the lacrimal gland is located. Medially, there is a medial upper fat pad. The tendon of the superior oblique muscle partially separates the central pre-aponeurotic and medial fat pads as it extends down to attach to the globe medial to the medial edge of the levator muscle belly.

The tarsal plate of the upper eyelid is a semilunar-shaped semi-rigid structure made up of meibomian glands set in a fibrous stroma with the conjunctiva firmly adherent to its undersurface. It attaches via the upper limbs of the medial and lateral canthal tendons to Whitnall's tubercle laterally and the frontal process of the maxilla just anterior to the anterior lacrimal crest (anterior limb of the medial canthal tendon) and to the posterior lacrimal crest (posterior limb). The upper tarsal plate is about 9–10 mm in vertical height centrally and about 1 mm thick.

There is a vascular arcade overlying Müller's muscle at its lower edge, and under the levator aponeurosis, just above the superior border of the tarsal plate. A smaller vascular arcade runs just above the lash bulbs near the margin of the eyelid, within fibres of orbicularis oculi.

Aetiologic factors

When considering the causes of blepharoptosis, it is conventional to divide them into congenital and acquired. However, there is some overlap in these two groups.³ The classification shown in Table 74.1 is useful.

Congenital and acquired neurogenic ptosis can be considered together in terms of investigation and management. Third nerve palsies may be partial or complete, and there may be aberrant regeneration complicating the clinical picture. Congenital aponeurotic ptosis is rare, but can also be managed in the same way as acquired aponeurotic ptosis.

Congenital myogenic or dystrophic ptosis may be unilateral or bilateral (symmetric or asymmetric) and is due to a maldevelopment of the levator muscle, which is variably replaced by fatty and fibrous tissue. The muscle contracts and relaxes less than normally. This type of ptosis often occurs with a positive family history and there are a number of genetic defects that continue to be identified. Although it is usually an isolated abnormality, there are well-recognized associations, including poor functioning of

Table 74.1 Classification of blepharoptosis

A. Congenital ptosis	1. Congenital neurogenic ptosis	1a. Congenital 3rd nerve palsy 1b. Congenital Horner's syndrome 2a. Isolated congenital dystrophic ptosis
	2. Congenital myogenic ptosis (sometimes called congenital dystrophic ptosis)	2b. Congenital dystrophic ptosis with associated weak globe elevator(s) 2c. Congenital dystrophic ptosis as part of a syndrome i. Blepharophimosis syndrome ii. Other syndromes (e.g. Noonan's, Saethre Chotzen)
	3. Congenital ptosis with synkinesis (jaw-winking ptosis, others)	
	4. Congenital aponeurotic ptosis (rare)	
B. Acquired ptosis	1. Neurogenic ptosis	1a. 3rd nerve palsy 1b. Horner's syndrome 2a. Myopathic ptosis i. Mitochondrial cytopathies (chronic progressive external ophthalmoplegia or CPEO and its variants) ii. Myotonic dystrophy iii. Oculopharyngeal muscular dystrophy iv. Other myopathies 2b. Myasthenia i. Ocular myasthenia ii. Myasthenia gravis 3a. Involutional aponeurotic ptosis 3b. Involutional myogenic ptosis
	2. Myogenic ptosis	
	3. Involutional ptosis	
	4. Mechanical ptosis (lumps and swellings of the eyelid)	
C. Pseudoptosis	5. Traumatic ptosis (may be multiple factors)	
	1. Globe hypotropia	
	2. Enophthalmos, or a small eye	
	3. Blepharospasm	
	4. Dermatochalasis	

the superior rectus muscle and double elevator palsy. Children with these additional muscle problems will usually have a vertical strabismus with the globe pointing downwards (hypotropia).

Congenital myogenic ptosis may be part of a more extensive syndrome. The best known of these is blepharophimosis or BPES (blepharophimosis, ptosis, epicanthus inversus) syndrome. Numerous other less common syndromes may have ptosis as one manifestation of the condition; Noonan's and Saethre Chotzen syndrome are two examples.

Congenital ptosis with synkinesis is a special subcategory of congenital ptosis. The best-recognized form is Marcus Gunn jaw-winking ptosis. The child will have unilateral ptosis and with contraction of the ipsilateral lateral pterygoid muscle, the levator muscle is stimulated and the lid rises, sometimes higher than the normal position. Synkinesis with other muscles is less common but well described. These include lid elevation with contraction of the medial pterygoid, digastric, the lateral rectus muscle, and with swallowing.

Acquired myogenic ptosis is an important diagnosis to make because of the significant general health aspects of these conditions.

Involutional or age-related ptosis is by far the commonest form of ptosis seen in clinical practice. A mild degree of ptosis is common in the elderly, but more advanced degrees may occur and impact on visual function. Traditionally, the majority of

involutional ptosis has been attributed to dehiscence and/or stretching of the levator aponeurosis, but in a significant proportion of older patients with acquired ptosis, the levator muscle itself appears to be abnormal with infiltration by fatty tissue into the muscle belly.

Some lumps on the upper eyelid may induce ptosis by their weight alone, and this is the classic cause of so-called mechanical ptosis. Other mechanical factors may be at play in some cases of ptosis, including scarring or adhesion between parts of the muscle and other structures such as the orbital roof after fractures, or adhesions between the eyelid and the globe.

Traumatic ptosis is often multifactorial. Traumatic nerve injuries may lead to ptosis, as may injuries to the muscle belly or aponeurosis. Some patients will develop ptosis after episodes of eyelid bruising or swelling and it is thought that such swelling may lead to dehiscence of the levator aponeurosis. Other forms of ocular surgery may sometimes lead to ptosis. The mechanism is not always clear.

Repeated minor trauma may also lead to ptosis. This is thought to be the cause in patients who develop ptosis after many years of wearing hard or rigid gas permeable contact lenses, who often remove the lenses by applying traction at the lateral canthus to remove the lens.

Pseudoptosis has a number of causes. An important one to recognize is hypotropia. With the globe looking down compared

to its fellow eye, the lid will also be lower than normal because the lid follows the eye downwards. If the normal eye is covered and the patient takes up fixation with the hypotropic eye, the lid will usually rise at least partially as well. This uncovers the element of the ptosis which is due to 'pseudoptosis'. Ideally, the hypotropia should be corrected first to eliminate any element of pseudoptosis.

A small or enophthalmic eye will often have a narrower than normal palpebral fissure, due to inadequate posterior support of the eyelid, but this is not due to any abnormality of the levator apparatus or eyelid and is classified as a form of pseudoptosis.

Patients with increased tone in the orbicularis oculi muscle will often have a narrowed palpebral fissure also. This may be due to increased tone with or without an element of aberrant reinnervation after a facial palsy. The lid fissure may narrow further when muscles in the lower face contract. Similarly, patients with hemifacial spasm will display episodic narrowing of the palpebral fissure associated with spasm of other facial muscles on the same side.

Finally, marked dermatochalasis will often look like true ptosis but if the hood of abnormal skin is lifted out of the way, the lid position can then be properly assessed. Many cases of true involutional ptosis will of course also have a degree of dermatochalasis.

Preoperative assessment

As in any medical condition, a proper history and examination is essential. The history will usually easily separate congenital from acquired ptosis. Questions should be asked about time and rapidity of onset and variability of the ptosis, associated diplopia, other neuromuscular problems, any history of trauma, lid swelling or contact lens wear. A family history of ptosis or muscle problems should be sought. Older patients should be asked about a history of dry eye problems as elevating the eyelid in a patient with dry eyes may exacerbate the dry eye problem.

A standard sequence of examination steps will usually give an answer, in addition to the details of the history, as to what has caused the ptosis, and make surgical planning safer. The examination steps may seem arduous, but with experience, a targeted ptosis examination can be performed in a few minutes. The steps should include the following:

- 1 *Measure and record the patient's vision.* This is especially important in children in the amblyogenic age group (less than 10 years of age). There is a strong association between ptosis and amblyopia.
- 2 *Measure the degree of ptosis.* This can be done by measuring the MRD1 (upper lid to corneal light reflex), or the distance from upper lid to lower limbus (normal is 9–10 mm). The degree of ptosis should be measured with the brow held in a neutral position to eliminate any elevation of the lid by the brow.
- 3 *Measure the levator excursion or 'levator function'.* This is the distance the lid moves between extreme up and down gaze. Normal should be about 13–15 mm.

- 4 *Look at the position of the ptotic lid on down gaze.* In congenital myogenic ptosis, because the levator relaxes less well, the lid does not drop as much as normal on down gaze. With involutional aponeurotic ptosis, the lid will drop more than normal on down gaze.
- 5 *Measure the position of the eyelid crease.* This helps in diagnosis and surgical planning. The crease is usually elevated compared to the normal side in aponeurotic ptosis. The crease may be absent in more severe congenital ptosis.
- 6 *Examine the patient's eye movements.* This is a vital part of the examination. Patients with myogenic ptosis will usually have signs of abnormal eye movements, which may be subtle at the first presentation. The range of eye movements, rapidity of eye movements (tested by examining saccadic eye movement), and presence or absence of strabismus in any position of gaze by performing a cover test, should all be performed.
- 7 *Examine the pupils.* The pupil will be enlarged in many third nerve palsies, and in Horner's syndrome the pupil will be smaller on the affected side. This difference is more obvious in dim light where the normal side dilates more.
- 8 *Examine eyelid closure.* Gentle eyelid closure should be tested first, looking for lagophthalmos, indicating orbicularis weakness. Forced closure should be tested also, looking for weakness.
- 9 *Examine for Bell's phenomenon.* This is where the eye rolls upwards on forced lid closure. If absent, the risk of corneal exposure problems is increased with ptosis surgery.
- 10 *Evert the upper eyelid and examine the conjunctival fornices.* The tarsal surface of the lid may be abnormal after previous ptosis surgery, and the conjunctival fornices may be hiding rare disorders such as subconjunctival lymphoma (seen as a salmon patch) or conjunctival amyloidosis.
- 11 *Look for associated clinical signs.* These include brow position (the frontalis muscle on the side of a unilateral ptosis will be usually overacting, elevating the brow), the presence of synkinetic movements (ask the patient to open the mouth and move the jaw side to side), and evidence of other features of myopathies or myasthenia (frontal balding in myotonic dystrophy, muscle weakness elsewhere, fatigability with myasthenia on sustained upgaze, a twitch of the upper eyelid on upward saccades of the eyes, known as Cogan's lid twitch, and occurring with myasthenia).
- 12 *Check that corneal sensation is normal.* This can be done with a strand of cotton teased from the end of a cotton tip. Reduced corneal sensation is rarely seen in ptosis patients, but if present, it markedly increases the risk of corneal complications after ptosis surgery.
- 13 *Examine the ocular surface.* This is best done with a slit lamp after placing a drop of fluorescein in the eye. Alternatively, a direct ophthalmoscope with a cobalt blue filter can be used. The adequacy of the tear film should be assessed as well as the presence of corneal drying. If the tear film is deficient or the cornea stains with fluorescein, there is a significant risk of worsening corneal drying or even ulceration with ptosis surgery.

At the end of the history and examination, a confident diagnosis of the cause of the ptosis can be made in most patients. If there is doubt, or certain diagnoses are suspected, then further investigations or referral to other specialists may be appropriate. The following scenarios deserve further investigation or referral:

- Acquired third nerve palsy. Should be referred for neurological assessment and imaging.
- Acquired Horner's syndrome. Neurological referral.
- Suspected myasthenia. An ice-test is a simple investigation that can help establish a diagnosis of myasthenia. An ice pack is placed over the eye with ptosis for 3–5 min. In myasthenia, the lid should elevate by at least 2 mm, and in patients with diplopia, this may temporarily resolve or reduce. All patients with suspected myasthenia should be referred for neurological assessment and investigation.
- Suspected myopathic ptosis. Neurological referral. Most patients with myasthenia are managed medically. Occasionally, myasthenic ptosis may resist medical therapy and in some cases, surgery can be cautiously offered.
- Reduced vision in a child under 10 should be referred to an ophthalmologist.
- Presence of strabismus. Referral to an ophthalmologist.

There may be other scenarios requiring further investigation or referral and if in doubt about the cause of the ptosis, then assessment by an ocular plastics specialist ophthalmologist or neuro-ophthalmologist is appropriate.

Treatment principles

The principles of surgical and other forms of management of ptosis can be divided into the following groups:

- congenital ptosis
- blepharophimosis syndrome
- jaw-winking ptosis
- myopathic ptosis
- involutional ptosis.

Congenital ptosis

The management of congenital ptosis can be divided into several steps.

- 1 Refer all congenital ptosis for ophthalmological assessment at first diagnosis. Ideally this should be as soon as the diagnosis is made, shortly after birth. The ophthalmologist will assess the infant for refractive error and amblyopia and initiate appropriate treatment. If the ptosis is severe and the vision is not developing normally despite amblyopia therapy (glasses, patching or atropine penalization with drops), then early surgery may be indicated. Otherwise, the surgery can be delayed until accurate measurements of the degree of ptosis and levator function can be made, allowing proper surgical planning.
- 2 If there is a squint present, then the patient should also be referred as early as possible to an ophthalmologist.

The commonest squint present with unilateral congenital ptosis is a hypotropia. If present, the hypotropia should be corrected before any ptosis surgery, because there will often be an element of pseudoptosis present which will be reduced by correcting the hypotropia. Additionally, correcting the hypotropia will reduce the risk of corneal exposure problems by improving globe elevation and the Bell's phenomenon.

- 3 If the vision is developing normally and there is no strabismus, then ptosis surgery can be planned around the age of 3–5 years, when good measurements can be obtained that help in the planning of ptosis surgery.

Surgery for congenital ptosis

If early intervention is required because of the presence of amblyopia, then normally a brow suspension is performed using non-autogenous material such as silicone. This allows for later removal and more definitive ptosis surgery at a later age if required. The technique for brow suspension is discussed later.

If the ptosis surgery is performed electively at or after the age of about 4 years, when good and reproducible measurements of the degree of ptosis and levator function can be obtained, then the choice of procedure will be determined principally by the degree of ptosis and levator function. If the levator function is poor (5–6 mm or less), then performing a levator resection or shortening procedure will lead inevitably to undercorrection. In this setting a brow suspension is preferred.

For congenital ptosis with 7 mm or more of levator function, a levator shortening or resection is preferred. The amount of resection is determined by the degree of ptosis and levator function. Complex tables are published, and the best known of these is that of Beard.⁴ In general, levator function can be divided into poor, moderate and good. 'Good' levator function in congenital ptosis is 8 mm or more. Moderate is 6–8 mm. The range of shortening or resection for congenital ptosis is of the order of 10–25 mm. For genuine congenital ptosis with a myogenic cause, a resection of less than 10 mm is likely to lead to undercorrection. Within the group of patients with moderate levator function, the amount of resection will depend on the degree of ptosis (Table 74.2). An additional useful guide for the amount to resect the levator is Berke's rule of 7's. Under general anaesthesia, if the levator function is 7 mm then the lid should be set to the desired level (usually 2 mm below the superior limbus). If more than 7 mm, it is set lower, and if less than 7 mm, it is set higher.

Additional factors that may influence the amount to resect the levator muscle include:

- The presence of milder ptosis on the other side. Smaller resections may be performed to try and achieve symmetrical bilateral mild ptosis.
- The presence of poor superior rectus function – an additional 4 mm is resected.
- The decision to preserve Müller's muscle or not – an additional 2 mm is resected.

For very mild congenital ptosis (1–2 mm) with near normal levator function (13 mm or more), a posterior approach with

Table 74.2 Amount of levator resection according to degree of ptosis and levator function in congenital ptosis

Amount of ptosis	Levator function	Levator function	Levator resection
2 mm or less	Good	10 mm plus	10–13 mm
3 mm	Good	10 mm plus	14–17 mm
3 mm	Fair	7–10 mm	18–22 mm
4 mm or more	Fair	7–10 mm	23 mm
4 mm or more	Poor	6 mm or less	23 mm or more

Adapted from Callahan & Beard, 1990.⁴

resection of conjunctiva and Müller's muscle, or conjunctiva, Müller's muscle and tarsus (Fasanella–Servat procedure) can be performed (see below).

Techniques

Levator resection

The amount to resect the muscle will have been determined preoperatively. The patient will usually be under general anaesthesia. The following steps will be performed:

- 1 *Mark the upper lid skin crease.* This will be generally marked to match the other (normal) side and should have been measured preoperatively, as it is not always obvious under general anaesthesia.
- 2 *Inject local anaesthetic.* Inject some local anaesthetic with adrenaline into the eyelid; 1–2 mL is injected under the skin in the region of the planned incision. A very small bleb can also be injected subconjunctivally in the superior fornix after everting the upper eyelid.
- 3 *Make an incision.* Make an incision in the marked crease through skin. The wound is then deepened through orbicularis muscle (Figure 74.2a).
- 4 *Expose the tarsal plate.* Dissect downwards onto the anterior surface of the tarsal plate. As the tarsal plate is exposed, the lower end of the levator aponeurosis will be transected, giving it a free lower edge. The tarsus should be exposed for most of its width and over its upper 3–4 mm (Figure 74.2b).
- 5 *Open the orbital septum* (Figure 74.2c). To do this, the upper edge of the wound is grasped and pulled upwards. An assistant grasps the lower edge of the levator aponeurosis. Sharp dissection is performed between the orbicularis muscle and levator aponeurosis, extending under the orbicularis. As the septum is opened, the anterior part of the levator muscle is exposed. A key landmark is the pre-aponeurotic fat pad, a mobile, bright yellow pad of fat that overlies the anterior portion of the levator muscle belly. Once exposed, the fat pad is pulled upwards and the dissection of the muscle belly is extended into the superior orbit. The transverse or Whitnall's ligament will be seen. The dissection can proceed beyond this if a larger resection is required.
- 6 *Separate the levator from the tarsus and conjunctiva* (Figure 74.2d,e). The aim here is to isolate the levator

aponeurosis and muscle on its under surface to allow it to be resected. The initial separation is at the upper border of the tarsal plate. There is a vascular arcade here that bleeds. Müller's muscle lies on the conjunctiva and can be separated by initial sharp dissection, then blunt dissection. As the dissection proceeds, a thicker layer of connective tissue that is white in colour is seen. This is the layer known as the common sheath or suspensory ligament of the fornix, and is the layer of connective tissue that extends forwards between the levator and superior rectus muscles. If damaged, prolapse of the superior conjunctival fornix can occur.

- 7 *Divide the medial and lateral horns* (Figure 74.2f). If the resection is larger (>14–16 mm), then the medial and lateral horns should be divided to allow the muscle belly to advance more easily. The cuts should be made towards the belly of the muscle to avoid injury to the superior oblique tendon medially, and the lacrimal gland laterally. Once these cuts are made, the levator aponeurosis and muscle are able to be shortened or resected the desired amount. In some cases Whitnall's ligament may need to be separated from the LPS to allow it to advance (Figure 74.2g).
- 8 *Place a central suture* (Figure 74.2h). Use a non-absorbable or long-acting absorbable double-armed suture such as 5-0 polyester or vicryl. Pass one needle partial thickness through the tarsus about 2 mm below its superior border at a point just medial to the pupil line, where the peak of the lid contour will be. Pass both needles up under the belly of the muscle to emerge through the muscle 2–3 mm apart at a point determined preoperatively by reference to Table 74.2. Have an assistant hold the muscle on tension and tie a bow in the suture and assess the lid height. Using Berke's rule of 7's, the suture can be adjusted if necessary. Undo the bow once the lid height is satisfactory.
- 9 *Place medial and lateral sutures* (Figure 74.2i). Sutures are then passed in a similar fashion medially and laterally. The central suture can be tied, then the medial and lateral sutures put on a bow to assess the lid contour. Adjustments are made as needed to achieve a satisfactory lid contour, then both are also tied.
- 10 *Trim the muscle.* The levator muscle and aponeurosis and Müller's muscle are then trimmed below the sutures.
- 11 *Reform the lid crease* (Figure 76.2j). This can be done by using an absorbable 6-0 suture from the orbicularis of the lower wound edge to the cut edge of the levator muscle at three or more points. This gives a more natural 'soft' crease. Alternatively, a suture from skin to levator to skin can be used to create a harder crease.
- 12 *Suture the skin.* Absorbable sutures can be used.
- 13 *Apply ointment.* In children, copious ointment is inserted into the eye and over the wound without an eyepad. In older patients, a temporary Frost suture can be used in the first 24 h for corneal protection, with a double eyepad.

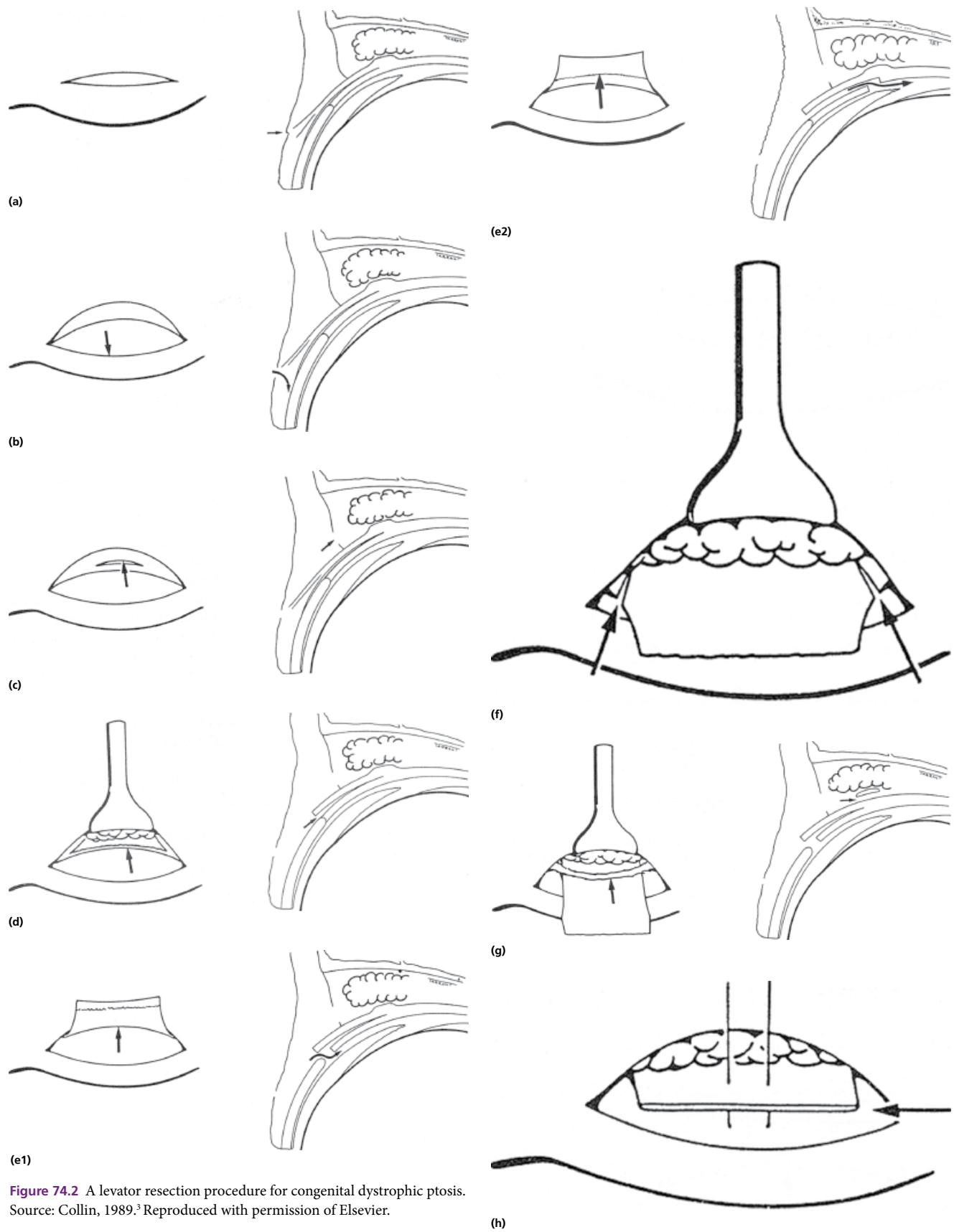


Figure 74.2 A levator resection procedure for congenital dystrophic ptosis. Source: Collin, 1989.³ Reproduced with permission of Elsevier.

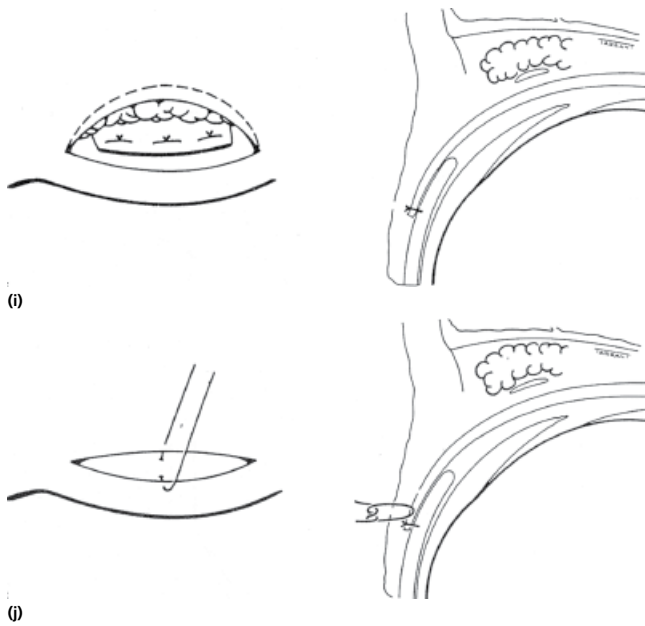


Figure 74.2 (Continued)

Tarso-conjunctival resection (Fasanella-Servat procedure)⁵

For very mild congenital ptosis with near normal levator function, a tarso-conjunctival resection can be performed. This is an uncommon clinical scenario and the large majority of congenital ptosis will require levator resection or brow suspension surgery.

- 1 **Evert the upper eyelid.** This is most easily done after placing a 4-0 traction suture in the lid margin and using a Desmarres retractor.
- 2 **Inject local anaesthetic.** Anaesthetic with adrenaline is injected subconjunctivally just above the superior border of the tarsus.
- 3 **Mark the area of tarsus and conjunctiva to resect.** This will be 3–4 mm of tarsus centrally (leaving at least 4 mm of tarsus from the margin), tapering medially and laterally. A similar amount of conjunctiva is marked.
- 4 **Resect the tarsus and conjunctiva/Müller's muscle.** Obtain haemostasis.
- 5 **Suture the wound.** This can be done by passing a non-absorbable suture such as 6-0 prolene or nylon from the external surface of the lid to emerge at one end of the wound, then suturing continuously with a small number of bites and emerging externally at the other end of the lid. The suture is pulled tight to minimize the risk of corneal abrasion, and the ends tied together over the skin. It is removed at one week. In very young patients, an absorbable suture can be used with the knots tied on the skin surface.
- 6 **Apply ointment.**

Brow suspension

A brow suspension procedure is indicated when the levator muscle function is poor (5–6 mm or less). If there is bilateral symmetrical ptosis with poor levator function, then the procedure is clearly bilateral. If there is bilateral asymmetric

ptosis and one side has poor levator function, and the other side has moderate function, then again, a bilateral brow suspension is preferred so as to give better symmetry both in primary position and in down gaze.

If the brow suspension is being done in a young infant because of the development or risk of amblyopia, then a non-autogenous material can be used, such as a silicone sling or a strong suture such as 2-0 prolene.

For elective brow suspension beyond the age of about 3 years, then fascia lata is the most widely used material. Alternatively, if present, plantaris tendon can be used.

1 **Harvest the fascia lata.** A small incision is made in the thigh just above the knee on the lateral side overlying the ilio-tibial tract, which runs from the iliac crest to the head of the fibula. There are strong transverse fibres on the fascia lata, which can be stripped off bluntly with toothed forceps to reveal the parallel longitudinal fibres that are to be harvested. For a bilateral procedure, the strip should be about 12–15 mm wide. Less is taken for a unilateral brow suspension. A blunt curved artery forceps can be used to split the fibres and begin the dissection. A length is isolated in the wound and clamped. It is divided at the lower end of the wound and the end then passed into the mouth of the fascial stripper which is then pushed up the thigh, bluntly separating the strip of fascia. Once the stripper is in the upper part of the thigh, the strip is cut and retrieved, and the wound sutured.

2 **Divide the fascia into strips.** The strips should be 3–4 mm wide. Wider strips produce large knots that are harder to bury. The strips can be made by dividing the large strip near one end for a few millimetres, grasping that part and pulling it away from the remainder to make a thin strip. Sharp dissection should not be needed.

3 **Mark the incisions in the lid and brow.** The brow suspension can be performed in a 'closed' or open-sky technique. A closed technique works well for fascia lata. For redo procedures or if silicone or prolene is used, an open-sky method works better. For an open-sky method, the tarsus is exposed as for a levator resection and the material sutured to the tarsal plate before completing the brow suspension (see below under Myopathic Ptosis).

For a closed brow suspension, three small marks are made at a level about 2 mm below the desired level of the skin crease, centrally, medially (just lateral to a point above the punctum), and laterally (2–3 mm medial to the lateral canthus). Two small incisions (about 4–5 mm in length) are marked almost directly above the medial and lateral marks at the upper border of the eyebrow and the third brow incision placed in the forehead to form an equilateral triangle with the two brow incisions.

4 **Make the incisions.** In the eyelid, these are made as small stab incisions with the end of an 11 blade. In the brow and forehead, they are longer to allow the knots in the fascia to be buried, and should be down to pericranium.

- 5 *Insert the fascial strips in the eyelid (Crawford pattern).*⁶ Using a Wright's fascial needle, a pass is made from the medial eyelid wound to the central wound, passing deep to orbicularis and anterior to tarsus, across the lid. Place one strip in the eye of the needle and pull it back through the wound. Place another strip similarly from the central to the lateral lid wound. Then pass the fascial needle from the medial brow wound, at a depth just superficial to pericranium, and beneath the orbicularis in the eyelid, being careful to avoid passing the needle through the lid into the fornix or eye. An eye shield should be used. One end of the medial loop is retrieved, then the other so both ends emerge from the medial brow wound. The same is done from the lateral brow wound with the other loop.
- 6 *Tie the knots in the brow wounds.* The fascial strips are pulled up, leaving one end longer (for later passage to the forehead wound). The lid should rise to the desired level about 2 mm below the upper limbus. If the lid pulls away from the globe before this point is reached, stop at the point just before the lid pulls off the globe. A single throw is made and the square knot completed with a second single throw. The fascial knot should be reinforced with a suture such as 6-0 vicryl passed through the fascial knot.
- 7 *Pass the strips to the forehead wound and tie.* The longer ends of the fascial strips are then passed to the forehead wound. They should be tied with some tension to maintain the desired lid level and reinforced.
- 8 *Suture the brow and forehead wounds.* The eyelid punctures do not need sutures.
- 9 *Apply copious ointment in the eye(s).*

Blepharophimosis syndrome

Children (or adults) with BPES syndrome have poor levator function and will need brow suspension surgery. It is customary to correct the telecanthus and epicanthic folds initially, usually at the age of 3–4 years, and then perform the brow suspension about six months later. Some patients with BPES will benefit from lateral ectropion surgery.

Medial canthoplasty

A variety of techniques have been described for the management of telecanthus and epicanthic folds in BPES. These include Mustarde's technique,⁷ its variation, the five-flap technique of Anderson,⁸ a Y-V plasty, and trans-nasal wire. My personal preference is for the Anderson five-flap technique, which better addresses the epicanthic folds.

The details of these procedures is beyond the scope of this chapter and the reader is directed towards the appropriate sources in the reference list.

Brow suspension

The technique is the same as described for poor levator function congenital ptosis (above).

Jaw-winking ptosis

The surgical management of jaw-winking ptosis is complex and there are a number of options available, depending on the desire to abolish (or ignore) the wink, the desire to achieve symmetry, and any concerns held by the patient or their parents about operating on the contralateral (normal) side.

If the main concern is with the ptosis (always unilateral) only, and the wink is of less concern, then a simple levator resection can be performed. The amount to resect the levator muscle is determined in a similar way to simple dystrophic myogenic congenital ptosis, but a slightly larger resection is often needed to achieve good symmetry in primary position.

If it is desired to abolish the wink, the only reliable way to achieve this is to ablate the levator muscle, creating complete ptosis with no levator function, and then perform a brow suspension procedure. If this is done unilaterally, there will be asymmetry often in primary position, with a degree of under-correction, and more marked asymmetry in down gaze, with hang-up of the upper lid on down gaze on the operated side. This is the reason that some surgeons recommend bilateral surgery in this scenario. The levator can be ablated on the affected side, then a bilateral brow suspension is performed, so that there is better symmetry in down gaze. Further improvement in symmetry can be achieved by ablating the levator muscle on both sides and performing bilateral brow suspension surgery (the Beard procedure). However, many patients and their parents are reluctant to consider surgery on the normal side, which converts a normal eyelid into one with limited excursion and potential complications in later life from incomplete closure and corneal exposure complications.

Levator ablation

If levator ablation is chosen (either unilateral or bilateral), it is most easily achieved via an anterior skin crease approach.

- 1 Make an upper eyelid skin crease incision.
- 2 Open the orbital septum and expose the anterior portion of the belly of the levator muscle.
- 3 Dissect superiorly to expose the belly of the muscle above Whitnall's suspensory ligament. Pass two squint hooks under the belly of the muscle and resect several millimetres of the muscle belly making sure all fibres and the muscle sheath are divided, otherwise the wink will not be abolished.
- 4 Proceed with brow suspension surgery, with the fascial strips sutured directly to the tarsal plate (see above).

Myopathic ptosis

Surgery for myopathic ptosis will depend on several critical factors which include:

- the diagnosis;
- the expected long-term progression of the disease;
- the levator muscle function;
- eye movements; and
- orbicularis oculi muscle strength.

The exact diagnosis of myopathic ptosis should generally be pursued before any surgical correction, because different forms of myopathic ptosis have different natural histories, different relative involvement of extraocular muscles and orbicularis oculi, and some are better treated medically (myasthenia). Additionally there may be genetic counselling issues in many of these patients.

Myotonic dystrophy often carries a worse prognosis than some other forms of myopathic ptosis as orbicularis oculi is more severely affected. Oculopharyngeal muscular dystrophy generally does well with brow suspension surgery, as orbicularis oculi tends to be better preserved, and eye movements are less severely affected, reducing the risk of corneal exposure. Chronic progressive external ophthalmoplegia (CPEO) varies in its severity, depending on the subset. Early-onset CPEO in the form of Kearns–Sayre syndrome is more severe and more rapidly progressive. Systemic problems are also commoner and include cardiac disease, generalized severe myopathy and early-onset diabetes.

If there is any suspicion of a myopathic form of ptosis, appropriate referral should be made to a neurologist with expertise in neuromuscular diseases.

Myasthenia

Surgery is not usually indicated for myasthenic ptosis, which should be treated medically. However, in some longstanding patients, surgery may be indicated for stable ptosis which does not respond to medical therapies. The surgery should be performed cautiously, bearing in mind the increased risk of inducing corneal exposure problems.

Levator muscle surgery in myopathic ptosis

Caution should be exercised in performing levator muscle surgery in myopathic ptosis. It should only ever be used where the levator muscle function is good. Often larger degrees of muscle shortening are required to elevate the lid sufficiently despite the apparently good levator muscle function and this leads to an increased risk in later life of corneal exposure problems. If performing levator muscle surgery (or brow suspension surgery) in these patients, lifelong follow-up is required as these diseases progress and ocular surface complications increase. The surgeon should be prepared to reverse the ptosis surgery if the cornea is at significant risk.

Brow suspension surgery for myopathic ptosis

Often, a more conservative brow suspension procedure is safer in these patients than a levator shortening procedure. The lid can be lifted by the often well-preserved frontalis muscle to a point just above the visual axis, allowing the patient to function reasonably well, and at the same time allowing reasonable eyelid closure and minimizing corneal exposure.

The choice of material used for the brow suspension surgery will depend on personal preference as well as other factors,

such as the ease of reversibility or adjustment later, and the requirement of general anaesthesia if harvesting fascia lata. For oculopharyngeal muscular dystrophy, which carries a relatively good prognosis, fascia lata brow suspension surgery is preferable. Under general anaesthesia, the eyelids should be set open just 2–3 mm, and when awake and the frontalis muscle is activated, the lids will generally rise above the visual axis, and eyelid closure will be reasonably well preserved.

For the more severe forms of myopathic ptosis, many surgeons prefer to use non-autogenous material such as silicone, Goretex® or heavy prolene suture. Local anaesthetic can be used for this procedure also. If using these materials, an open-sky technique is preferred with the sling material sutured directly to the tarsal plate with non-absorbable sutures. The material is inserted in a pentagonal shape (Fox pentagon), with the final knot tied in the forehead. Again the lid should be set just open, about 2 mm.

Involutorial ptosis

The majority of surgical procedures for ptosis will be for involutorial ptosis. The same techniques are used for involutorial ptosis as for aponeurotic ptosis that may occur with long-term hard contact lens wear, after some cases of trauma or lid swelling, or in the blepharochalasis syndrome.

The surgical steps can be broken down into the following:

- 1 *Mark the proposed incision(s) in the upper eyelid.* The placement of the incision will be determined by the desired height of the eyelid crease, which will be made to match the opposite side in unilateral cases. In many cases of involutorial ptosis, excess upper eyelid skin will be excised also, not just for appearance, but to reduce the increased overhang of skin that would otherwise occur if the lid alone is elevated. The amount of skin to be excised should be judged carefully in each patient, and is more easily determined in bilateral cases. Removing too much skin should be clearly avoided, as the risk of corneal exposure is increased when ptosis is corrected at the same time.
- 2 *Inject the local anaesthetic.* This should be kept to a minimum volume, and 1–1.5 mL is usually adequate. An excess volume may cause a mechanical ptosis, and a larger volume is more likely to spread deep to the orbital septum and partially paralyse the levator muscle fibres, making the assessment of the lid height more difficult. Recheck the lid height and levator function before proceeding.
- 3 *Make the skin incisions.* Excess skin is removed at this stage. If the surgery is bilateral, each step can be performed on each side so that the surgery advances in parallel. This makes achieving symmetry easier.
- 4 *Expose the anterior surface of the tarsal plate.* This is achieved by sharp dissection below the orbicularis muscle at the inferior edge of the incision, heading obliquely towards the tarsal plate. As the tarsal plate is exposed, the fibres of the levator aponeurosis that are destined for the tarsal plate, are divided, creating

an inferior edge on the aponeurosis which can be used later for attachment or advancement on the tarsal plate.

- 5 *Open the orbital septum.* Sharp dissection proceeds under the orbicularis muscle superiorly, with an assistant holding the inferior edge of the levator aponeurosis downwards. Dissection proceeds until the pre-aponeurotic fat is exposed, which is then dissected gently off the underlying levator muscle and aponeurosis.
- 6 *Recheck the eyelid levels.* Sometimes the lid may elevate without advancing the aponeurosis, possibly because of adrenaline in the local anaesthetic stimulating the Müller's muscle, or the lid may drop because the aponeurosis has been detached from the tarsus.
- 7 *Pass a double-armed 5-0 or 6-0 non-absorbable suture partial thickness* through the tarsal plate about 2 mm below its superior border, at a point where the peak of the arch of the lid contour should lie. This will usually be just medial to the mid-pupil. Pass each needle superiorly under the levator aponeurosis, between it and the Müller's muscle, to emerge through the aponeurosis. The level at which the sutures pass will be based on several factors, including the appearance of the muscle (is there fatty infiltration and degeneration), and the length and appearance of the aponeurosis. In any event, the level of the initial pass is by trial and error. Once both ends are passed, a bow is tied and the lid height assessed. In most cases, the aim will be to set the lid at or just below the superior limbus (bilateral cases) or 1–2 mm higher than the unoperated side (unilateral cases). Some surgeons assess lid height with the patient lying down, some sitting up. Either is acceptable and each surgeon should be consistent to better achieve more predictable outcomes. If the lid height is not correct, withdraw and reposition the sutures until the desired height is achieved.
- 8 *Insert medial and lateral sutures* once the desired lid height is achieved. The central suture is untied, and similar sutures are passed medially and laterally. The central suture is then tied, and the medial and lateral sutures are secured with a bow. The lid contour is checked and any adjustments made that might be required.
- 9 *Reform the upper eyelid skin crease.* Pass an absorbable suture from the orbicularis muscle adjacent to the inferior skin incision, onto the levator aponeurosis, making sure that the anterior lamella of the eyelid is draped over the tarsus without it being too tight or too loose. Three sutures are sufficient. The skin is then sutured separately.
- 10 *Apply antibiotic ointment.* Dressings can be applied if desired, and ice packs also.

Complications

The potential complications of ptosis surgery are many, but the commonest will be addressed here. These are:

- undercorrection,
- overcorrection,
- poor lid contour, and
- poor eyelid closure with corneal exposure.

Undercorrection

This is common. In many instances, the surgeon may aim for 'undercorrection', particularly in older patients with poor tear film and dry eyes, or in patients with myopathic ptosis where the risk of corneal exposure is much higher.

If undercorrection is present at the first postoperative visit, it is usually better to wait for any swelling or possible underaction of the levator or Müller's muscle due to haematoma to settle before reoperating. This usually means waiting six weeks or more.

Overcorrection

Mild initial overcorrection is common and should be expected in unilateral cases. There is loss of function of the pretarsal orbicularis muscle that will recover over about eight weeks, by which time the lid will drop about 1 mm, occasionally a little more.

More significant overcorrection should be corrected early. The wound can be reopened under local anaesthetic and the sutures released. The levator aponeurosis (in involutional ptosis cases) will recess by a small amount only usually. More formal recessions may be required in more severe cases, with 'hang-back' sutures from the tarsus to the free edge of the aponeurosis.

Poor lid contour

This will not resolve with time and should be corrected. This can be performed early or late. One or more sutures may need to be replaced, higher or lower.

Poor eyelid closure with corneal exposure

This is commoner in the early weeks after ptosis surgery, and can often be managed with increased ocular lubricants including ointment, especially at night when the lid may be open during sleep. Eyelid closure improves in the first eight weeks after surgery, and if after that time the corneal exposure does not settle, then consideration should be given to lowering the lid.

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CHAPTER 75

Liposuction and liposculpture

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Introduction

Liposuction is the core procedure for body contour surgeons. It has evolved progressively and at times dramatically over the past three decades. Techniques have been honed and instrumentation expanded to meet the demands of the patient and surgeon for more precise fat sculpting, more refined results, less morbidity, downtime and fewer complications. Non-invasive fat sculpting tools have also been developed to reduce even further the morbidity of more invasive, traditional techniques or even avert the need for surgery or touch-up surgical procedures.

No matter which instruments or techniques are used, a thorough knowledge of the aesthetic and morbid anatomy of the area to be reshaped, is essential for a safe, reliable and attractive result. The aesthetics of each particular area need to be understood prior to and during the design and marking for the procedure.

The indications for and limitations of the results need to be appreciated. Patients' expectations need to be assessed prior to embarking on liposculpture because the results are not always predictable and may not comply with the patients' needs. Complications in the intraoperative and immediate postoperative period can be reduced by astute patient selection and following aspiration guidelines and avoiding prolonged operative time. The patient needs to be informed of these possible complications and management strategies should be in place in case problems arise.

Historical perspective

When the first presentations of liposuction reached the American Plastic Surgeons at the Hawaii meeting of the American Society of Plastic Surgeons in 1982, this heralded a transformation in body contour surgery which soon resulted in the technique of blunt liposuction espoused by its champion, Yves-Gérard Illouz, being enthusiastically endorsed internationally.¹ Courses soon sprung up, conducted in conjunction with members of the American Society of Plastic Surgeons and those who had a

particular interest in body contour surgery, including Frederick Grazer² from Newport Beach, California.

Over the last 30 years, there have been multiple contributors to the science, technology and performance of liposuction. The groundbreaking contributions of Illouz,¹ Bahman Teimourian,³ Gerald Pitman,⁴ Luis S. Toledo⁵ and others contributed greatly to the armamentarium for body contour surgery and the development of the field that has occurred due to increased bariatric surgery as a consequence of the obesity epidemic. The magnitude of the contributions made in the 1980s and 1990s has led to the performance of safer and more reliable liposuction.⁶

Indications and contraindications – patient selectivity

The optimal patient is one who has successfully completed a diet and exercise regime, but still has areas of fatty accumulation. In men these are usually in the abdomen, 'man-boobs' and 'love handles' areas, whereas in women they are usually the outer thighs, inner thighs and hips. Obese patients should only occasionally be judiciously accepted for liposuction, perhaps in isolated areas and with the understanding that there are limitations based on their health and expected overall improvement. It is generally accepted that large-volume liposuction is anything over 5000 mL of fat removal and that greater volumes of aspiration are associated with increasing complication rates.

A history of deep venous thrombosis, especially during pregnancy, long airline flights or a history of blood dysplasias, should be sought. As in any medical history, the patient's prescription medications, including antithrombotic medications, allergies, tobacco use, recreational drugs and herbal medication intake should be obtained. If patients are taking anti-anxiety medications or psychotropic medications, whether to carry on taking these medications or to desist from them needs to be discussed with the anaesthetist if the patient is to proceed with the surgery.

Physical examination

I examine the patient in front of a three-way mirror, in their underwear, to examine the whole figure and discuss the potential improvement that can be achieved with liposuction alone. The examination of each aesthetic area includes the evaluation of how fat is contributing to the distortion of the shape. Patients who have a history of exercise or sports and good muscularity as well as good skeletal axial proportions are generally better candidates for the procedure than those with poor skeletal proportions and flaccid muscularity.

Areas of fat should be observed, palpated and pointed out to the patient in the examination, and skin elasticity assessed visually and by pinch. Patients with excessively aged skin secondary to sun exposure or with stretch marks either from weight gain, weight loss or pregnancy should be told about the limitations in expectations of result based on the quality of their skin. Patients who have had multiple pregnancies with loose, lax abdominal skin and diastasis recti are generally not good candidates for liposuction; an abdominoplasty or resective body contour procedure is more likely to be suggested in this type of patient.

In examining the back, any asymmetries can be pointed out; a mild scoliosis is quite common for example. In examining, the abdomen, the plastic surgeon needs to assess whether there are associated abdominal hernias or peri-umbilical hernias which need to be dealt with as a separate issue. In patients with prior abdominal incisional scars, an abdominal ultrasound or CT scan might be necessary to clarify whether hernias are present if clinical examination is indeterminate.

Aesthetic units and design of liposuction

It is helpful to think of the body as a composite of aesthetic units which combine to form a corporal matrix of the body: the more curvaceous figure in the female and the more angular masculine shape in the male. If these composites are shaped and sculpted in an appropriate manner with liposuction, then the overall figure or shape of the patient will be improved.

Although I use computer imaging for facial surgery, I do not use it for body contour surgery, as I think it is quite possible that this could lead to patients having unrealistic expectations.

The surgeon should not be deterred by comments made by patients in an attempt to limit their results, such as 'my diet will take care of this or that area' or 'I am not worried about that, just do this and do that'. The aim of the liposuction surgery is to create an overall improvement in the shape of the torso and if areas with residual deformity are left untouched the overall shape will not be harmonious.

Before surgery the patient should have been counselled on the risks and complications of the procedure, as discussed in a later part of this chapter. No matter what machine is used, the key to the procedure will be the design, the performance of the procedure, the quality of the patient's tissues and the realistic expectations of the patient.

Preoperative marking (design)

The expected contours are drawn in on the body and a squeeze contour map of the height of the bulges of fat made using semi-permanent, different colour markers (Figure 75.1).

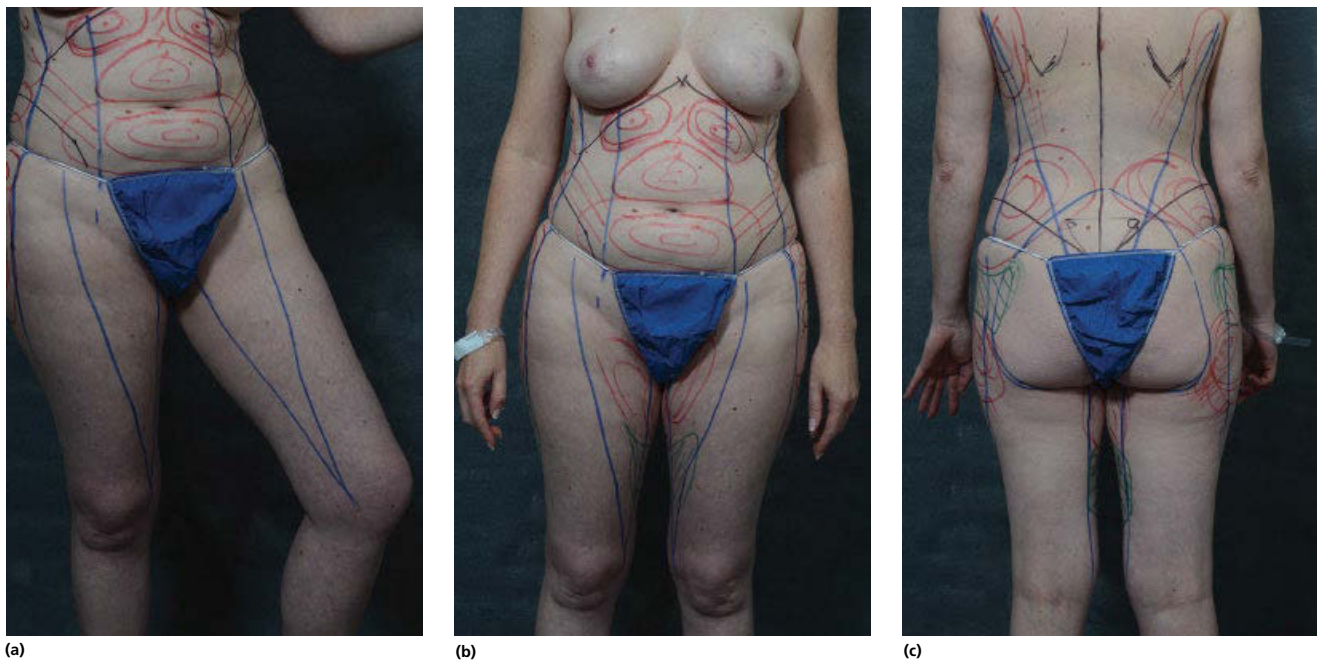


Figure 75.1 Sequence of design of figurative liposuction in a 45-year-old female prior to surgery, anticipating a 3 h procedure under general anaesthesia as an outpatient. Bones are represented in black, muscles in blue, fat is red and green is depressions.

Any divots, as well as cellulite and zones of adherence, should be marked with a different coloured pen during the marking process⁷ prior to the surgery. Patients may inquire about where the incisions are and how many incisions there will be; these are pointed out to the patient and those that cannot be placed in a crease are put, where possible, in the line of underwear or swimwear. Patients are often surprised to see in the three-way mirror and after the drawings that asymmetries exist; they should be reminded that any asymmetries due to skeleton or muscular asymmetries will not be improved by the liposuction procedure.

Liposuction machines

The original liposuction machines were modified aspirators used for uterine suction curettage, and the initial cannulas used by Teimourian³ were also curettage cannulas. The first commercial machines, such as the popular Grams machine, generated about 30 mmHg negative pressure. I have two of these machines still functioning; they require a minimum of maintenance on an annual basis and have been used for most of my 30 years performing liposuction.

Ultrasonic machines became popular in the 1990s⁸ and are still retained with variable amounts of enthusiasm by plastic surgeons. The newer, more contemporary, Vaser[®] machine is a more sophisticated ultrasonic machine with aspiration. It is espoused by those surgeons who claim it as valuable for body etching. Ultrasonic-assisted liposuction machines had solid probes and were of two types: one in which the emulsified fat was aspirated subsequently and the other which had associated suction attached within the probe.

Power-assisted liposuction (PAL) machines, initially air-driven⁹ but now with electrically activated motors have become quite popular. They reduce surgeons' fatigue and give more control than traditional, mechanical lipo-aspiration. Further developments include laser liposuction,¹⁰ ultrasonic¹¹ and hydro liposuction. External devices which offer modest non-invasive results over traditional techniques have also been developed utilizing ultrasound (Liposonix[®])¹² and cryolipolysis, which freezes fat externally, resulting in resorption.¹³

As with any technique, overzealous removal of fat can result in contour deformity. Newer machines that have been developed in the last 30 years introduce risks for the novice surgeon when adapting to the technique. The traditional technique stressed deep to superficial, sub-superficial aspiration with blunt cannulas. Any technique that violates the superficial plane, above the superficial fascia, risks damaging the subdermal structures with resulting irregularity to the skin, corrugations, pits and a 'moth-eaten' appearance which is difficult to treat.¹⁴

Liposuction cannulas

Traditional manual liposuction required a variety of cannulas of varying diameters and configurations to aspirate fat from a progressively deep to a superficial layer, avoiding damage to the

dermis and subdermal plexuses. A large variety of tip designs continue to be advocated and manufactured to surgeons' designs and particular affinities.

Pneumatic PAL, initially gas-powered, now electrical, facilitates back and forwards motion of a cannula, and reduces the effort required of the surgeon in passing the cannula through the fatty deposits. The cannula tips are similar to those used in suction-assisted lipectomy (SAL) cannulas. The handpiece incorporates special design tubing and the intensity of the back and forward vibration of the cannula tip can be adjusted. Near maximum speed is used in most cases. Syringe aspiration with a Tulip[®] system is also available, obviating the need for an aspiration machine. I use it for small areas or for fat harvest and fat injection techniques. Others have used it almost exclusively.⁵

Cannula choice is very individual from surgeon to surgeon, but the trend has been to use more narrow-bore cannulas.

Anaesthesia considerations

Local anaesthesia aided by sedation may be used for moderate amounts of liposuction, usually for less than 1000 mL reduction in selective areas, such as the abdomen, hips or outer thighs. The tumescent technique involves infusion of dilute lidocaine and adrenaline solution into the tissues until they are turgid, producing analgesia and vasoconstriction, allowing the removal of moderate amounts of fat without the patient undergoing general anaesthesia. For larger amounts of fat, up to 5000 mL, it is much easier on the patient and the surgeon to use general anaesthesia.^{15,16}

When contouring buttocks, posterior, inner thighs and with the patient in the prone position, general anaesthesia expedites more precise liposculpting. In the prone position, a special 'prone pillow' is used by anaesthesiologists to stabilize the endotracheal tube.

Positioning on the operating table is important. A common position is the lateral decubitus position, which was first espoused by Gasparotti¹⁴ in the early 1990s because it gives a much more precise contouring to the junctional areas between the buttock, thigh and hip, called the 'G spot' by Gasparotti¹⁴ or the 'sensual triangle' by Schlesinger.¹⁷ Although some anaesthetists are comfortable with the use of a laryngeal mask in the prone position, a well-secured endotracheal tube offers more equanimity to the whole process of liposuctioning in the prone position. Pillows positioned under the hips and chest prevent pressure in the prone position and the time spent in this position can be limited hopefully to no more than half an hour. In the lateral decubitus position, a bean bag can be used to facilitate stabilization. Care should be taken when changing the patient from the supine, to the prone and from one lateral decubitus position to the other.

Anaesthetists must be confident with the technique of the infusion of large volumes of saline, lidocaine and adrenaline into the tissues and should not overinfuse them with additional intravenous fluids, adjusting the intravenous fluids to the

amount of fluid that has been infused into the subcutaneous tissues.¹⁸

When liposuction is combined with other procedures such as abdominoplasty, thighplasty or other body contour surgeries, an indwelling catheter to monitor urinary output is advisable.¹⁹

Tissue infusion

Wetting solutions have been used for liposuction after the 'dry technique' was abandoned due to excessive blood loss and the need for autogenous blood transfusions. The solutions are generally infused with a high-pressure infuser. The solutions are warmed and infused using Klein needles until the tissues become firm, but not as turgid as in the tumescent technique.²⁰ The tissues blanch with the infusion containing adrenaline and lidocaine. Whether the wetting solution actually causes cell hydrolysis is still a matter of conjecture. It is important to warm the solutions to 37°C to prevent hypothermia. The usual infiltration is a 1:1 or 1:1.5 of the total aspirate volume anticipated. The solution is 1 mL of 1:1000 adrenaline, 20 mL of 2% lidocaine and 1000 mL of Hartmann's (Ringer's lactate) solution.

Incision placement

For the arms, a small incision in the posterior elbow and then one in the axilla is needed. For the buttocks, small incisions, generally 2–3 mm long, can be placed in creases and for the chin, a small incision underneath the mentum and one behind the ear facilitate a criss-crossing pattern of the cannula insertion. In the abdomen, incisions in the umbilicus and in the suprapubic region facilitate the same criss-cross patterning and liposhaping. Incisions for traditional liposuction, being only 2–3 mm long, are sometimes left open by some surgeons or closed with one suture to allow for drainage of the excessive fluid which remains within the tissues at the end of the procedure.

Progress of the procedure – technique

After induction of anaesthesia and positioning on the operating table, the area to be liposuctioned is prepared with diluted betadine spray until the whole area is a light brown with the markings and designs are still visible. Sterile drapes are placed around the area to be aspirated and the procedure, no matter what technique is used, progresses, utilizing visual and tactile senses to shape the particular area. By carefully observing the aspirate from the areas, excessive bleeding is prevented. When the 1 L jars are filled, they are changed and left to settle to observe the true total aspirate of fat versus infusion fluid, which is usually a light pink colour due to a small amount of blood also being aspirated.

When each position is completed, the patient is turned and supported on a bean bag, which is suctioned until it is firm so that the patient can be stabilized in both lateral decubitus positions. This position was suggested by Gasparotti¹⁴ because it accurately sculpts the buttock, thigh and hip areas.

The anaesthetist should maintain the blood pressure at about 10–20 mmHg below the patient's normal blood pressure. Urinary output monitoring is advisable for larger volumes, for longer time procedures or combined procedures.

Calf compressors are used both throughout the procedure and also in the recovery room.

Ambient room temperature should be maintained at 26°C and a heating blanket should be available during and after the procedure to maintain the body core temperature as close as possible to normal, as hypothermia is an ever present danger in large liposuctions or liposuctions lasting for an extended period of time with extensive exposure of the trunk. Antibiotics are administered intravenously (1 g cephalothin or cephalazolin).

Dressings, compression and postoperative management

After closure of all the access ports with an absorbable suture, a gauze dressing and Tegaderm are applied. There will be some fluid leakage from the incisions overnight. A compressive garment is applied by the nursing staff on the operating table and the patient is transferred to the recovery room. Analgesia will be needed after the lidocaine within the wetting solution has dissipated and then the patient is instructed to take oral medications which are individualized to the patient's needs.

All patients are outpatients and accompanied postoperatively by an adult carer to a facility close to the clinic. Contact with nursing staff and the surgeon is maintained by telephone and is available overnight. The patient is reviewed in the clinic in the morning and the dressings are changed. The patients remove their garment to shower and reapply the dressings on a daily basis, after which the garment is put on again.

Garments can be a special order but those commercially available, such as SPANX, are suitable for the majority of patients. Most patients tolerate the garments well but are pleased to be relieved of them by six weeks. They usually wear them continuously for two weeks and taper off their wearing of them over the next few weeks.

Adjunctive measures postoperatively

Early ambulation and return to normal activities are encouraged. Although bruising will resolve usually within 2–3 weeks, swelling may persist in the hips, thighs and abdomen, and lymph drainage, massage and ultrasonic treatments by a qualified physiotherapist are recommended for persistent swelling and tissues that are 'woody', indurated or bruised.

Serum needle aspiration is also indicated for collections or suspected collections of serum in the sacrum, back or hip areas. Encouraging return to regular exercise helps to facilitate swelling resolution as well as mental wellbeing.

Complications

Immediate

Haematoma and infection are generally rare, but tragic cases of necrotizing fasciitis have been reported.²¹ Venous thrombosis and pulmonary embolism are an ever present danger which should be anticipated in thrombosis-prone patients.²² Screening by history, examination and laboratory testing should identify patients who are particularly at risk and consideration should be given not only to calf compression, calf stockings and early ambulation but also low-dose heparin subcutaneously in the postoperative period for those patients deemed high risk. An episode of tachycardia or difficulty breathing are other classical signs of pulmonary embolus,²³ and a subtle elevation of temperature, malaise or failure to progress all might suggest a pulmonary embolus. Radiological evaluation, V/Q scanning or CT angiography to rule out pulmonary embolism may be necessary.

Shortness of breath in the immediate postoperative period might also alert the physician to fluid overload or an iatrogenic pneumothorax, in which case the patient should be investigated appropriately with the help of the anaesthetist. If the patient is deteriorating, transfer to a major hospital with emergency room and intensive care facilities must be considered and expedited with alacrity.

Other iatrogenic injuries include rare but documented viscous perforation, if the surgeon unwittingly enters the peritoneal cavity. The risk may be minimized by fastidious preoperative evaluation of the abdomen, including an ultrasound, especially if a previous surgical incision and possible incisional hernia is suspected. A wide diastasis of the rectus muscles after pregnancy could also be a prelude to inadvertent entry into the abdominal cavity. A patient presenting with an acutely painful abdomen after abdominal liposuction could herald this disastrous complication, needing emergency laparotomy and bowel repair.

These cases may be followed by significant infection of the abdominal wall, resulting in necrosis and requiring extensive debridement and reconstruction. A fatal outcome could be the result of such a horrific complication. High index of suspicion and expedited general surgical intervention is vital.

Hypotension in the postoperative period could be due to excessive blood loss. Knowing the preoperative haemoglobin and carefully assessing blood loss in the aspirate should indicate where a blood transfusion might be needed. Postoperative blood work should be ascertained to assess whether the haemocrit has fallen and the patient may require blood transfusion.

Plasma expanders as a volume substitute may be sufficient in a hypotensive patient or be expedient prior to transfusion, although such a sequel is very rare.

Beyond week 1

Seroma formation in areas of firmness and deep tissue induration or infections, can occur and this necessitates the careful monitoring of patients in the immediate postoperative period. Aspiration of seroma and antibiotic coverage for collections if present, are indicated.

Necrotizing fasciitis/toxic shock syndrome

These conditions are fortunately rare, but an awareness of them and a rapid response, referral to an emergency department, in particular to an infectious disease consultant and debridement are the likely sequelae to prevent progression and a possible fatal complication.^{21,24}

Other early complications

The most common problem in the first few weeks postoperatively is swelling and bruising and small seroma collections, which might require needle aspiration.²⁵ Referral to a physiotherapist for lymph drainage and/or ultrasound and massage is often very helpful physically and psychologically.

Late complications

Contour irregularities are less common with more experienced surgeons but if certain areas do not resolve, where the skin is quite adherent in the lateral thigh, posterior thigh or in the medial thigh, secondary liposuction and/or lipofilling may be needed to correct the irregularities.⁷ Usually surgeons rely on time, massage and exercise to smooth them out. Lack of skin contraction and loose redundant skin may require secondary skin resection and lifting procedures, but usually not until 9–12 months after the liposuction, to facilitate maximum skin retraction and tissue resolution.

Overzealous or novice surgeons must avoid removing too much fat, which can cause buttock ptosis or leave linear or circular defects and depressions, requiring secondary remedial surgery, such as liposuction, lipofilling or resective surgery.

Numbness and paraesthesia can occur in any area; however, the hips and arms are quite prone to this annoying sequela. The numbness and paraesthesiae are usually self-limiting within 2–3 months.

Results

Postoperative results are usually photographed six months after the procedure when the patient has resumed all their normal activities. Good results in a male patient's abdomen and a female in the typical areas, namely the hips, thighs and buttocks, are shown in Figures 75.2 and 75.3.



Figure 75.2 Abdominal liposuction in male. (a, b, c) Before; (d, e, f) after.

Secondary procedures

Touch-up procedures should be delayed for six months or more after the initial procedure.

Weight gain should be checked in the postoperative period to ascertain whether fat is residual or newly acquired. Irregularities may require lipofilling, which is available as the best remedial option if a patient's results can be realistically improved.

Certain areas, especially the buttocks, can sag after liposuction and the 'banana roll' is prone to being overresected. A 'hanging' buttock is 'ageing' and might require skin excision, although the scar after excision is undesirable for most patients. Surgical wedge excision is the only acceptable alternative for improvement in some of these patients, however, and the scarring and down side of this surgery may well convince a patient to be satisfied with the improvement in their shape and to be able to accept some degree of looseness of their skin.



Figure 75.3 Liposuction in female – abdomen, hips and thighs. (a, b) Before and (c, d) after.

Summary

In a woman, the creation of smooth and well-defined curves will always improve their figure. As surgeons, we aim to improve the shape of our patients, but the results of our efforts are closely linked to the expectations of each patient being realistic rather than unrealizable. In a male, a more angular and muscular shape is sought, paying attention to the definition of abdominal and chest wall muscularity.

Improvements in shape and figure generally make patients feel more comfortable about themselves, enabling more versatility wearing different clothes, sporting fashions and/or swimwear. Liposuction has established itself as the most valuable

body-contouring plastic surgical procedure available and the demand for it is not abating. Improved equipment and anaesthetic techniques have facilitated better and upgraded results since the introduction of this technique 30 years ago.

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CHAPTER 76

Abdominoplasty and body contouring

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Anatomy

A profound knowledge of the entire anatomy of the lower trunk and extremities is essential for aesthetic and reconstructive skin-tightening procedures. In addition to knowledge of the main perforating vessels, the course of any important nerve is mandatory to avoid postoperative complications. Furthermore, the knowledge of Borgess lines (relaxed skin tension lines) is crucial for the best possible scar development.¹

Skin

Abdominal skin and fat tend to become distributed in the lower abdomen with ageing and pregnancy. Striae also become common, especially as a result of multiple pregnancies, resulting in a rupture and separation of dermal collagen with consequent skin thinning. Following post-bariatric weight loss, patients have been shown to have pre-damaged skin in recent studies evaluating the content and structure of collagen and elastin compared to patients without bariatric procedures.

The skin of the abdomen has areas of increased adherence to the underlying fascia ('zones of adherence') in the vertical midline area, and occasionally in a horizontal direction just superior to the umbilicus (supra-umbilical skin fold), as well as in a horizontal direction above the anterior superior iliac crest.

Fascia

The abdominal subcutaneous tissue is divided by two layers of fascia: the superficial Camper's fascia and the deep Scarpa's fascia, a strong fibrous layer of connective tissue which is continuous with the fascia lata of the thigh.

Fat

The superficial fat layer has a more compact character with smaller lobules and a rich vascularization, while the deeper fat layer contains larger lobules with a more scattered pattern.²

The umbilicus

In most patients, the umbilicus is probably the most important landmark on the abdominal wall. It is normally situated in the midline, approximately 9–12 cm above the upper border of the mons pubis region. A round or ellipsoid shape may characterize it, in general with a depression of 4–6 cm in diameter. The abdominal fascia in this region can present as weak with an increased incidence for hernias. The blood supply derives from branches of the subdermal plexus, from both deep inferior epigastric arteries as well as a blood supply from the median umbilical ligament. In general, the umbilical stalk is blunted and dissociable from the surrounding fat tissue.

Musculoponeurotic system

The abdominal musculature includes four paired muscles: the *rectus abdominis*, connected in the median linea alba, the *external oblique* and *internal oblique* and the *transversus abdominis* muscle, which incorporate into the anterior and posterior rectus sheath at the linea semilunaris. The rectus abdominis muscles are enclosed by an anterior and posterior sheath, originating at the infracostal margin and attaching at the symphysis pubis. Above the arcuate line (situated approximately midway between umbilicus and pubic symphysis), the anterior rectus sheath is formed by the external oblique and internal oblique aponeurosis, whereas the posterior rectus sheath is formed by the internal oblique and transversus aponeurosis. Below the arcuate line, the posterior rectus sheath is absent, except for the transversus fascia and the peritoneum. At the inferior aspect of the rectus muscles, the pyramidalis muscles are present in 80–90% of patients (Figure 76.1).

Lymphatic drainage

The abdominal lymphatic system is divided into supra-umbilical drainage to the ipsilateral axillary lymph nodes and an infra-umbilical drainage into the superficial inguinal lymph nodes. The lymph vessels in the infra-umbilical area pass through the subscarpal plane, explaining the importance of Scarpa's fascia preservation in abdominal wall surgery.

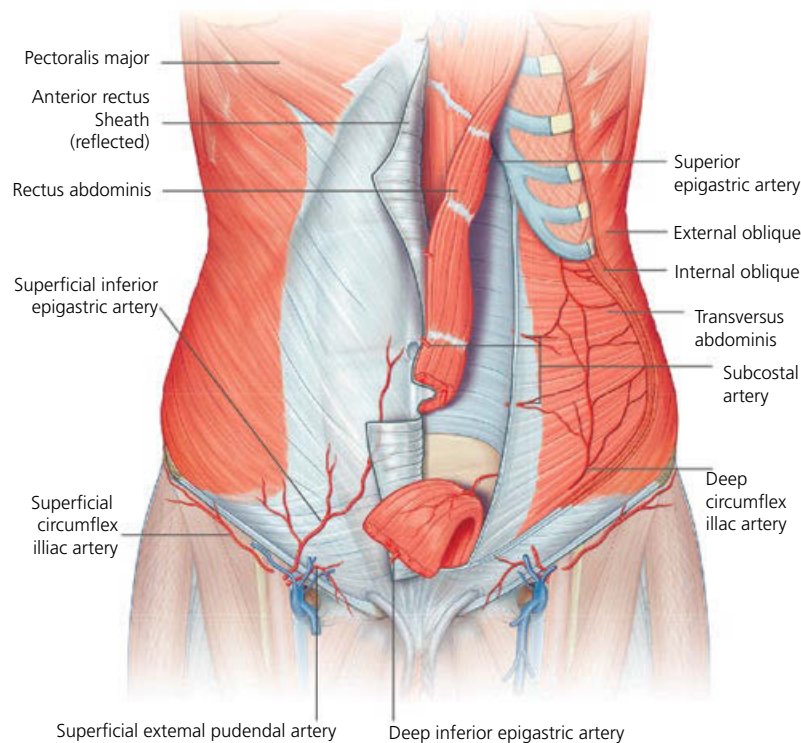


Figure 76.1 Anatomy of the abdominal musculature with abdominal blood supply. Source: Plastic Surgery, Volume Two, Third Edition, Peter Neligan (ed.). Reproduced with permission of Elsevier.

Blood supply

The blood supply to the abdominal wall comes from numerous major arteries of the thorax and pelvic region, which communicate through many anastomotic interconnections. Two types of cutaneous blood supply are known: direct vessels that supply the skin directly and indirect vessels that 'emerge from the deep fascia as terminal spent branches of arteries whose main purpose is to supply the muscles and other deep tissues'. Moon and Taylor were able to demonstrate connections between the deep superior and deep inferior epigastric systems and their relationship to the cutaneous circulation.³⁻⁵

A detailed knowledge of the arterial supply of the abdominal wall is crucial to abdominal wall surgery, especially in cases of prior abdominal wall surgeries. Huger described different zones of the abdominal blood supply which should guide the surgeon in planning and performing a safe operation.⁴ Huger defined zone I of the abdominal wall as the area that is fed anteriorly by the vertically oriented deep epigastric arcade. Zone III was defined as the lateral aspect of the abdominal wall (flanks) that are fed by the six lateral intercostal and four lumbar arteries. The lower abdominal circulation is provided by the superficial epigastric, superficial external pudendal, and superficial circumflex iliac systems (zone II). A rich plexus between these systems allows collateral flow (Figures 76.1 and 76.2).

In standard abdominoplasty procedures, the blood supply to zone I and main parts of zone II is disrupted after flap mobilization, resulting in an abdominal flap perfusion mainly supplied by zone III. The lateral perforating branches from the lateral

intercostals and lumbar arteries are essential for the remaining flap perfusion. Therefore it is crucial to study any preoperative existing scar, such as subcostal cholecystectomy incisions (Figure 76.2).

Nerve supply

The sensibility of the abdominal wall derives from the anterior and lateral cutaneous branches of the 8th to 12th intercostal nerves, which reach the skin and fascia by passing between the internal oblique and transversus abdominis muscles and finally through the rectus abdominis muscle. The lateral cutaneous branches penetrate the intercostal muscles in the mid-axillary line, ending in the subcutaneous layer.

The motor branches of the rectus abdominis muscle derive from lateral branches of the intercostal nerves 5–12, which pass behind the rectus muscles and enter the muscles at the junction of the lateral 1/3 and medial 2/3. The oblique and transversus abdominis muscle are innervated by branches from the lower thoracic and lumbar dorsal nerves (Figure 76.3).⁵

Peripheral nerves such as the iliohypogastric and ilioinguinal have to be recognized, since they may be injured in lateral transverse lower abdominal incisions, resulting in consistent loss of sensory in the area of the groin and medioventral thigh.

Anatomical landmarks

Normal abdominal anatomic proportions (Figure 76.4):

- The umbilicus is located in line with the most superior point of the iliac crest in 99% of patients.

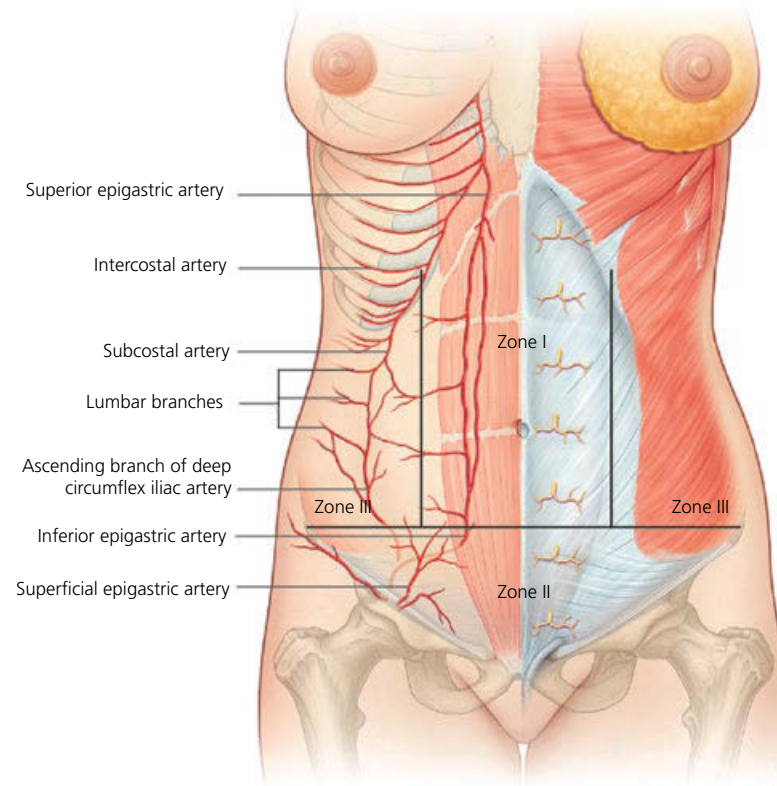


Figure 76.2 Huger's zones of superficial abdominal blood supply. Source: Plastic Surgery, Volume Two, Third Edition, Peter Neligan (ed.). Reproduced with permission of Elsevier.

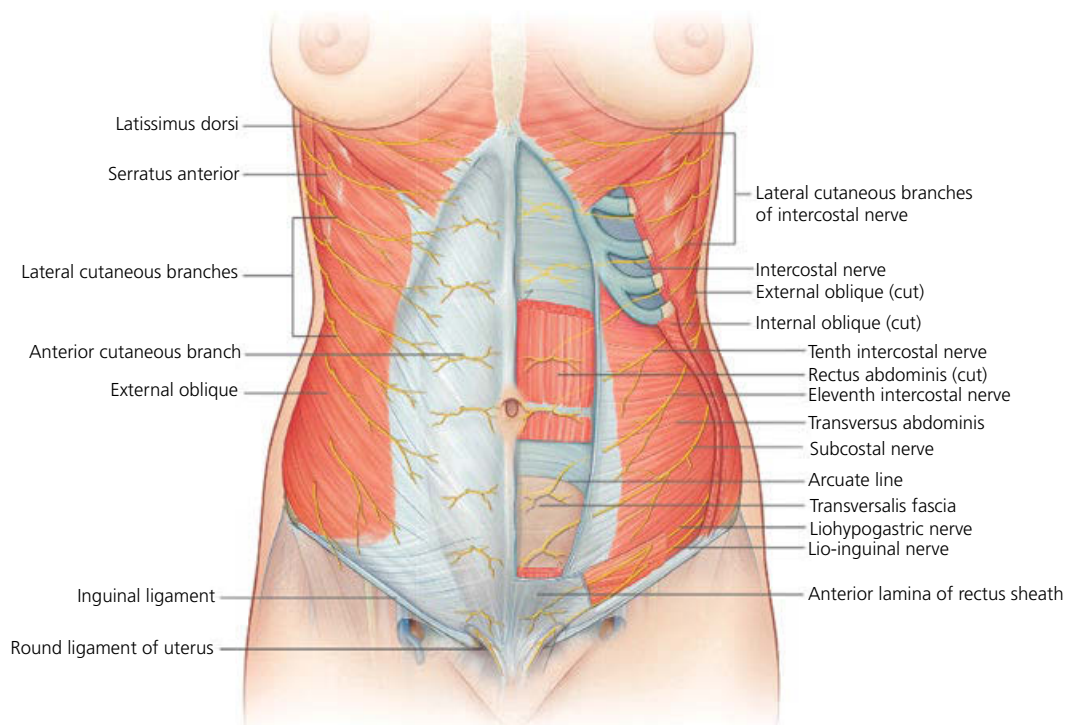


Figure 76.3 Anatomical nerve supply. Source: Plastic Surgery, Volume Two, Third Edition, Peter Neligan (ed.). Reproduced with permission of Elsevier.

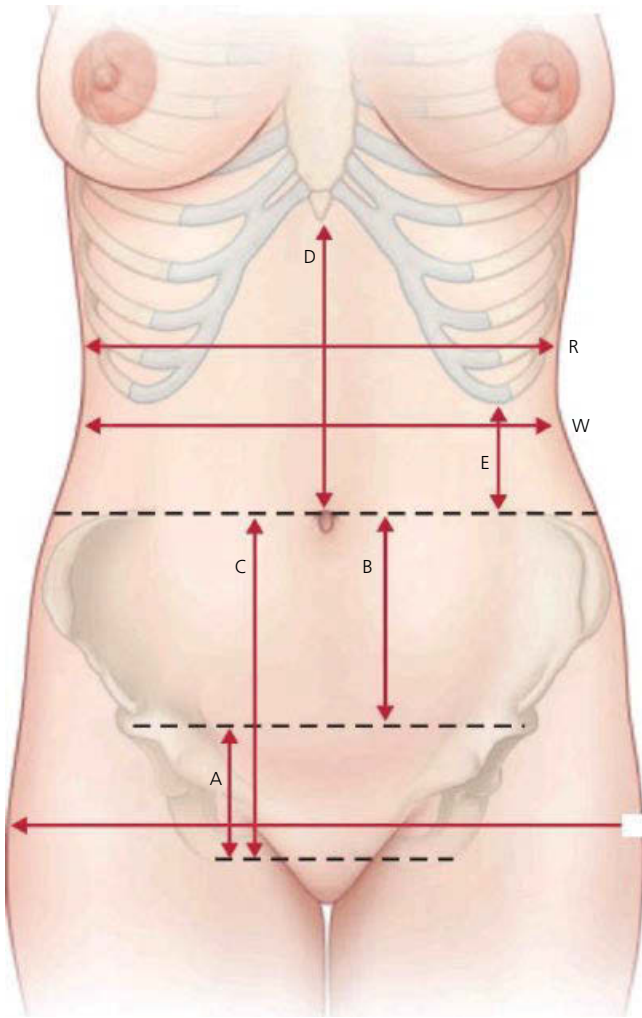


Figure 76.4 Anatomical landmarks with normal anatomical proportions.

- Distance between the top of mons and anterior valvar commissure (A): average height is 5–7 cm.
- Distance between umbilicus and top of mons (A + B): average height is 11–13 cm.
- Distance between umbilicus and top of anterior and vulvar commissure (C = D).
- Distance between the costal marginal and the iliac crest (E). The proportion of this distance to the width of the base of the rib cage (R) determines whether the patient is long waisted or short waisted. The normal proportion (E:R) is roughly 1:3 (long-waisted approaches 1:2, short-waisted approaches 1:3).
- The rib cage tapers inferiorly. A narrower lower rib cage relative to the width under the armpits helps to emphasize the waist by creating a subtle V.
- Hip width (H). A wider pelvis than rib cage emphasizes the waist; the waist is more defined when $R < H$.
- Natural waist (W) – the narrowest point of the torso. (Note that the umbilicus usually sits below the natural waist

by about 1–4 cm.) Relative to the hips, this waist-to-hip (W:H) ratio in healthy women is roughly 0.72:1; in healthy men, it is roughly 0.83:1. Note that the natural contour of the healthy abdomen reveals a subtle epigastric sagittal depression transitioning to a mild infra-umbilical convexity. A subtle vertical sulcus at the lateral rectus border, which is more distinct in a muscular person, may also be seen (Figure 76.4).⁶

Aesthetic assessment

Abdominal wall

The aesthetic assessment of the abdominal wall includes:

- regions of local adipose tissue and their thickness in centimetres,
- local adipose tissue in the upper and central abdominal region,
- regions of local skin excess,
- local skin excess in the upper and central abdominal region,
- presence of skin folds and their depths,
- areas of maximal looseness and adherence,
- local adipose tissue in the adjoining regions of the flanks, hips and breasts,
- local adipose tissue in the mons pubis region,
- skin laxity in the mons pubis region,
- any asymmetric aspects,
- any bulginess,
- any pre-existing scars and their quality, and
- presence and degree of striae.

Umbilicus

The aesthetic assessment of the umbilicus includes:

- shape and depth of the umbilical stalk,
- position of the umbilical basis on the abdominal wall (to be demonstrated to the patient),
- height of umbilical descent,
- any existing umbilical herniation or adipose tumour,
- any existing eczema, hyperpigmentation or pre-existing scars, and
- any prior operation in the umbilical region has to be clarified, since impaired perfusion after dissection may occur in these cases.

Back, thighs and buttocks

The aesthetic assessment of the back, thigh and buttocks includes:

- regions of local adipose tissue and their thickness in centimetres in the back,
- mobility of the dorsal skin in the lower back region,
- local skin excess in the back region,
- presence of dorsal skin folds and their depth,
- local adipose tissue in the gluteal region,

- regions of local skin excess, measured by pinching in vertical distance in the gluteal and flank region,
- presence of gluteal skin folds and their depths,
- areas of maximal looseness and adherence,
- local adipose tissue in the anterior, posterior, lateral and inner thigh region,
- skin laxity in all thigh regions,
- presence of skin folds in the thigh region (e.g. inner thigh, distal anterior),
- any asymmetric aspects,
- any bulginess,
- any pre-existing scars and their quality,
- any existing eczema or hyperpigmentations, and
- presence and degree of striae.

Patient assessment

History

During the patient's first consultation a review of the medical history is mandatory. We recommend a repeat of evaluation prior to surgery, if it is performed with a delay since the first consultation. The assessment of medical history includes the current weight, the ABSI (a body shape index) and body mass index (BMI), the weight history with weight fluctuations and constancy periods, the frequency of exercises, former bariatric procedures, nutritional disorders, medication, the number of pregnancies and children, history of caesarean section, abdominal surgeries and abdominal hernias, gastrointestinal, cardiac and pulmonary history, and a smoking history. Patients should be asked specifically about previous liposuction in the abdominal area, since they might conceal this prior treatment.

Physical examination

Physical examination is part of every medical history assessment. It evaluates the entire region the patient is concerned about. An examination of the skin tissue and the different fat layers is performed by pinching and measuring the subcutaneous layer thickness. Skin quality with the presence of striae should be evaluated, clarifying with the patient that supra-umbilical striae will not be resected in abdominoplasty procedures (except for fleur-de-lis procedures). In this context, it is helpful to hand out studies on skin quality in post-bariatric patients that have demonstrated damage to collagen structures.^{7,8}

Next, it is important to assess and measure the lower and upper abdominal pannus, documenting any existing eczema or consequent hyperpigmentation. The entire skin excess in the areas inferior to the lower abdominal fold, in the area of the lateral and upper abdomen, the waist, hips and thighs as well as the lower chest have to be included in the preoperative assessment, during upright standing, supine as well as sitting positions. Any pre-existing scars (subcostal, midline, horizontal) in the abdominal area have to be documented, since they can impair the blood supply. Furthermore, the status of the

abdominal musculature should be assessed and any existing rectus diastasis, incisional, epigastric or umbilical hernia excluded. Supportive CT or MRI scans may be performed.

In this context, it is mandatory to evaluate intra-abdominal pressure to avoid any postoperative pressure increase or abdominal compartment syndrome. In cases of elevated intra-abdominal pressure, in the supine position, the abdominal wall elevates above the costal margin and the level of the iliac crest. Abdominoplasty procedures, especially with fascial reconstruction have to be performed cautiously in such cases.

Documentation

It is mandatory to ensure a complete photographic documentation pre- and postoperatively, consisting of different views (anterior, oblique, side and posterior). Postoperative photo documentation can be made at 3, 9 and 12 months. In individual cases it is recommended to perform additional photo documentation of certain arm and thigh positions, tissue characteristics with relaxed and contracted muscles, the patient in sitting position to demonstrate redundant upper abdominal laxity and tissue excess, additional views with the patient holding up and pinching excess tissue in the specific region.

Informing the patients in detail about the operative procedure, alternative techniques and the general and operation-related risks and benefits is an essential part of preoperative documentation. Besides standard and individualized consent forms, which have to be reviewed and signed by the patient at the earliest opportunity and at the latest 24 h prior to the operation, we recommend an audio-visual demonstration for exact understanding. In general, we recommend informing every patient ruthlessly about any imaginable complication. Moreover, patients have to be informed about their postoperative care, including their expected level of activity, and the limitations of the surgical result in cases of existing variables of bone structure, fat distribution and any existing scars.⁹⁻¹¹

Aesthetics

The preoperative aesthetic status of the body region of concern has to be well documented in written and photographic form after a precise physical examination. For a classification of the different deformities, we found the Pittsburgh Rating Scale to be helpful for patient demonstration and selection of the best operative approach.¹² However, we experienced a wider indication for the combined technique of liposuction and excisional procedures in all body regions.

General

Patients are advised to stabilize their weight for at least 6–12 months preoperatively; any desired weight loss should be completed prior to the surgery. Smokers should stop smoking at least six weeks prior to surgery. Patients should take antiseptic showers in the evening and morning prior to the surgery; the skin folds and the umbilicus should be cleaned thoroughly with antiseptic solutions.

If a postoperative blood transfusion is considered (e.g. body lifts), patients have to be informed preoperatively, as this requires a blood typing and informed consent. Anticoagulant drugs and supplements must be avoided 14 days prior to surgery. In cases of a planned abdominal procedure, we advise patients to perform bowel purgation the night before surgery. In severe cases, patients can be restricted to a fluid diet for 24 h prior to surgery.

Patients have to be informed about the intraoperative procedures, including any change of position with the risks of postoperative complication, antithrombotic arrangements, Foley's catheter, drains and garment placement, as well as patient-controlled analgesia. Moreover, they have to be instructed about the postoperative course, regarding thromboembolism prevention, respiratory exercises, early mobilization, avoidance of high abdominal pressure, the estimated time point for drain and suture removal as well as the minimum time required off work and away from exercise. Also, it has to be noted, if the operation cannot be performed as an outpatient, how long the patient will expect to have to stay in hospital.

Diabetes may be a relative contraindication to this procedure, especially in poorly controlled patients. Furthermore, it is important to exclude any skin irritation or inflammation in the area of the abdominal fold and umbilicus. An inspection of these areas during consultation, as well as immediately prior to the operation is strongly advisable.

Prophylaxis for pulmonary embolism and deep vein thrombosis is an essential consideration, especially in smokers and patients taking birth control medications or hormone replacement therapy.¹³ We initialize heparin therapy in all patients the evening before surgery or at latest 2 h prior to the surgical procedure. We inform patients that this specific risk will be decreased if hormonal therapy is discontinued 3–4 weeks prior to surgery.

In cases of an existing extended diastasis or hernia, the reconstruction of the fascia results in an increased intra-abdominal pressure, which may be a source of respiratory difficulties. Therefore, we advise preoperative breathing exercises (utilizing an incentive spirometer) beginning 1–2 weeks prior to and continuing for 2 weeks after the surgery. A cold, dry cough or any kind of respiratory infections should lead to postponement of surgery, since fits of coughing may provoke a rupture of the fascial sutures with consequent secondary bleeding.^{9,10,14}

Operative principles and approach

Various procedures are available with various indications as listed in Table 76.1.

Liposuction

The indication for sole liposuction of the abdomen is seen very rarely. In patients with an abdominal contour impaired by an irregular and solely thickened abdominal fat layer, liposuction may improve the abdominal shape dramatically. Nevertheless, as soon as a patient highlights his or her frustration due to a laxity of skin, it will not be worthwhile to perform liposuction without simultaneous skin excision. Ideal cases for liposuction are patients with localized adipose tissue in the flank/hip region, the infra- and/or supra-umbilical region. They can be optimally treated by liposuction under local anaesthesia as an outpatient. For achievement of maximum outcomes, it could be reasonable to acquire one of the assisted liposuction techniques nowadays available (e.g. laser-, ultrasonic- or radiofrequency-assisted liposuction), which allow simultaneous skin tightening.

In all cases of liposuction in the abdominal region it is mandatory to exclude any existing rectus diastasis or abdominal hernia. If required, diagnostics should include an ultrasonic scan, CT or MRI (Figure 76.5).

Any adjunctive liposuction of the abdominal wall should be performed prior to the abdominoplasty procedure in order to sufficiently thin out the different layers, including the epifascial and subfascial layer.¹⁵ It is important to assess the relation of excess skin and fatty tissue, since in cases of extensive skin excess in the upper and mid-abdominal region a fleur-de-lis procedure may be indicated. In these cases, there is usually no necessity for liposuction in the abdominal region, since a maximal amount of abdominal tissue is resected.

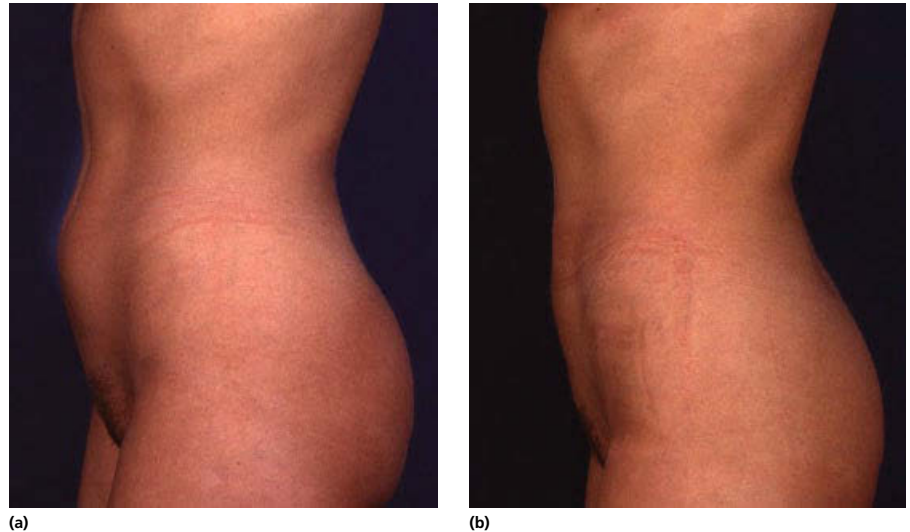
Adjoining regions (e.g. the flanks and hips) may be treated by liposuction during the abdominoplasty procedure, preferably at the end of the operation. Infiltration of tumescence solution should be performed at the beginning of the procedure and liposuction can then be performed after a sufficient time of exposure during the abdominoplasty procedure.^{6,11}

Table 76.1 Indications for different techniques dependent on redundant adipose tissue⁶

	Mini	Standard	HLT	Fleur-de-lis	Body lift	Reverse
Lower abdomen	+	+++	+++	+++	+++	0
Peri-umbilical	(+)	++	++	+++	++	(+)
Upper abdomen	0	++	++	+++	++	+++
Diastasis/hernia	(+)	++	++	+++	++	(+)
Flanks/hips/thighs	0	(+)	+	++	+++	0

HLT, high lateral tension.

Figure 76.5 A 37-year-old woman (a) with preoperative local adipose tissue in the lower abdomen without significant skin surplus. (b) Six months after vibration-assisted liposuction of 600 mL fat tissue.



Standard abdominoplasty

The standard abdominoplasty is one of the most common procedures in plastic surgery. It enables the simultaneous treatment of the upper abdomen, peri-umbilical area and lower abdomen with optimally concealable scars.

Former traditional abdominoplasty procedures involved wide mobilization of the abdominal wall up to the costal arch in order to ensure a tension-free wound closure. Multiple studies have since demonstrated that circulatory disturbance of the abdominal skin flap is the result of extensive undermining and destruction of the lateral perforators of Huger's zone II. In modern abdominoplasty undermining cranially to the umbilicus is mainly performed in the midline area anterior to the linea alba and both rectus muscles, including the zones of adhesion described by Lockwood and specified by Aly. The lateral abdominal flap may be securely released by additional liposuction or the Lockwood dissector for discontinuous undermining, preserving the lateral perforators and the lateral attachment to the rectus abdominis muscles.^{16–19}

The standard abdominoplasty is indicated in patients that typically demonstrate a localized and diet-resistant adiposity of the abdominal wall in combination with coexisting slack and atonic skin, such as patients after multiple pregnancies, patients with weakness of the connective tissue or patients after weight loss (Figure 76.6).

Markings

The markings are started in upright position with the midline, the borders of the patient's underwear and the expected and desired scar position. Next, the patient is asked to elevate the redundant abdominal tissue for identification of the abdominal fold. A vertical distance of 6 cm is measured from the upper vulvar commissure to the lower incision, followed by marking of the lower incision line parallel to the abdominal fold. By pinching of the abdominal tissue, the upper incision line is

marked next, which can be checked in supine position. It is advisable to mark parallel lines at various heights to ensure a symmetric resection. The patient should always be informed about the risk of an inverted T-scar in the midline if the resection does not completely include the umbilical incision.

Evaluation of possible pre-existing hernias and diastasis should be performed and marked in the supine position. Furthermore, the length of the scar line may be measured bilaterally starting from the midline to ensure symmetry. In this context the surgeon has to be aware that a longer inferior incision will lead to a gently curved upward course of the scar (Baroudi) and a longer superior incision will cause a straighter or downward course of the scar (Pitanguy).^{20,21}

Technique

The operation is started with a repetitive check and trace of the markings. It has to be ensured that the umbilical stalk is thoroughly cleaned.

Initially, the lower incision line is infiltrated with local anesthetics to reduce postoperative pain and the consecutive risk of blood pressure increase. After incision of the lower incision line, preparation is performed down to Scarpa's fascia after ligation of any superficial epigastric vessels. For adequate preparation of Scarpa's fascia, we recommend use of the Colorado® Microneedle and to ensure a sufficient upward pull of the abdominal flap. By preservation of Scarpa's fascia the subjacent lymphatic vessels are preserved. In general, we find a thin fat layer below Scarpa's fascia. Continuous dissection superficial to Scarpa's fascia is performed cranially up to a level as wide as 3–4 fingers inferior to the umbilicus. At that height, Scarpa's fascia is incised, and dissection continues down the anterior rectus sheath.

At the height of the umbilicus, the abdominal flap is incised in the midline and the umbilical stalk is transected circularly and completely mobilized (skeletonized without any remaining fat tissue) from the abdominal flap. Especially in patients after



Figure 76.6 (a, c) A 42-year-old woman with a significant abdominal pannus after three pregnancies and a fluctuant weight of 40 kg. (b, d) Three months after a standard abdominoplasty with a good contouring of the umbilical region.

massive weight loss, the umbilical stalk has to be shortened and fixated to the anterior rectus fascia at the height of the stalk's base, preferably with absorbable sutures at 3, 6, 9 and 12 o'clock positions. Further dissection continues superior to the anterior rectus fascia up to the xiphoid. The linea alba can easily be identified as it is distinctively connected to Scarpa's fascia. In the area of the costal margin, it is essential to restore the lateral perforators for sufficient flap perfusion. At this time point, after finalization of the abdominal flap mobilization, in cases of concomitant rectus diastasis, the plication of the anterior rectus sheath from the xiphoid to the symphysis is accomplished using non-absorbable or absorbable suture material in any preferred technique. In case of a diastasis at the lower abdomen, Scarpa's fascia and underlying tissue may be bluntly mobilized to allow for further midline fascial plication (Figure 76.7). For cases of asymmetrically located umbilical stalk or for further accentuation of waist tightening, an additional paramedian plication of the anterior rectus sheath may be facilitated. Since lateral dissection is avoided up to the costal arch for preservation of the lateral perforators, a discontinuous separation of lateral adhesions using the Lockwood underminer may enable a further flap mobilization.

During tissue resection, bullet forceps or a Pitanguy tissue demarcator can support the estimation of the upper resection line. Further evaluation of the amount of resection may be facilitated by additional vertical flap incisions and temporary wound closure utilizing bullet forceps. The upper incision line is then marked and the resection is performed oblique to the wound edge in a 45° angle to enable a more precise adaptation of varying thick layers. In selected patients with a pronounced abdominal fat layer the fatty tissue beneath Scarpa's fascia should be resected in the entire mobilized area. This may avoid postoperative complications due to superinfected fat necrosis.

After meticulous coagulation the wound is closed temporarily. The new umbilical position is estimated and marked. Several umbilical incision patterns have been described; we prefer the superior based inverted V or U incision. This umbilical section of the abdominal flap is then circumferentially defatted for a circular umbilical depression. The supra-umbilical midline may also be reduced through midline liposuction or open excision. After repeated meticulous haemostasis, Scarpa's fascia is sutured down to the anterior rectus fascia with absorbable progressive tension sutures (Baroudi stitches), starting

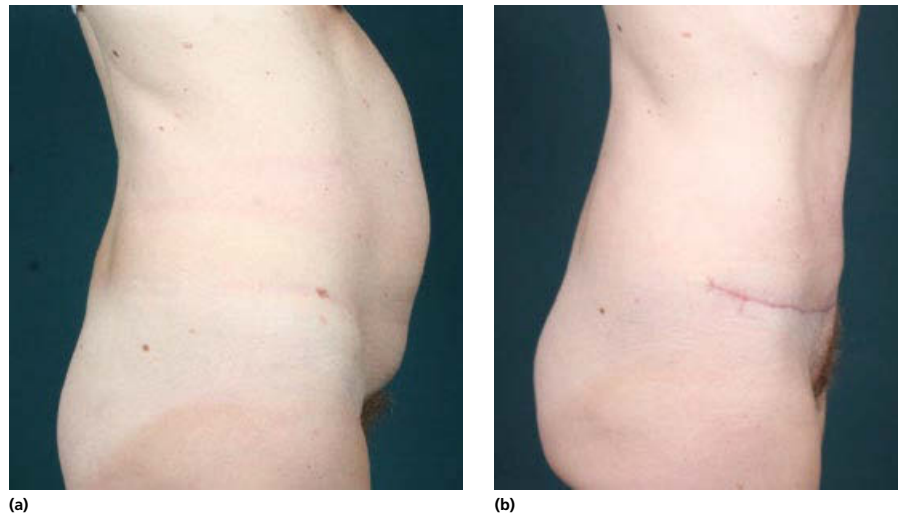


Figure 76.7 (a) A 42-year-old woman after two pregnancies and a subsequent rectus diastasis. (b) Three months postoperatively after fascial tightening and standard abdominoplasty.

superior to the umbilicus. Alternatively, the supra-umbilical subcutaneous tissue may be sutured in the midline to the anterior rectus fascia to accentuate a midline depression for superior aesthetics. After the umbilical stalk is pulled through the new umbilical tunnel, additional progressive tension sutures are placed in the lower flap area.

Finally, the patient is bent into beach-chair position and temporary wound closure is performed with bullet forceps, starting laterally to medial. In order to avoid dog-ears, the longer superior wound edge is shifted slightly medially to adapt to the shorter inferior. After the placement of 2–3 drains the final wound closure is performed for all layers, starting with a reconstruction of the superficial fascial layer with absorbable sutures, followed by a subcutaneous readaption and completed with intradermal running sutures. For time reduction in wound closure the use of absorbable barbed sutures (e.g. Quill®) is routinely advisable, such as PDO 2×0 for fascial and subcutaneous closure and Monoderm 3×0 for intradermal closure. Optionally, skin closure time can be drastically reduced applying the two-component skin closure system PRINEO™.²²

After a final wound antiseptic cleaning, suture strips are affixed transversely to the scar. These supportive dressings should remain for three weeks postoperatively. Finally, a sterile wound dressing and an adjustable compression girdle are applied.

We prefer to perform the standard abdominoplasty procedure on an inpatient basis with a minimum 1–3 nights' hospital stay. A slight postoperative discoloration of the umbilicus is usually not precarious, especially when there was no previous surgery of the umbilicus. A complete necrosis of the umbilical stalk is seen very seldom and may be treated conservatively with satisfying results.

Patient satisfaction after standard abdominoplasty is usually high due to a possible treatment of the entire abdomen.

However, the legacy of a long scar with limited treatment effects to the hips and the back region is an unfortunate aspect of this procedure for a certain patient group. In cases of fascial midline plication a tissue gathering may result in the epigastric region. This tissue excess may be easily and safely reduced by open excision or additional liposuction.^{6,11}

Mini-abdominoplasty

The mini-abdominoplasty differs from a standard abdominoplasty in a shorter resulting scar and restricted skin resection. The results of the short scar technique were limited until adjuvant liposuction and endoscopy-assisted procedures had been added for improved results. In this procedure, the extent of mobilization can be increased and the umbilical stalk may be released pre-fascially for inferior repositioning to the anterior rectus fascia (Figure 76.8).

This procedure is indicated in patients with a mild to moderate skin laxity and tissue excess of the lower infra-umbilical abdomen, together with a sufficient distance between the umbilicus and symphysis, mostly more than 12 cm. Liposuction of the upper abdomen and flanks has a supportive effect by improving the abdominal contour. In cases of coexisting diastasis recti or epigastric herniation they can be treated through an endoscopic approach. With respect to vascular anatomy, the mini-abdominoplasty is a safe procedure since a single Huger's zone (zone II) is compromised. In cases of peri-umbilical skin laxity, an umbilical transection can be performed to allow a more effective tightening and an improved visualization of the diastasis recti (Figure 76.9).

Markings

Markings are performed with the patient in the upright standing position. The distance between the umbilicus and the upper incision line should respect a minimum distance of

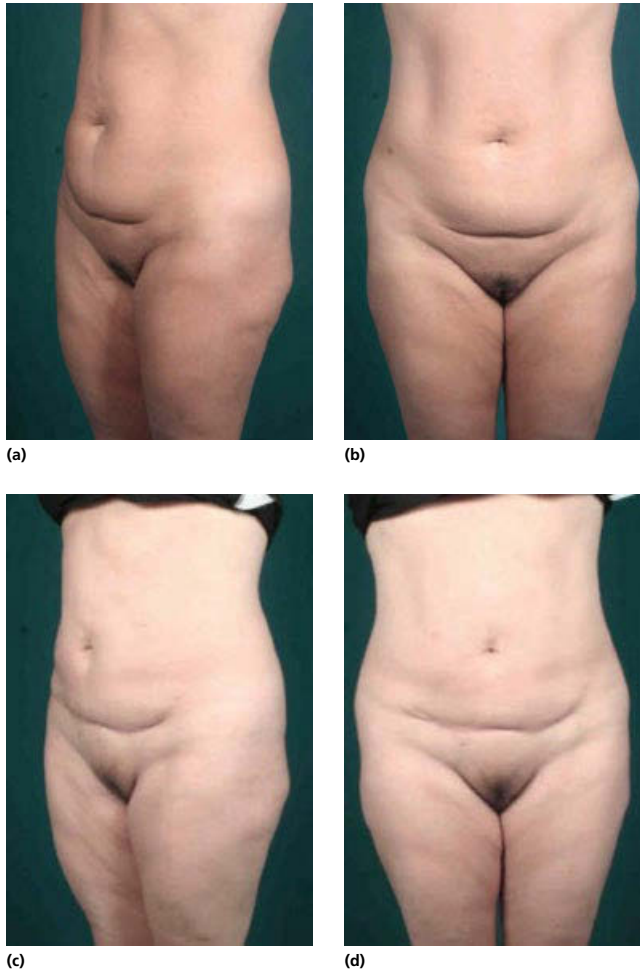


Figure 76.8 A 31-year-old patient after two pregnancies with two caesarian sections. Pre- (a, b) and postoperative (c, d) oblique and front views of a short scar (mini) abdominoplasty without umbilical transection. Note the low placement of the previous scar.

9 cm to avoid an unaesthetic appearance. For cases with redundant tissue in the lower abdomen and a relatively high position of the umbilicus (approximately 14–18 cm above the pubic symphysis), the lower incision line is marked 6 cm above the labial commissure and the upper incision line 9 cm below the umbilicus. In cases of a distance less than 9 cm after skin resection, an inverse T-scar with umbilical transposition should be preferred.

Regarding the required scar length, the fat distribution in the region of the iliac crest should be evaluated preoperatively and the patients has to be informed about an optimized cosmetic outcome despite a prolonged final scar length.

Technique

This procedure can be performed under local anaesthesia and adjuvant sedation. If a reconstruction of the anterior rectus sheath is required, we recommend general anaesthesia or spinal anaesthesia.

Any supportive liposuction of the abdominal wall and flanks should be performed prior to the abdominoplasty procedure in order to sufficiently reduce the epi- and subfascial layer. Techniques such as radiofrequency- or ultrasonic-assisted liposuction may generate shrinking of the skin for additional skin-tightening effect. In young patients with an elastic skin tone and a long distance between the umbilicus and symphysis an umbilical transposition may be avoided.

The operative technique is similar to a standard abdominoplasty, although abdominal flap mobilization is performed adjusted to local tissue characteristics. In all cases, tissue dissection is performed above Scarpa's fascia. The abdominal flap is mobilized cranially up to the level of the umbilical stalk. If a supra-umbilical rectus diastasis was diagnosed preoperatively we recommend a transection of the umbilical stalk from the anterior rectus fascia and continuing the dissection to the xiphoid and costal margins. The umbilical base should be marked for its refixation. After plication of the anterior rectus fascia the umbilical stalk is fixated to the abdominal wall. For this manoeuvre non-absorbable or absorbable suture material can be used. The abdominal flap is drawn caudally and the umbilical range of motion can be assessed. Hereby, the umbilicus may shift caudally. Prior to the final determination of the upper incision line the fixation of the umbilicus should be completed. Markings should always be done on stretched skin and adhere to a distance of minimum 9 cm between umbilicus and upper incision line.

In patients without a diastasis recti or epigastric hernia flap mobilization is performed cranially up to the umbilical stalk. The estimation of the amount of tissue resection and wound closure are always performed in the same manner as described above. In cases of fascial reconstruction or hernia repair drains may be inserted, in simple procedures without any adjoining procedure they may be dispensable. Patients with a diastasis recti and consequent potbelly with mild skin excess may be appropriate candidates for this technique with impressive improvements after concurrent plication of the anterior rectus sheath.^{6,11}

High lateral tension (HLT) abdominoplasty

In the 1990s Ted Lockwood presented this modified abdominoplasty procedure, which allows a full treatment of the abdomen and limited treatment of the hips and the lateral thigh in terms of a modified incision pattern.¹⁶ Lockwood enumerated several surgical principles that were in many ways diametrically opposed to the ones of a standard abdominoplasty, namely, an extensive discontinuous undermining and a more conservative resection centrally with wider excision of the lateral skin. This procedure finds its rare indication in patients after moderate to massive weight loss, who refrain from circumferential procedures. Nowadays, since most of these patients are likely treated by a circumferential tissue resection, indication for the HLT abdominoplasty is rarely given.

Female patients usually suffer from an additional skin laxity in the lateral thigh region, whereas male patients complain about the typical fat distribution in terms of 'love handles'.

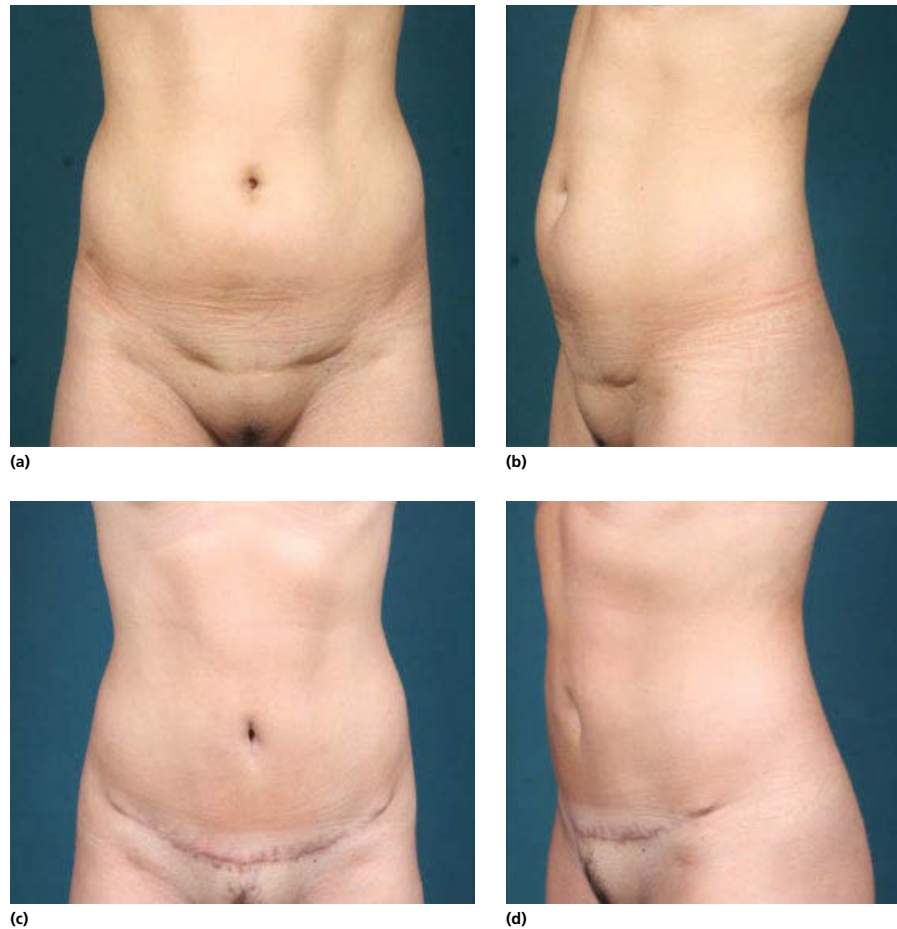


Figure 76.9 A 44-year-old patient after three pregnancies with two caesarian sections. Pre- (a, b) and postoperative (c, d) oblique and front views of an abdominoplasty with umbilical transection. Note the low placement of the previous scar and the high-riding umbilicus as well as the infra-umbilical diastasis recti.

The experienced surgeon may however be confronted with a HLT abdominoplasty procedure that results with lateral dog-ears, despite the extent of lateral tissue excision and scar elongation. Additional liposuction may be supportive, although often insufficient due to a surplus of lateral skin.

Markings

Preoperative markings are similar to those for standard abdominoplasty markings. The inferior incision line is continued laterally to the anterior axillary line and runs upwards to the mid-axillary line at an angle of 45°. The upper incision line starts at the highest point of the umbilicus, continuing parallel to the lower incision line until the anterior axillary line, then turning downwards at an angle of 45° to meet the lower line in the mid-axillary line to form an angle of 90°.

Technique

For this procedure it is helpful to position the patient on an additional cushion in the gluteal area for elevation of the flanks and lateral thighs and optimal access to these regions. Alternatively,

the patient may be changed into a lateral position. The operative technique is similar to that for standard abdominoplasty, although central abdominal flap mobilization is more limited and as far laterally as necessary for a required rectus plication. Additional flap advancement can be achieved through vertical discontinuous undermining, utilizing the Lockwood dissector. The lateral skin resection pattern with significant lateral resection is performed with a subsequent high-tension wound closure. For the lateral wound edges a reduction of subdermal fat and, if necessary, a lengthening of the incision may be performed in order to avoid dog-ears. A lateral elongation of the scar has to be acceptable for a satisfactory result; this is generally well accepted by the patients. Since the lateral abdominal perforators are preserved, extensive liposuction of the entire abdominal wall may be securely performed.^{6,16}

Fleur-de-lis abdominoplasty

Fleur-de-lis abdominoplasty is the most powerful procedure for maximal tightening of the entire abdomen and reshaping the lower body. It was popularized in 1985, and derives from

techniques previously described by Regnault (classic W technique) or Kelly (1910) and Babcock (1916).^{23–26} The procedure enables the elimination of vertical and horizontal tissue excess on the lower and upper abdomen. Using this approach, a maximum improvement of body shape and reduction of waist circumference is achievable. In cases of large abdominal pannus or patients with existing medical handicap, it can be performed as a two-stage procedure. In general, we advise refraining from lateral undermining for maximal preservation of perforators. Furthermore, we recommend a check of the markings intraoperatively prior to resection to avoid postoperative complications.

The suitable indication for this technique is an adequate, mainly horizontal tissue surplus of the lower and, in particular, the upper abdomen, usually in patients after moderate to massive weight loss. Any patient who presents with a pre-existing midline scar may be considered primarily for this technique. To date, we have not experienced a single patient complaining about the midline scar, although we have observed a more pronounced scar formation in the upper median abdomen. However, in cases where horizontal tissue resection was avoided for less scar formation, we have had patients complaining about postoperative existing tissue surplus in the upper abdomen. In these cases, a secondary horizontal tissue resection may be indicated.

In patients with a vertical tissue surplus in the upper abdomen and a request for a breast procedure requiring a submammary incision, an additional reverse upper abdominoplasty may be considered.

Markings

Preoperative marking should be performed while the patient is standing upright. In supine position the lateral tissue moves backwards and makes an accurate estimation of tissue excess difficult.

After marking the lower horizontal standard abdominoplasty incision line at a height 6 cm above the vulvar commissure, the midline should be marked precisely, with the highest point approximately 2–3 cm below the submammary fold. Next the tissue surplus is estimated through precise pinching of the skin and marked in an elliptical pattern. With the patient in supine position the distance from each line to the midline should be measured and equalized if necessary, since any small mismatch may result in an uneven midline scar. Next, the upper horizontal incision line is marked. This line should generally run 3–5 cm inferior to the umbilicus. Finalized markings usually result in an omega-like figure (Figure 76.10). We recommend limiting the superior extent of the scar line primarily by local superficial liposuction rather than a more superior excision. Finally, all vertical and horizontal markings should be checked and measured in upright and supine positions.

Technique

Single-shot antibiotic prophylaxis should be mandatory in this procedure due to the large wound areas and a prolonged operating time. Abdominal flap mobilization is performed as in

standard abdominoplasty procedures, except that the lateral flap mobilization should strictly be limited to the height of the upper incision line to preserve maximal perfusion of the lower medial apex of the flap. As soon the mobilization of the umbilical stalk has been completed, the entire estimated vertical and transverse resection area is temporarily closed using bullet forceps and the required marking adjustments performed. Next, the incision lines are incised and the redundant tissue excised in a monobloc fashion. After haemostasis the flaps are temporarily adapted and drains inserted.

The umbilicus has to be integrated into the vertical scar, and this should be performed in straight and not bended position. At the appropriate umbilical height a limited transverse incision is made on both abdominal flaps with reserved circular fat excision in the peri-umbilical region. The umbilical stalk is then adapted to the abdominal flaps, allowing skin excisions for optimal incorporation. A secondary aesthetically displeasing trumpet-like widening of the umbilicus may result from extensive peri-umbilical skin removal. Therefore we recommend a step-by-step excision with observation of any transverse tensile forces. In general, we insert three or four drains (including the upper abdomen) during subcuticular and intradermal wound closure.

If flap mobilization in the lateral aspect is limited to the resection line and therefore a maximal preservation of perforators established, flap perfusion and dermal wound healing should be uncomplicated. Despite the long scar formation, the aesthetic improvements to the hip and waist are dramatic and not comparable to any other abdominoplasty technique. Furthermore, the back and flank folds may be improved due to maximal recruitment of lateral skin (Figure 76.10).

Postoperative care

The different operations can be performed as outpatient or inpatient surgery. If the hospital offers an intensive or intermediate care unit, patients may be monitored after circumferential procedures for the initial 24 h postoperatively. For reduction of superficial wound tension, we intraoperatively apply transverse vectored Steri-Strips™ for the first three weeks. During the initial 48 h postoperatively, laboratory checks for electrolytes and haemoglobin are performed several times. After abdominoplasty and circumferential procedures, patients are positioned in beach-chair position, if available on an air mattress, with electronically adjustable positions. Thrombosis prophylaxis is administered using low-molecular-weight heparins and compression stockings. Initial wound care includes an antiseptic fluid-collecting dressing, in abdominoplasty and body lift cases two compression girdles, in breast cases a zip sports bra and in arm and thigh cases a circumferential winding. The immediate postoperative compression therapy reduces shearing forces supporting the adhesion of different tissue layers. In this context, drains may remain for a minimum 4 days postoperatively for a



Figure 76.10 (a, c) A 49-year-old woman with a weight loss of 50 kg after Roux-Y bypass. (b, d) Three months postoperatively after fleur-de-lis abdominoplasty and breast reshaping. (e) Markings. She was recommended for a lower body lift, but denied the circumferential procedure due to her financial situation.

negative intracavitary pressure. They are removed when drainage is less than 30 mL in 24 h. Patients are instructed to move their feet frequently and not to cross their legs.

All patients receive pain treatment within the initial 48 h postoperatively, utilizing an individualized patient-controlled analgesia (PCA) pump. Patients are mobilized on the first postoperative day and instructed to do deep breathing exercises to prevent pneumonia. The urinary catheter, which is indicated in any prolonged procedure such as circumferential or combined procedures, is removed after 2–3 days to help with patient mobilization.

Showering is generally permitted after drain removal. Postoperative clinical examination should investigate the possibility of seromas. In cases of hernia or diastasis rectus repair, patients should be asked about bowel function; abdominal auscultation, percussion and ultrasound might be advisable. Any non-resorbable sutures or wound closure material is removed at the latest three weeks postoperatively. For at least three months postoperatively we recommend our patients to cover the entire scars with silicon-sheets for improved scar formation.

Sporting activities should not be resumed for eight weeks postoperatively. Patients should be advised to avoid saunas and

tanning beds. Before patients are discharged, we individually adapt a compression garment for eight weeks postoperatively.

Complications

General symptoms after skin-tightening procedures include postoperative pain or soreness, numbness of the abdominal flap, bruising, general fatigue and discomfort due to increased skin tension for many weeks.

Local complications in all regions include haematoma, seroma, wound infection, fat necrosis, wound dehiscence, paraesthesiae and persisting numbness. Seromas are a very common problem in the abdominal region and usually handled with serial punctures and drainage. Persistent seromas may require a secondary surgical procedure. In general, seromas should be detected in time and treated by simple aspiration to avoid a superinfection or any consequent wound separation.

Minor wound dehiscences are common and self-limiting. Significant dehiscences may be caused by increased tension or marginal wound necrosis. Appropriate treatment of wound necrosis is initial conservative wound care, until the area of necrosis has been demarcated for surgical revision. Additional flap advancement may allow optimal secondary wound closure. Further local complications include dog-ears, hypertrophic or malpositioned scars, and in the abdominal region cosmetic problems related to the umbilicus. Most of these issues can be avoided with good preoperative planning and attention to surgical detail.

Treatment by liposuction may lead to postoperative contour irregularities and dermal tethering. Further swelling may occur in cases of circular liposuction of the extremities.

Systemic complications include deep vein thrombosis, pulmonary embolism, fat embolism, respiratory compromise due to increased intra-abdominal pressure in abdominal cases with fascial tightening and systemic infections including toxic shock syndrome. All of these complications may be potentially lethal. In general, abdominoplasty procedures have a higher systemic complication rate than any other type of routine cosmetic surgical procedures.^{6,9–11,19}

Body contouring after massive weight loss

Reconstructive procedures after massive weight loss resulting from bariatric surgeries or diet therapy include abdominoplasty, lower body lift, upper body lift, breast lift, upper lateral thoracic lift, bra-lift, thigh lift, lower leg lift, upper arm lift, lower arm lift and facelift/neck lift.

Preoperative management

A patient's first consultation should include a detailed description of all procedures planned for reconstruction. It is essential, that the patient be informed about the number of surgical stages

that will be necessary for the desired changes, including any secondary skin-tightening procedures. We recommend clarification of all important issues using a PowerPoint presentation with all available images, including photo documented complications. The patient should be informed about all possible complications. It is clear that the patient will base their decision to be operated on by the designated surgeon on this first consultation.

Prior to the surgery the patient should consult the surgeon for a second time to discuss all open issues. At this time, the patient has to sign the informed consent. We recommend schematizing all discussed scar courses and important tissue displacements. The patient has to be informed about the optional placement of drains, urinary catheter, and type and placement of suture material. Furthermore, any additional proposed manoeuvres (e.g. fascial reconstruction or Scarpa lift) should be clarified.²⁷

Since patients having bariatric procedures may suffer from nutritional deficiency due to a disturbance of nutrient uptake, we ask all patient to have a current blood test during their first consultation as well as prior to the surgery to avoid any late failures regarding the operative scheduling. Any coagulant dysfunction has to be evaluated and excluded preoperatively, where necessary by way of consultation with a haematologist. In this regard, we advise all patients about the supplement of all necessary nutrients (including zinc, selenium, vitamin C) four weeks prior to and four weeks after the surgery.

The sequence of surgical procedures may be discussed with the patient and optionally adapted between the single steps. We favour starting the reconstructive process with the lower body lift, followed by any further indicated procedures. The time frame between each stage should be at least 2–3 months for sufficient recovery.^{9,10,19}

As an important adjuvant therapy in body contouring we offer every patient with obvious connective tissue weakness (striae distensae) the repeated implementation of a surgical needling therapy, initially during the lower body lift procedure. Surgical needling is performed in all requested regions and should be performed in all existing scar areas, leading to a superior scar and skin quality.

Circumferential lower truncal dermatolipectomy

A lower body lift (circumferential lower truncal dermatolipectomy, CLTD, our modification of a lower body lift) is indicated in almost every patient after weight loss. The patient should be informed about the pro and cons of abdominoplasty versus circular procedures.^{9,10}

Markings

Markings are performed in upright standing position. First, the patient is asked to pull up their entire lower abdominal tissue in a symmetric manner. Meanwhile, after the midline has been drawn, a distance of 6 cm to the upper vulvar commissure is marked. At this height an almost horizontal lower incision line is marked to both flanks, followed by the upper horizontal incision line at the height of the umbilicus. Next, the patient has to

turn and the posterior markings are performed. The upper incision line is marked, starting at the highest point of the gluteal cleft and running upwards in a round curved shape to the lateral end of the anterior upper incision line. Finally, the lower incision line is assessed and marked by pinching and connected to the lower anterior incision line.

Technique

Dorsal preparation

The initial skin incision is made along the superior marking line and is carried out down to the level of the underlying superficial fascia, which is exposed using the Colorado Microdissection needle. This easily separates the superficial lamellar from the subfascial lobular fat. Next, the dissection is continued inferiorly just above the superficial fascia. The preservation of fascia to the deep gluteal fat as the 'gluteal superficial muscular aponeurotic system (SMAS)' is conceptually similar to the 'facial SMAS' used in facelifts. The superficial fascia is dissected at the height of the inferior resection line, before flap mobilization is continued further caudally. Lateral gluteal adhesions are released bluntly down to the height of the gluteal fold, before dissection is continued to the lateral thigh, where preparation level is above the tensor fascia lata muscle. Further distal mobilization of the lateral thigh can be carried out bluntly using the Lockwood underminer. With completion of dissection, the waist and gluteal fatty tissue is widely mobilized and covered with robust superficial fascia. This is particularly suited for autologous gluteal augmentations, since patients after massive weight loss frequently have flattened buttocks. For this purpose, 2–3 1 × 0 threads are sutured from lateral to medial, grasping the stable fascia and consequently displacing the gluteal fatty tissue to the middle of the buttocks. For cranial repositioning 3–4 1 × 0 non-absorbable threads are sutured from caudal to cranial at the medial aspect of the buttocks. Reconstruction and tightening of the gluteal adipose tissues lift the gluteal flaps cranially with a subsequent final skin closure under less tension. Furthermore, this manoeuvre improves the shape of the waist.

Detachment in the zones of adherence combined with extended mobilization allows an enormous tightening of skin. A symmetrical resection is then undertaken. We advise use of bullet forceps for determination of the amount of resection. Next, the flap is incised laterally and adjusted for maximum tension. The redundant gluteal flap arising in this way with its lower incision line is measured precisely in the tensed condition for symmetry before the resection of the flap is performed.

In general, the superficial fascial system (SFS) is extremely stable and allows a sufficient suturing for reconstructive purposes. After reshaping the SFS, wound closure is performed in multiple layers with resorbable suture material (see above). Two suction drains are placed in each gluteal/back and lateral thigh region. Before patients are turned into supine position, we perform a temporary closure of the lateral skin excess using a stapler and an occlusive dressing.

Anterior preparation

The inferior incision is checked and re-marked, adjusting the initial inferior line to the laterally ending gluteal incision line.

With all techniques, the dissection level is performed above Scarpa's fascia (see above), which has a number of key advantages: long-lasting swellings can be prevented because the underlying lymphatic vessels are preserved, and the tightening of the SFS craniomedially provides an additional 'inner traction' on the deep penetrating fascial system of the thigh (Colles' fascia). The SFS may then be fixed with 1-0 non-absorbable sutures to the anterior rectus fascia. Approximately three finger-breadths below the umbilicus, Scarpa's fascia is dissected and further mobilization of the abdominal flap is performed cranially above the rectus fascia. The central supra-umbilical adhesion zone is sharply dissected between both rectus muscles, preserving the laterally incoming perforator vessels.

Further preparation is performed as in the abdominoplasty procedure described above. After meticulous haemostasis, the patient is bent and several progressive tension sutures are placed superior to the umbilicus, the umbilical stalk is pulled through the abdominal flap and further progressive tension sutures are placed for reduction of deadspace and superficial tension. Next, temporary wound closure is performed with bullet forceps. Superficial fascial reconstruction, wound and skin closure may then be performed alternatively with barbed sutures or the two-component skin closure system as described previously (Figures 76.11, 76.12, 76.13 and 76.14).^{9,10}

Therapy of 'love handles' in male patients

Male patients frequently complain of local fat distributions in the hip and dorsal region, commonly known as 'love handles'. In the past, these were treated by liposuction. However, patients often complained about a local recurrence with a minimal weight increase. During circumferential preparation, these isolated fat pads can be easily identified and excised (see Figures 76.12 and 76.13). A meticulous haemostasis is advisable, since strong vessels are generally detectable for their isolated blood supply.

Thigh lift

Thigh lifts are an essential part of body-contouring procedures and may be performed through different approaches. Preferentially, the scar is placed at the inner thigh region in a vertical direction, in a horizontal direction in the groin region or in a combination of the vertical and horizontal scars (T- or J-shaped). In very rare cases additional scar placements are required, such as a horizontal scar just superior to the knee. The staging of the procedure should be discussed with the patient. In general, abdominoplasty or lower circular procedures should be staged prior to a thigh lift, since they enable an improvement in vertical direction to the thigh region.

Prior to thigh lift surgery, all patients should have an examination of the deep vein system and should have had any necessary therapy of varicose veins.



(a)



(b)



(c)



(d)

Figure 76.11 (a, c) A 28-year-old woman with a weight loss of 64 kg after BPD-bypass. (b, d) Twelve months postoperatively, after circumferential lower trunctal dermatolipectomy (CLTD), breast reshaping with implants and inner thigh lift.



(a)



(b)



(c)

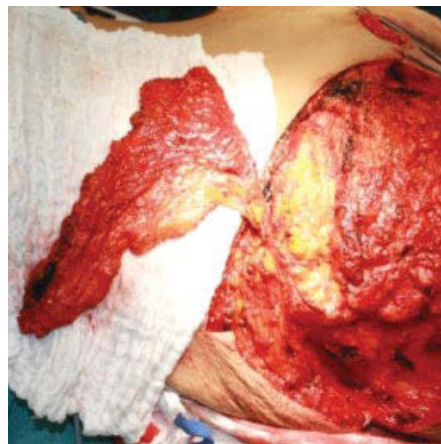


(d)

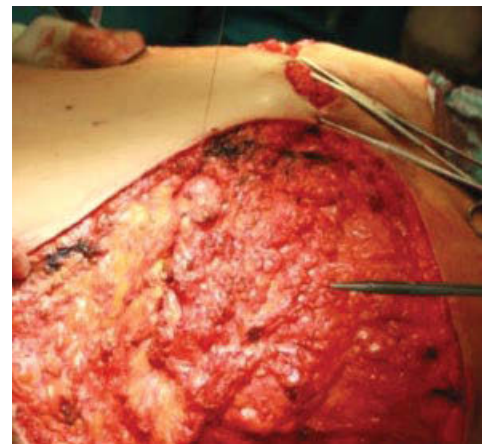
Figure 76.12 (a, c) A 54-year-old man with a weight loss of 100 kg after gastric bypass. (b, d) Three months postoperatively, after a circumferential lower trunctal dermatolipectomy (CLTD), anterior fleur-de-lis excision and open excision of the fat distribution at the lateral flank region ('love handles').



(a)



(b)



(c)

Figure 76.13 Intraoperative images from the patient in Figure 76.12. Demonstration of the open excision of 'love handles' with (a) the markings during dorsal preparation, (b) after completion of the *in toto* excision with the restored main vessels and (c) final closure of the resulting lesion.



Figure 76.14 (a, c) A 42-year-old woman with a weight loss of 67 kg after gastric bypass. (b, d) Twelve months postoperatively, after circumferential lower truncal dermatolipectomy (CLTD) with anterior fleur-de-lis excision, breast reshaping with autoaugmentation, upper lateral thoracic lift and inner thigh and arm lift.

Preoperative assessment

The preoperative evaluation includes the skin quality (e.g. thickness, laxity, presence of striae) as well as the thickness of the subcutaneous tissue in all thigh regions. The circumference has to be measured at the height of the greater trochanter, the middle of the thigh and just above the knee. The thigh should be photo documented in different views (front, side, back, inner side and in an abducted position). With the patient standing and the thigh in a 90° abducted position the overhanging redundant skin should be measured. All existing eczemas and hyperpigmentation should be assessed and photo documented.

Since the range of varying adipose distributions is wide, every patient has to be evaluated and plans for an individualized therapy regime prepared. Male patients generally present with skin and adipose excess in the upper inner thigh regions without any redundant tissue in the other thigh regions. Moreover, they very seldom complain about a local adipose tissue in either the outer and front thigh. Female patients, on the other hand, are characterized by minimum subcutaneous tissue in most parts of the thigh (except for the inner thigh). Other female patients complain about a massive adipose tissue surplus in the thigh region, which is totally resistant to any weight loss. In some female patients the weight may have settled in an acceptable range with maximal reduced adipose tissue in the entire upper and lower body but not in the entire thigh region, where they present with subcutaneous tissue several centimetres thick (DD: lipedema).

Markings

The marking is performed in upright position first. The desired scar course is marked bilaterally and demonstrated to the patient. The symmetry can be easily checked while the patient is standing upright with legs together. The groin crease should be marked precisely to avoid any dislocation or vertical lengthening of the scar, which can be easily observed in vertical procedures. This groin crease line demonstrates the highest point of incision in either vertical, all horizontal or combined procedures.

Depending on the tissue characteristic and any asymmetries, the resection lines are marked next. This can be initially performed in upright and checked for modifications in supine position. It has proven to be helpful to mark these lines in relation to the proposed scar line.

A helpful tool is the double-ellipse marking technique of Aly, which allows safer and more precise marking.

Technique

Regardless of scar placement, we have standardized our technique of thigh lifts as a combination of liposuction and tissue resection. Depending on the local adipose distributions, the liposuction is generally performed first. If patients present with adipose excess in the anterior, lateral and/or posterior thigh, these regions are thinned out through standard vertical liposuction (4 mm cannula), avoiding any horizontal cannula movements. After completion, the marked area of resection in the inner thigh is extensively treated by liposuction (5 mm cannula), removing any detectable fat tissue. Next, the marked resection area is checked using bullet forceps for a temporary approximation. After confirmation of the resection lines the skin is incised and excised by a scalpel or electrocautery-assisted avulsion from proximal to distal. It has been shown to be important to tear off the skin without any mechanical transection, as this allows the sectioning of all vessels, including lymphatic vessels, at the weakest point, reducing the risk of lymphocele, for example. In addition, this technique avoids the development of heat-induced seromas.



Figure 76.15 A 49-year-old woman after 55 kg of weight loss following diet and sportive action. Preoperative (a, c, e) and 12 months postoperative (b, d, f) anterior, medial and posterior view after a lower body lift, inner thigh lift and additional distal thigh and calf lift. Although 55 kg is a moderate weight loss, the skin and tissue excess was significant in this patient, who desired additional tissue excisions in the lower leg region.

After meticulous haemostasis the wound is temporarily closed with bullet forceps, subsequently replaced by staples. It is important to perform the temporary closure as soon as possible to avoid wound closure problems due to an increasing tissue swelling. At this time point any required changes may be performed regarding surplus skin and fat tissue in the groin region or obvious adipose surplus in adjoining regions, which

may easily be treated through liposuction. No undermining is performed to minimize impairment of the wound edge perfusion.

In cases of a sole or additional horizontal incision placed in the groin crease we recommend fixing the distal skin flap to Colles' fascia of the pubic region to avoid any distal displacement of the final scar.

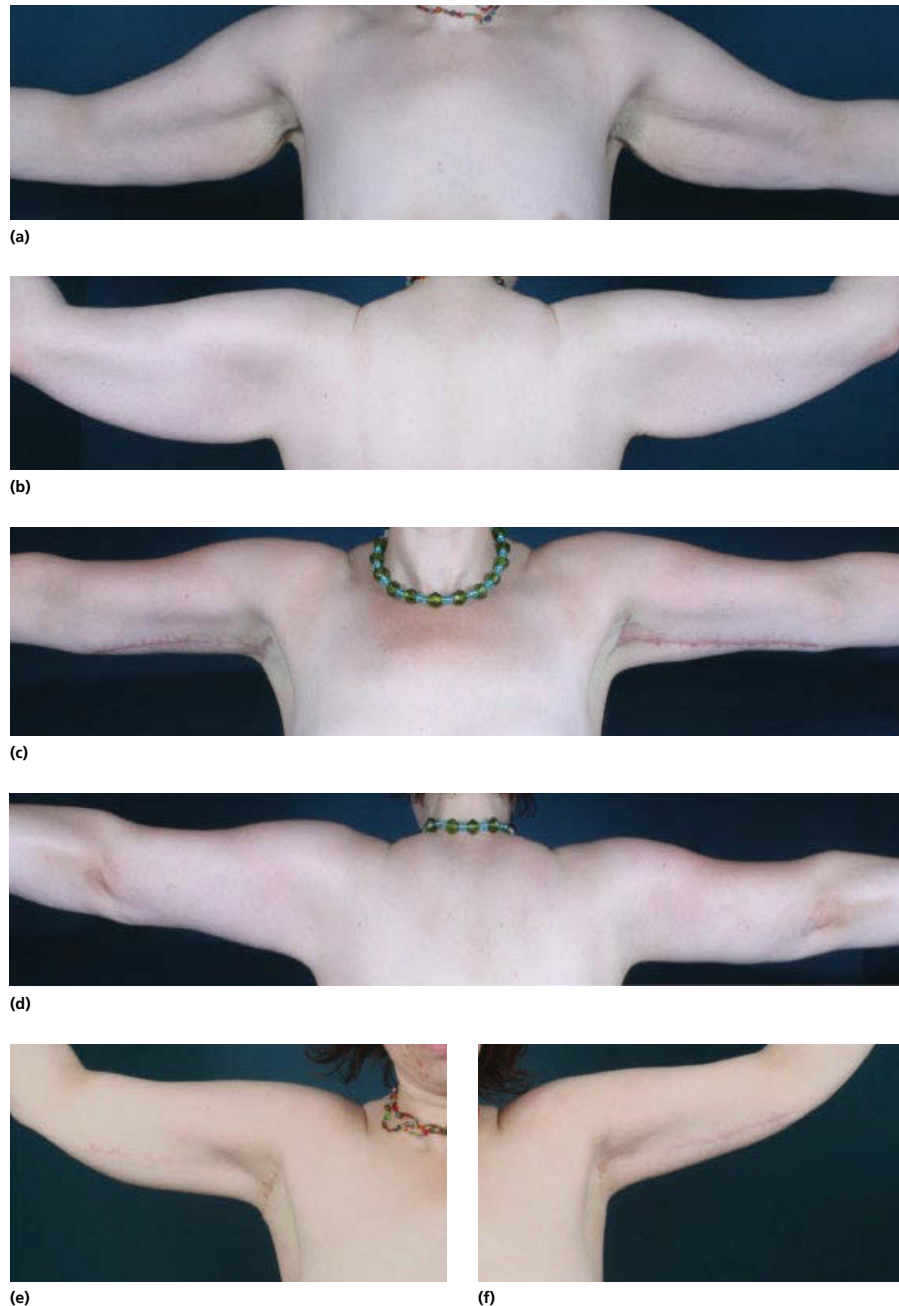


Figure 76.16 A 41-year-old woman after 60 kg of weight loss following gastric sleeve. Preoperative (a, b), three months (c, d) and eight months (e, f) postoperative front and posterior views after an arm lift with axillary T-scar. Note the scar development within the first eight postoperative months.

A drain may be placed before final wound closure commences. Wound closure is performed in the subcuticular and intracuticular layer, using absorbable sutures in single knot or nowadays barbed sutures in running manner as described above. Final application of Steri-Strips (transversely to the wound) as well as a sterile wound dressing is followed by a circular compression dressing which includes the feet and hips. During the first postoperative days, this compression winding is then changed to a custom-tailored compression garment.

During this period we initialize lymph drainage therapy and request a continuation for 4–6 weeks (Figure 76.15).

Arm lift (brachioplasty)

Arm lifts (brachioplasty) are commonly indicated in patients after weight loss to remove skin and fat excess and achieve pleasing and youthful upper and lower arm contours. Furthermore, the ageing patient may suffer from loose and flabby skin in the inner upper arm region. Plastic surgeons will consult with

patients about undergoing surgery for an arm lift if they wish to tighten this skin to look and feel more youthful. The incision extends from the elbow to the axillary crease, and sometimes extending on to the side of the chest. In some instances, liposuction may be used alone or in conjunction with an arm lift to remove excess fat in the upper arms.

In patients after massive weight loss, the redundant upper arm skin frequently crosses the axillary crease and extends onto the lateral thoracic region. This has to be demonstrated and marked with the patient. In a few individual cases, the skin excess extends to the lower arms. This has to be clarified to the patient, since this redundancy will be more conspicuous after an isolated upper arm lift.

Preoperative assessment

Skin laxity (presence of striae) and skin excess has to be evaluated in the entire upper and lower arm with the adjoining lateral chest region. The circular and vertical skin excess can easily be measured on the extended arm. Furthermore, especially in weight loss patients, local adipose tissue may be assessed in the entire arm.

Markings

The positioning of the incision lines depends on surgeon preference and should be demonstrated to the patient. In general, patients have to be informed preoperatively that in all cases the scar may be visible on the hanging arm from a dorsal view. There are several techniques, from which the ideal one can be chosen for the patient.

We perform markings while the patient is standing and elevating his or her straight arm in a 90° position. First it has to be determined whether there is a difference in surplus tissue between the sides. The first line marked should be the anterior line, just at the top of the medial bicipital groove. From this line the posterior line is evaluated through pinching. Alternatively, the double-ellipse technique described by Aly can be used. This technique may ensure a more accurate marking, especially for the novice surgeon. Finally, while the patient stretches both arms straight in front, a check of symmetry may be performed. In cases of tissue excess in the lower arm the markings should extend over the elbow to the lower arm.

Technique

Our technique of arm lifts is performed similar to the thigh lift procedure, as described previously, as a combination of liposuction and tissue resection. In general, patients present with adipose tissue excess in the inner arm region. The preoperatively marked area of resection at the inner arm is extensively thinned out by liposuction, removing any detectable fat tissue. This can ideally be performed with a 5 mm cannula. After completion, the marked resection area is checked using bullet forceps for a temporary approximation before the confirmed resection lines are incised and excised by a scalpel or electrocautery-assisted avulsion from proximal to distal. Again, it has been



(a)



(b)

Figure 76.17 A 48-year-old woman after 80 kg of weight loss following gastric bypass. Preoperative (a) and 12 months postoperative (b) posterior view after a lower body lift, breast and upper lateral thoracic lift and an arm lift. Note the improvement to the back region without any additional scar and the improvement to the axillary contour.

shown to be important to tear off the skin without any mechanical transection, as this allows the sectioning of all vessels including lymphatic vessels at the weakest point. Once meticulous haemostasis is completed, the wound is closed temporarily in the same manner as thigh procedures. Again, as in thigh procedures, it is important to perform the temporary closure as soon as possible to avoid wound closure problems due to tissue oedema. At this point any required changes may be performed regarding excess skin and fatty tissue in the axillary, elbow region or obvious adipose excess in adjoining regions, which may easily be treated through liposuction.

In cases of a sole or additional horizontal incision placed in the axillary crease we recommend fixation of the crease with fastening sutures.

Drains are uncommon but may be placed before wound closure, which is performed in the same way as described for the thigh lift. Final application of Steri-Strips, a sterile wound dressing and a circular compression dressing including the hands is performed in all cases. A custom-made compression garment may be adjusted prior to surgery and dressed intraoperatively. During the initial 4–6 weeks postoperatively we initialize lymph drainage therapy (Figures 76.16 and 76.17).^{28–33}

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PART IX

Military, simulation training and exams

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CHAPTER 77

Simulation learning in plastic surgery

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The era of simulation in surgical training

The traditional Halstedian model was the foundation of modern surgical training. A block of years with graded responsibilities culminated in a professional and/or academic assessment to acquire the title of surgeon specialist.^{1–3} Today, in a global healthcare system with economic, efficiency and safety challenges, traditional surgical training is questioned. It is no longer acceptable to expose patients to a trainee's learning curve, operative lists must be efficient, there is increasing litigation and a reduction in trainees' working hours. Attaining competency as soon as possible has become the current focus. Simulation has emerged as a tool to address these needs.^{4,5}

Simulation can be defined as a 'technique to replace or amplify real-patient experiences with guided experiences, artificially contrived, which evokes or replicates substantial aspects of the real world in a fully interactive manner'.⁶ The use of simulation concepts can be traced back to the writings of the twelfth-century physician Ibn Zuhr (also known as Avenzoar, AD 1093–1162). In his book *Simplification Concerning Therapeutics and Diet*, Ibn Zuhr documented performing a tracheotomy on a goat to demonstrate the safety of this operation in humans.^{7,8} During the eighteenth century in Naples, Raimondo di Sangro, Prince of Sansevero, built two anatomical machines that illustrate the cardiovascular system.⁹

Modern surgical simulation has evolved with distinct milestones; from the use of cadavers and animals in the nineteenth century to the development of manikin simulators in the late 1960s and early 1970s and most recently to laparoscopic, endoscopic and virtual reality simulators.^{10,11} The role of simulation in surgical training is now beyond 'proof of concept'. The advantages of simulation in modern healthcare systems have been well described in numerous reports establishing the validity and transferability of skills learned in simulation environments to the clinical setting with demonstrable advantages to the system, learners and patients.^{12,13}

That said, systematic analysis of the evidence base has yet to show conclusively added benefit compared to traditional methods.¹⁴ Simulation training is widely perceived as an adjunctive tool, not as a replacement for patient-based operative training experience.^{6,15}

Simulation methodology enables tailored training interventions in a low-threat environment. Simulation can address limited exposure to patients with low-incidence and high-complexity conditions and provide competency. It can also be used to evaluate the outcome of training in a more objective and structured way. Harden and others used this concept with the development of the objective structured clinical examination (OSCE).¹⁶ A shift towards competency-based surgical training comes with two key concepts: objective assessments and simulation lab training.^{2,3,17}

Assessment of surgical skills in plastic surgery

Assessment has two main functions: educational and evaluative. Assessment may aid learning as a 'formative' assessment. Assessment may also be a tool for evaluation as a 'summative' assessment. Beard classified assessment methods according to the skills they assess, summarized best in Miller's triangle (Figure 77.1).^{18,19}

To demonstrate competency, multiple tools are used. Although surgical direct observation of procedural skills (DOPS) and problem-based assessments (PBAs) can be used to demonstrate trainees' competence of 'know how' and 'show how' in relation to a particular procedure, they cannot demonstrate performance, which could, however, be achieved by video-recording of a trainee performing a procedure and conclusively demonstrating that they 'can do' the procedure.

Surgical skill has been traditionally determined by mentors' subjective reports on trainees' ability. One of the first

structured assessment tools was developed by Kopta in 1971.²⁰ The tool assigned a 3-point Likert scale or 'observation guide' to lists of steps he considered important indicators of skill. This tool helped establish that methods such as the Medical College Admission Test (MCAT) did not correlate with measured surgical skill.²¹ In 1993, Reznick introduced the Structured Technical Skills Assessment Form (STSAF) with a checklist and a 5-point Likert global rating scale (Figure 77.2). Reznick's tool demonstrated construct validity and good interobserver reliability.²² This group optimized this method into the Objective Structured Assessment of Technical Skills tool (OSATS).²²

OSATS are performance-based assessments that evaluate trainees in standardized stations each with a specific task (e.g. performing a small bowel anastomosis). The candidate is then rated using both the station-specific checklist and a global rating score sheet. The validity of this tool was not affected whether the models used were live anaesthetized animals or lower bench fidelity station models.²³ Although OSATS has brought major structure and objectivity to assessment methodology it remains dependent on raters' judgement and cannot be considered wholly objective.

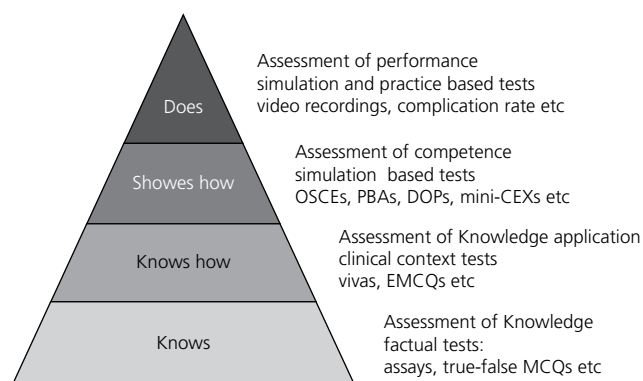


Figure 77.1 Miller's triangle relating clinical skill with assessment method.¹⁹ Source: Adapted from Miller, 1990.¹⁹

Skills assessment tools in plastic surgery training

Plastic surgery combines a variety of skills for reconstructive and aesthetic purposes. The level of skill required is reflected in the complexity of the procedure by 'the reconstructive ladder'. Free tissue transfer occupies the highest rung of the ladder, and largely defines the specialty.^{24–26} A key skill in free tissue transfer is vascular microanastomosis. Microsurgery requires very refined movements originating mostly from the intrinsic muscles of the hands in contrast of the wrist, elbow and shoulder movements that are required in, for example, laparoscopic surgery. It also requires a particular level of eye–hand coordination and dexterity and is associated with a steep learning curve.^{27,28} A competency-based curriculum in microsurgical training requires an objective assessment tool.²⁹ Objective assessment is important for feedback that improves skill, and for career planning. The validity of the tool must extend to incorporate all forms of validity: predictive, content, construct, face, concurrent, equitable and fair.³⁰

Methods of microsurgical skill assessment

Rater assessment tools

The study of technical skill in microsurgeons dates back to 1993 when Starkes *et al.* evaluated the accuracy of suture placement, time to complete a standard task, and vessel patency to evaluate skill acquisition in a training cohort. The time to complete a standardized task was the most sensitive measure of experience and skill.³¹ In 1998, they validated an assessment of repair on a slit in a surgical glove with two microsutures.³² In 2003, Grober *et al.* modified previously validated checklists and global rating scores and adapted them to microsurgery.³³

Several modifications of global rating scales have been introduced: by Kalu *et al.* (Figure 77.2),³⁴ Chan *et al.*,³⁵ and by Temple and Ross.³⁶ Global rating scales are based on OSATS and comprise: Likert scale of the components, and an errors list with or without a summative rating (Figure 77.3).

Global rating scale tools have construct and concurrent validity (Figure 77.4), which have been validated in the clinical

Please circle the number corresponding to the resident's performance in each category

Respect for Tissues	1 Frequently used unnecessary force on tissue or caused damage by inappropriate use of instruments	2	3 Careful handling of tissue but occasionally caused inadvertent damage	4	5 Consistently handled tissues appropriately, with minimal damage
Time and Motion	1 Many unnecessary or repetitive movements	2	3 Efficient time/motion but some unnecessary and repetitive moves	4	5 Clear economy of movement and maximum efficiency
Instrument Handling	1 Repeatedly makes tentative or awkward moves with instruments through inappropriate use	2	3 Competent use of instruments but occasionally appeared stiff or awkward	4	5 Fluid movements with instruments
Suture Handling	1 Frequently damaged, broke or lost sutures	2	3 Occasionally damaged, broke or lost sutures	4	5 Suture were consistently handled delicately under the control of the operator
Flow of Operation	1 Frequently stopped operating and seemed unsure of the next move	2	3 Demonstrated some forward planning with reasonable progression of procedure	4	5 Obviously planned course of operation with effortless flow from one move to the next
Quality of Final Product	1 Very poor	2	3 Competent	4	5 Clearly superior

Figure 77.2 The 5-point Likert scale assessment tool introduced by Reznick's Objective Structured Assessment of Technical Skills tool has been modified by Grober *et al.* and Kalu *et al.* for microsurgery skill evaluation.^{22,33,34} Source: Grober *et al.*, 2003.³³ Reproduced with permission of John Wiley & Sons.

Trainee:

Assessor:

Operation + date:

Anastomosis technique: triangulation / backwall / other:

Please tick level (scale 1-5)		1	2	3	4	5
Dexterity	Steadiness	Frequent exhibition of Tremor		Occasionally signs of Tremor		Fluid movements and obvious control of movements
	Instrument handling	Repeatedly tentative awkward moves through inappropriate use		Competent use, but occasionally stiff/awkward		Fluid movements with no stiffness or awkwardness
	Tissue handling	Frequently unnecessary force on tissue/damage caused		Careful, but occasionally inadvertent damage		Consistently appropriate handling with minimal damage
Visuo-spatial ability	Dissection	Frequently uncontrolled grasping of tissue or readjustment of view required		Correct use of instruments at right angle, but occasional uncontrolled grasping/view disturbance		Efficient use of instruments with vessel ends clearly prepared
	Suture placement	Frequently lost sutures & uneven placement		Occasionally uneven suture placement		Consistent delicately placed sutures with adequate spaces
	Knot technique	Insecure knots/frequent thread in knots		Secure knots, but occasionally awkward movement		Consistent secure knots placed effortlessly
Operative flow	Steps	Frequently stopped and unsure of next move		Reasonable progression of procedure		Obvious logically planned course of operation with effortless flow from one move to next
	Motion	Many unnecessary or repetitive moves		Efficient, but some unnecessary moves		Economy of movement and maximum efficiency
	Speed	Excessive time used for each step due to insufficient dexterity		Efficient time, but some unnecessary repetitive moves		Excellent speed and superior dexterity without awkward moves
Judgement	Irrigation	Poor use of irrigation, frequently desiccated vessels		Fair use of irrigation, but occasionally too wet, interfering with operative flow		Appropriate use of irrigation
	Patency test	Unsure of result of Acland test with possible thrombosed patency		Correct clamp removal, but repeatedly using Acland test		Appropriate sequence and time of patency test with confidence of unimpeded flow
	Bleeding control	No check for uncompleted anastomosis before clamp removal		Anastomosis checked but requires sutures for anastomotic leak		Places extra sutures as appropriate before clamp removed

Errors list: (please tick if observed)

Planning	Inappropriate operative field	
	Inappropriate vessel set up	
	Focus lost	
	Loss of central view	
Dexterity	Vessel damp re-application	
	Broken sutures/needle off suture	
	Wrong grasp/damage tissue	
	Opposite wall caught	
	Vessel tear	
	Thread in knot	
Visuospatial ability	Insufficient vessel preparation	
	Empty grasp	
	Unequal stitch bites	
	Suture pulled through	
	Suture cut through	
	Loose knot	
Operative flow	Re-do suture	
	Inappropriate magnification	
	Inadequate loop length	
Judgement	Vessel dessication	
	'pooling'	
	Anastomotic leak - extra sutures	
	'crushing' patency test	
	Excessive sutures	
	Impeded flow - re-do anastomosis	

Comments:

Overall performance:

1 2 3 4 5
 bad borderline satisfactory good excellent

Indicative skill for next performance:

1 2 3 4 5
 novice advanced beginner competent proficient expert

Figure 77.3 Structured Assessment of Microsurgery Skills (SAMS) global rating scale, error list and summative assessment.³⁵ Source: Chan *et al.*, 2010.³⁵ Reproduced with permission of Elsevier.

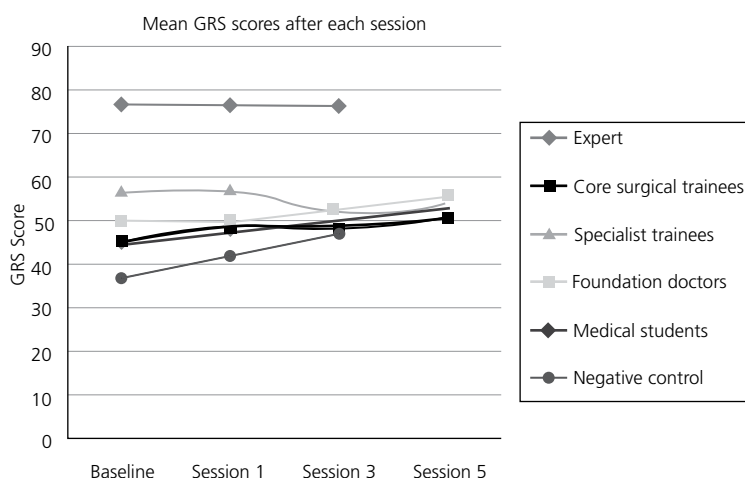


Figure 77.4 A global rating scale can demonstrate construct and concurrent validity when used to evaluate different cohorts and training interventions (5-day basic microsurgery course). Source: Grober *et al.*, 2003.³³ Reproduced with permission of John Wiley & Sons.

setting and found to be effective in live surgery.³⁷ However, such rater-dependent tools share several disadvantages. Checklists have lower construct validity than global rating scales.³⁸ This can be caused by novices approaching new tasks in a step-wise fashion whereas more experienced surgeons approach the task as a whole, and thus may be negatively scored as they miss some steps specified on the checklists.

Global rating scales address this issue to some degree by offering more flexibility. Despite the flexibility of a 5-point Likert scale in a global rating scale, the descriptions of the grades on the scale are not always clear or objective. For example, one of the criteria on the scale that refers to 'quality of product' in one of the tools is graded on a range of 'very poor' to 'clearly superior'. Both global rating scales and checklists may suffer from rater bias.^{34–36}

The global rating scale is the simplest, most readily available and validated tool in the literature. Nevertheless, there is no consensus on a standard scale nor confirmation of predictive or content validity. The assessments are time-consuming, and interest has grown in more objective methods.

Objective assessment tools

Objective assessment tools would always be favoured over biased rater-dependent tools. In microsurgery, where assessment models and hand movements are very small, significant advances had to be made in technology and equipment. There are currently two main objective assessment tools. The first is end-product assessment and the second is motion analysis. End-product assessments rely on testing the quality of the anastomosis formed at the end of a microsurgical procedure (the end-product), whereas motion analysis assesses the surgical technique used to form the anastomosis (the procedure).

End-product assessments

End-product assessments in microsurgery evaluate the patency of the microvascular anastomosis performed. When used in clinical or live animal setting, this tool can be considered the gold standard, since a patent anastomosis is the ultimate goal of the procedure. Ilie *et al.*³⁹ conducted a study using live

anaesthetized rats as microvascular models. Patency was assessed at 30 min and two weeks after the anastomosis. Measurement of vessel patency was possible and served as feedback to the trainee on how much further training they may require.

The major disadvantages of end-product assessment tools are ethical and economical. There are ethical issues in the use of live animal models. And this tool presents unacceptably high costs including those of licensing, sourcing live animals and housing them for a period of two weeks to assess long-term patency. Perfusion can be assessed in a non-living model by infusion of a fluid dye,⁴⁰ but leakage from the anastomosis can result from any difference in viscosity between the dye and blood, and the lack of clotting factors, rather than poor skill. Therefore, the limits of end-product assessments in non-living models and their ethical and costs limitation in live animal models reduce their utility.

Hand motion analysis

Hand motion analysis (HMA) uses a tool that tracks hand movements through 3D space, and has been developed and pioneered in laparoscopic surgery.⁴¹ Six published studies report its use in plastic surgery.³⁰ Two technologies have been used to assess hand motion for fine motor skills. The first uses electromagnetic (EM) motion sensors (Figure 77.5), and the second is video-based, with infrared cameras that detect hand motion by measuring the distance to reflectors on the operators' hands (Figure 77.6). Ultra-light EM motion sensors have been produced to trace small movements with little intrusion (Figure 77.7).

Electromagnetic hand motion analysis systems

EM motion tracking has been used in other fields, including virtual reality interfaces and gait analysis. Of the six papers referred to above, five have used an EM hand motion analysis device. They all found that HMA correlated well with global rating scores and skill level, although a global rating scale tool should be used with HMA, which provides quantitative data alone, and cannot reflect the quality of execution or its end-product.^{30,42,43} Dosis *et al.* introduced synchronized HMA with video-recording to provide both qualitative and quantitative assessments.⁴⁴

HMA is confirmed as an objective microsurgery assessment tool both in skills labs and in the clinical setting.⁴⁵ Comparison of HMA output (time to complete procedure, number of hand movements and hand travel distance) with global rating scales and checklists shows correlation with concurrent and construct validity (Figure 77.8). Grober *et al.* suggested that HMA may also have predictive validity and could be used to develop learning curves for skill acquisition and competence testing.

The cost of this technology, however, remains a major limitation to its dissemination. Availability is as much an obstacle as cost, and use is often limited to the research environment. For the quantitative data to be helpful, it needs to be defined in the context of skill acquisition learning curves.³⁰

Video-based hand motion analysis systems

This technology involves several 2D video cameras modified with infrared light-emitting diodes (LEDs). Video-based HMA has been used successfully in the assessment of microsurgical skills by Saleh *et al.*⁴⁶ No comparative studies have been conducted against EM systems. The obvious advantage of EM systems over optical-based ones is the lack of requirement for direct line-of-sight. Modern EM systems also have relatively small sensors (Figure 77.7), which may be discretely placed on the subject with minimal interfere with their movements. EM systems have a limited operating range, however, due to an inverse cubic reduction in magnetic field strength with distance, and can suffer interference from nearby metallic objects.

Figure 77.5 Imperial College Surgical Assessment Device (ICSAD) hand motion analysis. An electromagnetic sensor is placed on the dorsum of the hand.^{33,42,43} Source: Grober *et al.*, 2003.³³ Reproduced with permission of John Wiley & Sons.



Figure 77.6 Infrared light reflectors placed on dorsum of hand and fingers to be tracked with video-based motion tracking system as in Saleh *et al.*⁴⁶



Figure 77.7 New-generation ultra-light electromagnetic tracers are more suitable and less intrusive for fine finger microsurgery movements compared with older generation models.



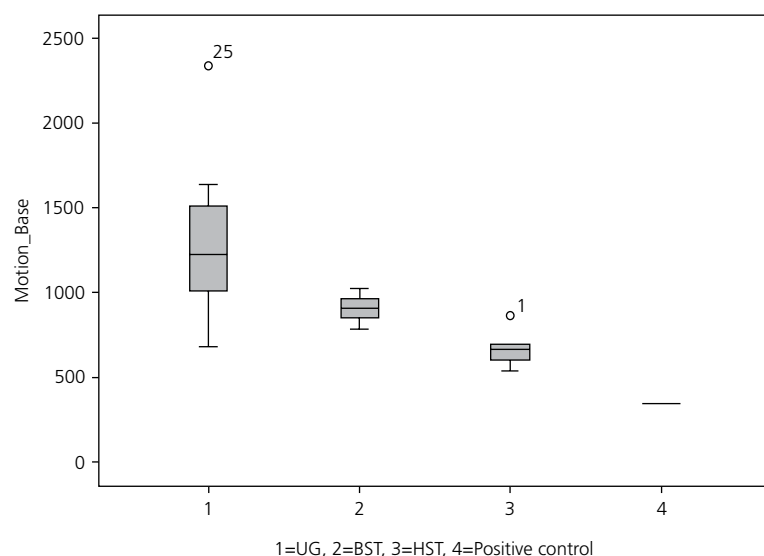


Figure 77.8 Hand motion analysis outcome measures (e.g. number of hand movements taken to complete an end-to-end arterial anastomosis) can demonstrate construct when used to evaluate microsurgery skills in different cohorts. UG, undergraduate medical students; BST, basic surgical trainees/junior residents; HST, higher surgical trainees/senior residents; Positive Control: expert microsurgeons. Average number of hand movements required to complete this task varied significantly between cohorts from approximately 500 to 1000 movements in expert microsurgeons and inexperienced undergraduate students, respectively.

Virtual reality

Virtual reality simulators possess theoretical advantages as assessment tools. The task can be standardized, repeatable and tailored in difficulty to challenge the trainee. They also have an ethical advantage in avoiding animal models.⁴⁷ Virtual reality simulators have demonstrated construct validity in laparoscopic surgery using outcome measures such as time to complete the task and economy of movement.^{48,49} Virtual reality is in its infancy in microsurgery, and the costs are significant.²⁹ The lack of haptic feedback may be of less relevance here. Studies using this tool suggest that virtual reality simulators should be combined with global rating scales or other objective assessment tools until more research results are available. An excellent review of virtual reality in microsurgery by Erel *et al.*⁵⁰ summarizes the current development of the technology in microsurgery and highlights the lack of evidence for validity of the tool.

Models and technologies for plastic surgery simulation training

Most of the developments in surgical simulation technologies have focused on laparoscopic, endovascular and endoscopic applications. However, plastic surgery has witnessed developments in non-living bench models, live animals models, cadavers, virtual reality and software-based computer simulators.

Bench models

Hands-on training using bench models is a cost-effective method to practise surgical skills. Many private companies specialize in the development of such models and Limbs and Things UK/USA, Pharmabotics Ltd. and SynDaver™ USA are among the leading manufacturers of surgical skills training models. Simple skin pads for suturing and skin excision models are common. A fundamental skill in plastic surgery is

the harvesting of a split skin graft. In one simulation model,⁵¹ a device is constructed from a wooden base with two side arms that secure and stretch a skin substitute over a synthetic limb segment formed from a 500 mL bag of fluid solution. The fluid volume is adjusted to simulate the required turgor and shape. The overlying simulated skin element is Microfoam™, which conforms to the saline bag. Laying chicken skin over a simulated body part has been used to teach three-dimensional suturing techniques and local flap surgery.⁵² Higher fidelity cadaveric animal parts can be used as simulators for whole procedures (e.g. tendon repair in the pig trotter).⁵² Formal validation with evidence of objective skill acquisition is rarely presented, however, and as such the role of these models in a comprehensive training curriculum remains ambiguous.

Ilie *et al.*⁵³ organized microsurgical models into five main groups:

- basic manipulation, movement, and orientation in the operative field;
- knot placement/tying principles – apposition of edges, non-dominant hand usage, deformable volumes;
- three-dimensional models;
- the real tissue experience; and
- virtual reality trainers.

This classification can be extended to many skills beyond an anastomosis. For the first, non-living bench models can safely and effectively be used due to their cost-effectiveness and favourable ethical profile. Many synthetic models have already been introduced, including surgical gauze, latex gloves (Figure 77.9), silicone tubes, beads, needles to name a few.^{54–56} These models can assist in rudimentary skill acquisition such as use of the microscope and instruments handling, but little has been reported of predictive validity beyond these basic skills.

More sophisticated simulators allow the experience of an entire procedure. A simple life-size model of the oral cavity of a patient has been developed to simulate the structure of a cleft palate for simulated palatal repair.⁵⁷

A further step in simulation is the integration of skills into a real-life surgical experience with convincing face validity. Both the anatomical and physiological components of the procedure must be simulated. Non-living animal models examples include the rat aorta or chicken thigh model in microvascular anastomosis (Figure 77.10a,b),^{58,59} and the use of fresh cadaveric sheep cranium to simulate craniostomy surgery.⁶⁰

The use of synthetic models has already contributed to reducing the number of live animal models needed to attain a particular skills threshold during training.⁶¹ The three Rs – refine, reduce and replace – increasingly guide the ethical use of animal models in research and training.⁶² The ‘practice rat’ is an example of a more sophisticated synthetic simulator.⁶³

Live animals

The use of live animal models in plastic surgery originated from experimental microsurgery research in dogs.^{64,65} The most widely used animal model in both research and training applications is the rat (Figure 77.11).^{66–68} This model is very versatile – up to 20 basic and advanced microsurgical procedures have been

described.^{66,69–73} Nevertheless, despite the live rat model’s excellent face validity it is limited by the different blood supply pattern, being a loose skin animal. Therefore, simulation of perforator flaps would not be possible in such models. With human perforator flap concept and composite tissue transfer advances in gonadal, limb and facial transplantation, research groups and simulation training have shifted from the rat to large mammals due to the higher fidelity required to simulate these advanced techniques (Figure 77.12a,b).^{74–77}

A very useful exercise in the rat is anastomosis of the hind limb vessels due to their similarity to the human digital vessels. For advanced microsurgery skills, abdominal epigastric flap, tail and limb replantation and kidney transplantation exercises in rodents provide highly effective and reproducible simulation models.^{66,67,78,79}

Despite their utility and popularity, live animal model use is increasingly regulated due to associated ethical issues and heavy costs. Confirmation of predictive validity is also required, by establishing the transferability of skills from animal models to patients and their outcome.

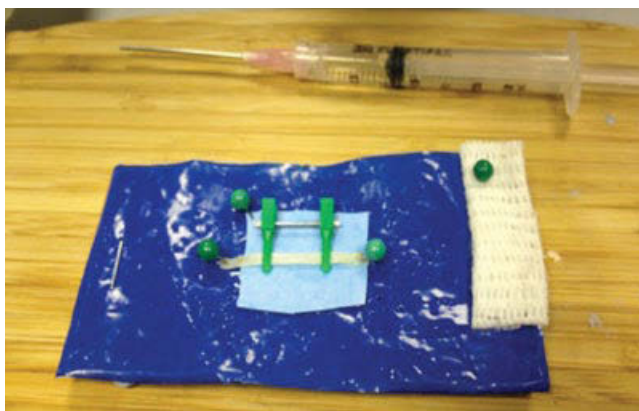
Human cadaveric models

Human cadavers have been used for plastic surgery training for a long time. In 2013, educators from the Plastic Surgery Department in the University of Southern California reported a significant increase in residents’ confidence by incorporating a fresh tissue dissection (FTD) simulation programme into their curriculum.⁸⁰ Confidence increase positively correlated with training year, and varied according to anatomical region and procedure. Confidence was lowest for the head and neck region, rhinoplasty and facelift, in contrast to radial forearm and latissimus dorsi flap surgery.

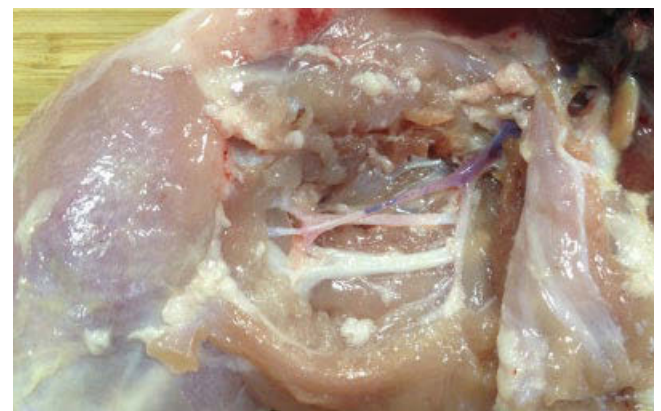
The same team reported an enhanced-fidelity perfused cadaveric model.⁸¹ Fresh tissue human cadavers were perfused via cannulation of large vessels and centrifugal circulation. This method allowed simulation of life-like surgery and the development of training models for basic and advanced surgical procedures



Figure 77.9 Latex sheets cards are a cost-effective bench model for teaching basic microsurgical suture techniques.



(a)



(b)

Figure 77.10 (a) Cryopreserved rat aorta and (b) chicken vessels can simulate real tissue experience in dissection and microvascular anastomosis early in trainees’ learning curves.

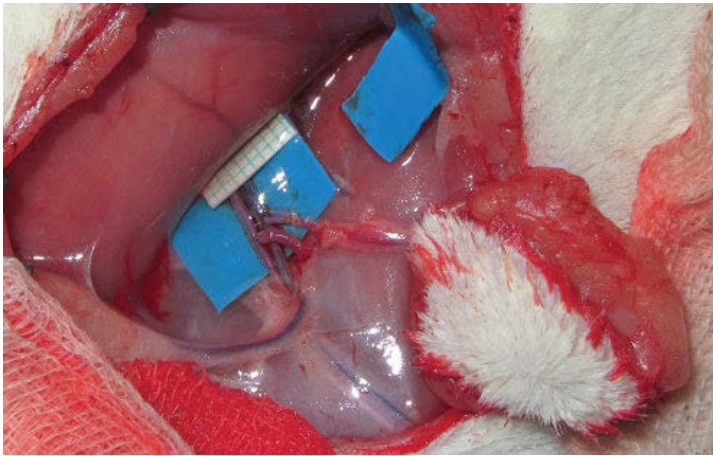
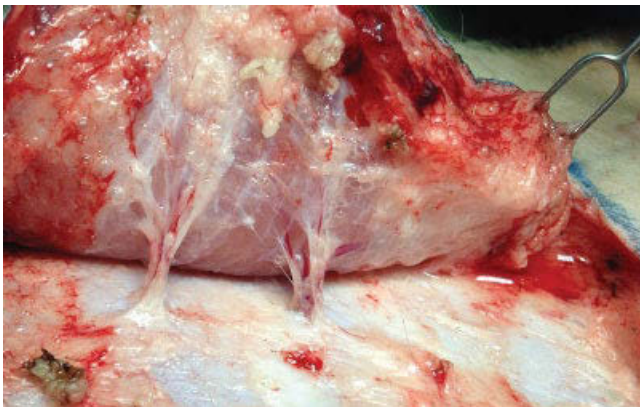
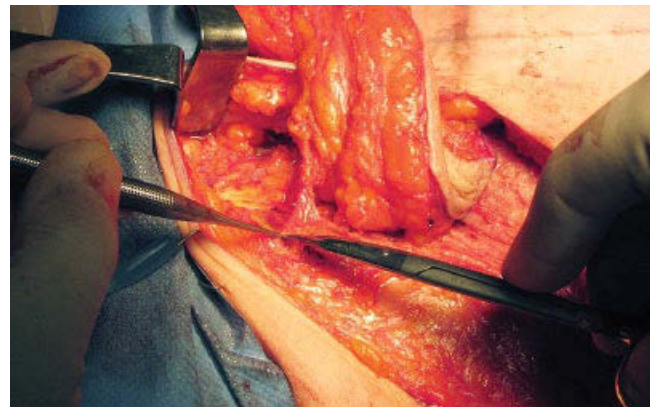


Figure 77.11 Rat femoral vessels and groin flap as high-fidelity models for microvascular anastomosis and free tissue transfer.



(a)



(b)

Figure 77.12 (a) Live anaesthetized pigs are useful simulation models to teach perforator flap raising techniques due to similarity with (b) human perforator vessels.

including local and distant flap surgery. Enhancement of the fidelity of fresh cadaver simulation models by artificial perfusion represents the highest form of whole procedure simulation.

Virtual reality, robotic and computerized mannequins models

Virtual reality simulation was born in the aviation industry. Rapid advances have been made in robotic surgery and simulation modelling for clinical case planning and outcome prediction with many applications in education and training (Figure 77.13).^{82–87} The technology of simulation models is evolving further towards the creation of an interactive computerized patient mannequins simulator that recreates a complete experience of surgery in a threat-free, patient-safe digital environment with reports confirming significant improvement in surgical knowledge (Figure 77.14).^{83,88}

With technological advances in robotic-assisted surgery, numerous applications have been described in plastic surgery.^{89–91}

In parallel, simulation-based training and assessment interventions have been developed to prepare trainees for this emerging and rapidly advancing field.⁹²

Simulation for non-technical skills

The make-up of a good surgeon is not based on technical skills alone. Surgical skills belong to three broad categories: cognitive/clinical skills, technical skills and social/interactive skills.⁹³ Non-technical skills for surgeons can be classified into four categories: decision-making, situational awareness, communication and teamwork, and leadership.⁹⁴

Both technical and non-technical skills are vital for safe surgical practice since communication failure contributes to almost half of errors in surgery.⁹⁵ Recent evidence suggested that non-technical skills can enhance or undermine technical performance.⁹⁶

Teamwork is based on non-technical skills and thus, evaluating and improving these skills is an important factor in improving teamwork, reducing medical errors and provide a

Figure 77.13 The da Vinci robotic simulator can be used as a virtual reality simulation tool for both robotic and microvascular surgery.



Figure 77.14 computerized mannequin patient simulators are useful to recreate training scenarios such as war surgery in a safe environment.



better quality of healthcare.⁹⁷ The UK Good Medical Practice, the USA Accreditation Council for Graduate Medical Education (ACGME) and the Royal College of Physicians and Surgeons of Canada CANMED frameworks all describe the skills of a good healthcare practitioner necessary to provide high-quality care. Multidisciplinary simulation of the clinical environment can facilitate individual non-technical skills acquisition and prepare clinical teams for infrequently encountered scenarios such as surgical emergencies.^{98,99}

Non-technical simulation skills are increasingly assessed in surgical training. Several methods have been developed to evaluate them, including the NOTSS (NON-Technical Skills for Surgeons), NOTECHS (NON-TECHNical Skills) and OTAS (Observational Teamwork Assessment in Surgery).^{98,100,101} Evidence-based simulation methodologies have been used effectively as training interventions to teach non-technical skills in surgery.^{102,103} This method has been used extensively in training of other high-risk professions such as the armed forces, aviation industry and the fire brigade with valuable lessons that can be learned and applied in surgical training.

Evidence for the benefit of plastic surgery simulation training

High-level evidence of the effectiveness of simulation in generic surgical training has been confirmed by a Cochrane review analysing over 30 randomized controlled trials.¹⁰⁴ A critical analysis of the surgical education research of the last decade investigating evidence for educational and training interventions relevant to plastic surgery can be inferred from this work as well as other reviews. The best evidence for a competency-based simulation training programme for plastic surgery supports a laboratory-based mixed-fidelity model programme – low fidelity (for basic skills) and high fidelity (for advanced skills) – that encourages and facilitates deliberate practice in a distributed fashion.^{105,106}

There is strong evidence that technical skills acquired on low-fidelity models transfer to improved performance on higher fidelity human cadaver models, and that self-directed deliberate practice leads to improved technical performance. Although there is significant paucity in the literature to support current plastic surgery education and training practices, the incorporation of regular simulated training on low-fidelity models in microsurgery

provides an effective intervention that leads to acquisition of transferable skills and improved technical performance. Further research to confirm the transferability of skill to the operation room and benefit of healthcare system and patients alike is still lacking.

Evidence for the role of non-technical skills simulation training in the improvement of healthcare systems has been evaluated by a comprehensive expert consensus. Using the CANMED non-technical competencies of the healthcare worker as an effective communicator, collaborator, scholar, professional, manager and health advocate to evaluate the literature, the review confirmed the benefit of simulation in promoting the competencies of medical expert, communication and collaboration. Further work is required to develop the exact role of simulation as a training strategy for competencies related to scholarly skills, professionalism, management and health advocacy.⁶

Priorities for plastic surgery simulation

Integration into training curricula

Once a competency simulation-based programme or an educational intervention has been designed and validated it is necessary to formally integrate it into a training programme. Several options exist but due to the novelty of simulation concepts coupled with logistical challenges, simulation-training methodologies are yet to be seen as a standard practice in plastic surgery. The most important aspect of integration of simulation training into a formal training programme is 'protection of simulation training time'. Our experience in encouraging attendance as part of optional study leave suggests that this does not work very well. Only 25% of trainees confirming their support of simulation training in microsurgery in London managed to attend self-directed and supervised training in microsurgery simulation facility over a 2 year period. Our experience, coupled with that of other educators, suggests that residents should be formally introduced to the simulation component of their curriculum. Four integration approaches have been described: the block-time approach, rotational-integration approach, skills-rotation approach, and the independent-study approach.¹⁰⁷

In the first approach, protected time blocks are provided weekly or monthly for the trainees throughout their rotations. In contrast, The rotation-integration approach provides simulation-training modules that are synchronized with a specific clinical rotation, for example, a basic bench model microsurgery skill course as part of a clinical rotation of reconstructive microsurgery. It is critical for these two approaches to be effective that time provided for simulation training is truly protected and that the simulation training is synchronized with clinical training provided so that there is a natural progression of skills learned from the lab to the clinical environment.

The third (skills-rotation) approach provides a simulation module within the training programme that all trainees must complete to progress in their training. This option guarantees

protected simulation training time that may be designed to provide all basic competencies required for a particular level of training. The University of Washington in the USA has pioneered such an option by designing a two week Emergency back-up, Vacation, Academic and Technical Skills (EVATS) rotation that has been very successful at ensuring integration of simulation training into the residency programme.¹⁰⁸

The last (independent-study) approach is an option that may be suitable for certain sets of surgical skills. For example, individual deliberate practice to improve microsurgical skills could be facilitated in this method following formal completion of a basic skills course. In this approach it is important to choose modules that do not require close supervision. That said, formal feedback and competency assessment (e.g. by video-recording of completed exercises) should be provided as independent learning here constitutes a formal component of the training curriculum.

Standardization

The need for standardization of training in plastic surgery at a global level is a major challenge. Variation extends from the scope of the specialty in general, to teaching and education of particular techniques. A review of microsurgical training centres worldwide established considerable variation in basic microsurgery courses' characteristics.¹⁰⁹ Given this wide variations, it would be helpful to have a basic standard of course duration, models, exercises, graded complexity and competency achieved, to ensure a universal standard of training and the possibility to compare and contrast the relative benefits of these divergent courses in training outcomes.

Skill maintenance and revalidation

Revalidation aims to assess surgical skill level and any skill loss over time.^{110,111} Assessment of senior clinicians should enable a safe threshold to be established that might be expected of a competent plastic surgeon. Once this threshold is fixed, future assessments can be made against it. Revalidation requires valid assessment tools that adequately evaluate competence. Simulation strategies offer a valid evidence-based foundation. Simulation-based interventions are particularly beneficial when used as part of on-going skills maintenance programme. It has been shown that distributed training leads to higher skill retention.¹¹² Even at more senior levels, simulation offers an effective tool to ensure continued levels of competence as part of the process of revalidation.

Simulation limitations and pitfalls

A nationwide survey by the Association of Surgeons in Training identified disparities in the availability of simulation training facilities by region and specialty in the UK.¹⁵ This variation may impact on the integration of simulation into the plastic surgery curriculum. The lack of validation studies of some simulation-training models and interventions cast a valid doubt as to whether they meet adequate educational standard. The costs of

high-fidelity training models and technologies are another major hurdle. This factor limits many training programmes' ability to acquire and develop advanced simulation models and educative interventions. It is estimated that the current annual cost of training surgical residents in the US is US\$53 million, when prolongation of the operation procedures is taken into consideration.⁶ Investment in simulation training is financially beneficial for healthcare systems. Evidence from neonatal care in the developing world confirms that low-fidelity task trainers and simulation-based educational strategies can be designed to provide cost-effective solutions with measurable advantage for both trainees and patients alike.^{113–115}

When such difficulties are overcome and a simulation facility is established, engagement of trainees and trainers continues to be a limiting factor for expanding simulation training. Engagement of the educative faculty is a further pressure. Some simulation centres have invested heavily in simulation equipment with no subsequent effective training programmes due to the lack of a motivated faculty. On the other hand, trainees may not appreciate the existence of simulation facilities, may not be released, or appropriately supervised in their use leading to poor utilization.

To optimize simulation training, an investment in coordinated multidisciplinary, multispecialty centres that ensure easy access from clinical areas and open access to simulation-training equipment is required to ensure utilization, educational benefit and return on investment.^{116,117}

A major and continuous challenge to expansion of simulation-based training is to establish conclusively its benefit on real clinical outcomes. As a result, inferential and cost-effectiveness studies are continuously required to establish the predictive validity of simulation models and educational interventions before advocating their integration into formal surgical training programmes.

Finally, even with any proven advantage of simulation-based training in shortening early parts of trainees' learning curves, by reducing medical errors and promoting good surgical practice, simulation cannot completely substitute key clinical experiences and learning from actual surgical practice. Plastic surgical expertise can only be achieved through clinical practice, no matter how long and frequent exposure is within simulation facilities. Simulation methodology can facilitate a more complete understanding of expert performance and, therefore, ensure that surgical training programmes of the highest quality are developed with this expert performance as the standard.¹¹⁸

Conclusion

The utility of simulation in surgical training is well established with proven validity and demonstrable transfer of skills to the clinical setting. The use of a step-wise simulation-based methodology to complement clinical training in plastic surgery has positive consequences on trainee confidence, leading to shorter

and flatter learning curves. Simulation training can better prepare trainee plastic surgeons for clinical practice, improve patient safety and service efficiency.

Despite the development of various models and simulation-based learning tools in plastic surgery, the role of simulation in the specialty's training curriculum is less well established. It is necessary for simulation-based training to be fully integrated and funded in formal plastic surgery training programmes. It is also necessary to develop a skilled faculty of educators in well-coordinated simulation facilities.

Further research is needed to expand the role of simulation in plastic surgery within training, performance evaluation, certification and revalidation. Simulation, therefore, has the potential to become an integral part of the development of better and safer plastic surgery services for patients worldwide.

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CHAPTER 78

Military plastic surgery

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Introduction

The field of burns, plastic and reconstructive surgery in the military covers a unique spectrum of surgical disease. Surgeons have long been found at the periphery of the battlefield and this tradition has continued into conflicts in Iraq and Afghanistan as surgeons develop new strategies to cope with the increasingly complex wounds produced from modern weapons.¹

This chapter focuses on the injuries to soldiers during conflict and the physical mechanisms that explain the type of tissue disruption seen. An explanation as to the decision-making process and surgical techniques used by military plastic surgeons will be explored.

The principles of war surgery

The practice of war surgery is starkly different from that of normal civilian surgery.^{2–4} The limitations on time, supply, surgical capability, access to evacuation and physical expertise are a few of the factors that influence the decision-making process when practising surgery in conflict conditions.⁵ There are several paradigm shifts in science that have had profound effects on the practice of war surgery.⁶

Paradigm shifts

The advent of triage by Dominique Larrey under Napoleon Bonaparte was a major shift in war surgery and medicine that improved survival.^{7–10} The requirement for prevention of contamination and haemorrhage is where the military plastic surgeon is vital. As high-intensity blasts have become a significant mechanism of injury there has been a large increase in the amount of soft tissue disruption, especially in the limbs.¹¹ Meticulous inspection and debridement of devitalized tissues are essential in conjunction with arrest of haemorrhage from small or large vessels. Some of the terminology used in war surgery is explained in Box 78.1.

Compounding 'X' factors

The factors that substantially impact surgical decision making are the logistical confounders on the ability to operate.¹² The lack of resupply for key equipment or of a secure evacuation chain for patients will influence the care that patients receive. Ultimately, the requirement is to operate for as short a time as possible so as to be ready for any further casualties that may need surgical care. In addition, conserving equipment so that one can do 'the most for the most' means often leaving certain surgeries for a later stage, especially if they are not life threatening. Specialist equipment such as microscopes may be unavailable, limiting microvascular procedures. Conflict is dynamic and chaotic; surgeons often need to be prepared to move quickly, and the prolonged evacuation time can often augment the triad of coagulopathy, hypothermia and acidosis. Fighting this deadly triad as well as meeting the clinical guidelines of the golden hour and platinum minutes is a huge challenge faced by military clinicians. In previous conflicts evacuation timelines have been prolonged (Op Iraqi Freedom USA 4.5 h, 1.5–72 h evacuation); this often necessitates the deployment of surgeons forward to undertake limited damage control surgery.

The concept of war wounds

Research from Afghanistan has highlighted the differences between the severities of civilian and military injuries. The application of wound management principles discussed in the literature can only begin once the complex energy transfer to tissue from projectiles is understood. This allows military surgeons to plan and comprehend the progression of a wound.

The wounds that result from these high-energy blasts are contaminated from a multitude of factors, such as foreign bacteria, soil, human tissue and metallic fragment debris. In addition, the tissue is devitalized from the shock wave of the blast, which causes cellular disruption on both a mechanotransduction and cellular hypoxia level. As cells are removed from

Box 78.1 Terminology

- *The golden hour* – The theory that the chance of survival is greatest if patients receive life-saving intervention within the first hour.
- *The platinum minutes* – Chance of survival in catastrophic injuries is increased with important life-saving interventions, such as tourniquets, within minutes of injury.
- *Damage control resuscitation* – Early permissive hypotension and haemostatic resuscitation.
- *Damage-control surgery* – Rapid, specific surgical intervention with predetermined goals to halt life-threatening haemorrhage and surgical pathology for stabilization of the patient. Time in theatre is limited to a minimum.

their natural extracellular matrix and unable to respond to changes in wound tension, many cells undergo anoikis. A subsequent cellular population undergo necrosis due to the local hypoxia and cellular trauma. Cells are unable to mount an immune response to local contamination or systemic inflammation as the energy disrupts gene transcription.^{6,13} At the local wound level there is a prolonged cellular necrosis, which maintains a destabilized tissue much longer than is traditionally seen in civilian patterns of injury. The cellular dysfunction affects the ability of local blood vessels to withstand the shear stresses from surgical reconstruction, making soft tissue reconstructive options limited.

The energy transfer into the tissues acts as a vector to force the contaminant substances into the tissue planes. This deep widespread contamination facilitates progressive tissue necrosis. It also is difficult to assess the depth of the contamination field on first inspection. Progressive tissue lavage and inspection allows the true contamination field to be assessed and reduced.¹⁴

The principles of war wound management involve prompt debridement of contaminated and obviously devitalized tissues. It is important to conserve tissue for later reconstruction. Anatomical structures must be identified for further reconstruction. Although vascular structures must be maintained or repaired, this should be weighed up against the requirement for swift damage control surgery. Prolonged operating times must be avoided. Limb salvage is crucial, but long operations to maintain marginally viable limbs has to be considered in view of further trauma that may arrive. Internal fixation of bony injuries should be avoided due to the degree of contamination. The use of negative pressure dressings to tamponade the wound is a new concept that appears to be successful. The aim of the immediate surgery should be to preserve tissue with minimal contaminants so that more definitive reconstruction and salvage may take place at specialist centres.¹⁵

Classifying war wounds

The assessment of cavitation size, wound contamination and involvement of neurovascular and bony structures¹⁶ provides plastic surgeons with one way to gauge wound severity. However the large soft tissue deficits seen in recent conflicts in Afghanistan and Iraq has meant surgeons require a new classification to

grade such wounds, which are often over 2 cm and may have a complex morphology. The use of CT to produce a precise impression of the soft and hard tissue injury involvement cannot be underestimated. In particular, this has proved invaluable in planning of subsequent reconstruction and in focusing the initial life-saving surgery.

The mechanisms of injury

Injury protection

The overwhelming mechanisms of injury in modern conflicts are penetrating injury from ballistics such as rifle rounds as well as overwhelming blast injury from powerful explosives.^{17–20} Protection from these mechanisms has evolved into a science of itself, with the aim of producing increasingly light materials that can withstand high-energy contact.^{17,18,21} Such materials must be robust enough to survive the arduous conditions that soldiers operate in and provide some of the expected modern material properties – namely waterproofing, breathability and a reduction in weight. This improvement in protection has shifted injury pattern back into the extremities. Particular breakthroughs have been in neck, ocular and perineal protection.

Blast mechanics

Blast injury is not solely limited to the battlefield and unfortunately is increasingly being seen in civilian settings.²² The terrorist bombings in London in 2005 and Boston in 2013 are examples of explosive devices in a civilian setting that may cause patterns of injury similar to those seen in conflict.²³ Understanding the physics behind these devices and how they disrupt air/fluid or air/tissue interfaces is important for management.

Blast physics

An explosion is a sudden release of energy from a source. The explosion and subsequent energy release perpetuate a wave of energy that travels outwards from the source. The largest explosion ever recorded was in Krakatoa in 1883 where the sudden eruption of molten lava from a volcano into the ocean produced enough energy to evaporate 1 cubic mile of ocean water; this equates to about 5 billion tonnes of TNT. The explosion was felt over 3000 km away.

Explosions release gas as a result of rapid energy release. The expansion and contraction of heated gas and the propagation of this heated gas away from the epicentre gives a diverse set of implications on tissue and structures. The rate of this energy release from a compound is termed the detonation velocity. It is important in subsequently determining the size of the shock wave and strength of the blast. The comparison of the detonation velocity from the epicentre against the speed of sound is termed the Mach number. The shock wave refers to the gas/air interphase that propagates out from the explosive epicentre, travelling faster than the speed of sound. It is this wave of energy that is pathognomonic of blasts. The wave is a high-pressure area that gradually

overtakes lower pressure areas, which in the initial phase, accelerate overall pressure. This is the pressure wave that individuals feel after an explosion. The wave acceleration is dependent on three factors: pressure, temperature and air density.

As the pressure wave is an area of high pressure and density, the subsequent cooling of the gas and negative pressure effect behind the wave creates a wind that follows the shock wave. This blast wave is dragged with the shock wave and pulls with it loose fragments in the vicinity. As the shock wave travels further away from the blast epicentre its velocity decreases and the distance between the shock wave and blast wind increases.

The shock wave itself can reflect off surfaces and therefore once again increase its disruptive power. As the speed of the gas is defined by density of air, a reflection of the shock wave on a surface can cause the shock wave to travel back through itself. The high temperature of the gas causes the shock wave to accelerate, and in some circumstance overtake itself, in the opposite direction, compounding the blast energy felt in between, and resulting in even more damage. This explains why blasts in confined areas result in more severe injuries.

Blast patterns of injury

The interaction between the shock wave, blast wind and the air/tissue interface produces several different patterns of injury.

Primary blast injury

Primary blast injury refers to trauma from the shock wave. The shock wave produces a rapid increase and decrease in air pressure and density. In addition, this rapid acceleration and deceleration causes severe disruption and shear to soft tissues. The air/fluid interfaces are particular vulnerable, as well as points for soft and hard tissue attachment, where shear forces could cause a ripping effect. The tympanic membranes, lung parenchyma and the large and small intestines are particular vulnerable in primary blast injury.

Secondary blast injury

The secondary blast injury refers to the effects caused by the blast wind. The wind not only carries explosive fragments from the device but also picks up surrounding loose material. These fragments provide a penetrating injury to soft and hard tissue. Such effects can be noted by the peppering of skin with small pieces of dirt or stones from the ground.

Tertiary blast injury

The interaction of the shock wave with surrounding structures and buildings can produce structural deformation. The collapse of walls or buildings or vehicle roll can produce crush and blunt trauma to the body. Crush injuries to limbs can produce a substantial myoglobin release and compartment syndrome. This is particularly found in long bone or extremity fractures, injury to the buttocks or abdominal wall. In addition, pelvic fractures may produce an abdominal compartment syndrome that may require decompression through an emergency laparotomy.

Quaternary blast injury

The blast also produces a reaction that combusts chemicals. However incomplete combustion produces thermal burns or airway compromise and/or asphyxiation through carbon monoxide or cyanide inhalation. Radioactive material is another factor that has to be considered in military blast injuries and the resultant medical problem could be a radiation injury. The exposure to a radioactive or chemical agent will require the use of special chemical/biological/radioactive/nuclear (CBRN) protection and patient decontamination prior to treatment.

Finally, the blast injury produces profound cellular dysfunction. Study of neutrophil function of blast patients has shown a significant reduction in functional neutrophils. This immunocompromise can mean that patients mount an overwhelming sepsis to local bacteria that colonize wounds from the blast.

Figures 78.1, 78.2 and 78.3 demonstrate the devastating injuries caused by blast injury through primary to quaternary mechanisms.

Penetrating injury

The study of ballistics refers to projectiles that have been launched or shot.²⁴ Although the tendency is for clinicians to associate this science with bullets, it must be remembered that fragments picked up in a blast wind can display ballistic properties.



Figure 78.1 Improvised explosive device blasts have caused a devastating injury zone to the lower limbs, perineum and the upper limbs, particularly the hands.

First, the physical attributes of the ballistic are important – namely the shape and composition and structure. Some historical compounds used for bullets were rounded and relatively soft. Modern-day bullets are often harder and have a pointed or rounded nozzle shape, which displays different travel characteristics. Second, the characteristics of the flight path for these ballistics, such as the velocity of the bullet, its stability in flight and finally its yaw or tumble, are crucial. The yaw refers to the degree of rotation of the bullet during its flight.



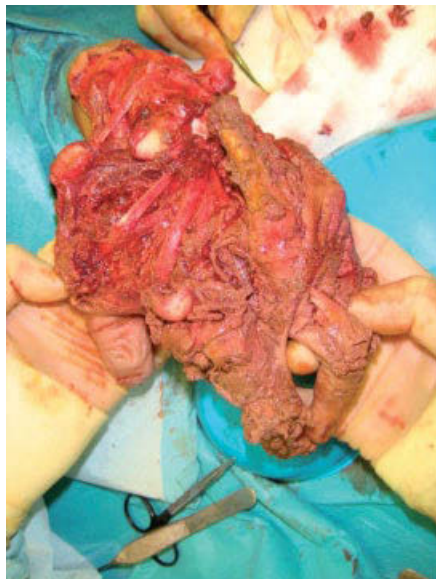
Figure 78.2 The decision for limb salvage is vital for soldiers, but this must be balanced against the available time and likelihood of success.

Historically, scientists and the literature have referred to bullets as high or low velocity. This is a misleading misnomer that refers to speed of flight. The low velocity was considered to be below 600 m/s or 2000 ft/s. Conversely, any bullet over 600 m/s (2000 ft/s) was 'high velocity'. In the study of bullets it is better to consider them as high energy or low energy, as low-velocity bullets can still be high energy, particularly shotgun cartridges. Low-velocity bullets were traditionally shot from handguns, especially in the civilian setting. Military weapons, such as the SA80 rifle used by British forces, fire a high-velocity round.

As energy must be considered to differentiate rounds, the link between kinetic energy, mass and velocity should be established and explained. Kinetic energy is equal to the sum of mass and the square of velocity. Hence although both mass and velocity will produce a proportional rise in kinetic energy, the increase in velocity will substantially increase kinetic energy over mass.

The efficiency of the projectile in its time of flight critically decides its ability to disrupt tissue. The more efficient its time of flight, the less energy it will impart to the atmosphere and therefore lose, hence the more it will deliver to the target. The speed of the projectile as it hits the target and the subsequent density of the target will govern the permanent and temporary cavity. Again, the thickness or depth of the target will also govern the amount of permanent and temporary cavitation.²⁰

Permanent cavitation refers to the disruption caused by the cross-sectional area of the projectile. The faster the projectile travels through the tissue, and the thicker or deeper that tissue is, the more energy that will be transferred as the projectile is allowed to yaw through the tissues, hence deviating from its



(a)



(b)

Figure 78.3 (a) The hand has sustained a particularly distinctive pattern of injury through improvised explosive device blast injuries that has not been documented before from other mechanisms of blast. The decision to salvage or not must be taken in the context of any other function-limiting injuries that have been sustained. (b) The upper limb injury, particularly to the hands, has required complex reconstructive choices with both new and old, often unused, flap choices. The decision was made to salvage this hand which, after delayed reconstruction, was of important functional use.

flight path. This energy transfer will produce a temporary cavity that will disappear as the round leaves the tissue and therefore creates a negative pressure gradient similar to that described in blast. This will pull the temporary cavity together and therefore mask its true size. In relatively thick tissues, such as the abdomen, there is more time for the round to yaw and transfer its energy. This results in more tissue disruption. The degree of disruption is proportional to the depth of tissue penetrated, or length of time the projectile is within the tissue cavity. Hence through and through penetration of extremities, such as the ankle or calf, present relatively little time for energy transfer from the projectile. Furthermore there is minimal time for the projectile to yaw, or rotate during penetration. The elasticity of the tissue is therefore important. Structures such as bone that are tough but relatively brittle will fracture easily and are poor absorbers of energy. Lung parenchyma has low density and is relatively elastic and therefore can undergo some relative deformation prior to breakage and hence is good at energy absorption from the projectile. Structures such as liver or spleen are excellent examples of tissues that have poor elasticity and are easily damaged by poor energy absorption.

Finally, it was originally believed that the speed of the projectile was the determinant in the amount of tissue disruption. Although this does govern the relative amount of kinetic energy in the projectile, it is in fact the density of the tissues that is the key factor in what determines tissue susceptibility to disruption.

Deployed plastic surgery

The war surgery team

Military surgery relies on critical teamwork. Plastic surgery brings a wealth of unique techniques that can help in improving patient outcome. However, in severe trauma patients such skills are futile without the full capabilities of the trauma military surgical team – skills brought from the orthopaedic and general surgical disciplines.

The military plastic surgeon

The role of the deployed plastic surgeon is to contribute to the surgical skill set of the team to ensure that maximum reconstructive potential of each patient is achieved. Initially this involves aiding surgical debridement to preserve as much viable tissue as is possible. The plastic surgeon is often best placed to determine what structures can or cannot be reconstructed to a good functional level long term. The use of early aggressive resuscitation techniques through pre-hospital care has increased the number of survivors with a high surface area of tissue devastation. These conflicts are unique in this sense. A wealth of different procedures have been required to salvage tissue for the reconstructive phase. The construction of an established hospital in the conflict area with a wide variety of expertise has meant that patients can undergo procedures previously thought to be outside the remit of damage control surgery; this allows

more scope for later reconstruction. Although bringing the full civilian trauma team closer to the point of wounding has, without question, improved outcomes, such resources cannot be expected in the future.

Principles of plastic war surgery

First, it is essential that damage control surgery takes place over all other surgery. All patients should be managed with a trauma principles approach. Life and limb must be preserved first through control of haemorrhage and management of life-threatening conditions. Subsequently, meticulous debridement and tissue conservation can take place. If time allows then procedures to preserve borderline tissue or important joints may take place. The phrase 'out of sight out of mind' is the clinical enemy in these patients and can lead to a missed large bleeding wound from a blast. It should be remembered that surgery on major trauma victims is resource intensive. The conflicts in Afghanistan have benefited from a quickly evolving well-established logistical chain that has allowed a gold standard in resource-intensive resuscitation of these patients.^{11,12,25–27} The aim in any complex wound from war is to achieve debridement with maximal tissue preservation, fracture fixation and wound control with dressings.

Wound debridement and management

The aim in the acute phase is to reestablish critical small vessel flow, assess tissue viability and debride dead tissue and preserve what may be recovered through resuscitation. Such patients have just come from the stressed environment of the battlefield. Although every effort has been made to meet the nutritional requirements of soldiering, these patients are often in nutritional deficiency, which concurrent to their already stressed state, affects their ability to mount an immunological response. Although nutritional supplements may be unavailable in the deployed setting, the restoration of adequate oxygenation through resuscitation with whole blood products (1 : 1) can partially bridge this gap and has significant positive outcomes for avoiding the lethal triad. Optimizing such patients prior to theatre is paramount as part of the damage control process. Salvaging these patients if they go into the lethal triad and towards multi-organ failure is difficult and therefore every effort should be made to adequately resuscitate them.^{27–32}

Study of the rate of osteomyelitis within the first 5 h of wounding as well as the rate of bacterial replication have identified that the first 6 h are crucial for primary debridement.³³ Jackson's model of the burn wound also has some translation to the open wound. The central wound area is one of necrosis, which is surrounded by an area of devitalized tissue and subsequently engulfed by normal cells. In many fields of surgery a radical debridement, which removes all necrotic and devitalized tissue, leaving normal tissue only, is appropriate. In military trauma where healthy tissue is at a premium, one has to adopt a more sparing approach. Meticulous assessment of tissue viability along with small marginal debridements of necrotic tissue

is essential. Devitalized tissue must be continually reassessed with the hope that it may be salvaged through aggressive resuscitation. This approach leads to multiple wound assessments and changes of dressings in theatre, yet it conserves vital tissue. It is also at the mercy of the progressing tissue necrosis, which can present gradually days after the injury.

The importance of microbiology in guiding tissue management cannot be overstated. Tailoring antimicrobial therapy is a feature of the definitive surgery phase, however early antibiotic therapy is vital and at the earliest possible occasion swabs of all wounds should be taken. Clinical Guidelines for Operations, the UK military's evidence-based guidelines for all clinical work on operations, has appropriate first-line antimicrobial advice for patients with severe trauma. Early antimicrobial therapy helps in arresting the substantial cellular, immune and inflammatory response to the injury.

Vascular control

In the severe trauma patient with an extremity amputation, proximal control is crucial.³⁴ In such patients aortic cross clamp through an emergency laparotomy may be necessary. Rapid assessment of a limb's future viability or the necessity to undertake a surgical rescue is a difficult decision for the deployed plastic surgeon. Experience alone has led to a small set of key questions to be asked when considering such a move. The decision to undertake such a procedure should be weighed up against the risk to the patient, the gains from salvaging tissue and the limb (taking into account any loss of function) and the

overall burden of trauma for the operating table. A vascular repair in a borderline mangled upper limb may salvage a much-needed hand. The limb salvage algorithm shown in Figure 78.4 highlights the decision-making process.

Nerve repair

In the acute stage nerve repair is not indicated and identification of the proximal and distal ends should warrant labelling and burying under viable muscle to provide nutrients and prevent early degeneration. Prevention of severe nerve retraction within the compartment is important and should be undertaken. However, such patients will undergo a migratory necrosis and this may affect nerve tissue viability and this is why primary repair is contraindicated. Although nerve repair is important in the subacute phase in the correct facility where time, patient and logistical constraints are not limiting factors, it should not impede prompt damage control surgery and evacuation.

Tendons and muscles

Tendon and muscle debridement, identification and apposition are important. However, in the acute period identification of the profoundly dead tissue should be the priority. Identification by muscle stimulation by finger stimulation, tone and colour are important in deciding viability. In the sedated patient anaesthetic agent will have an effect on the ability of muscles to contract and this should be kept in mind when finger stimulating potentially non-viable muscle. Once again a marginal approach to debridement should be employed. Furthermore, muscle compartments

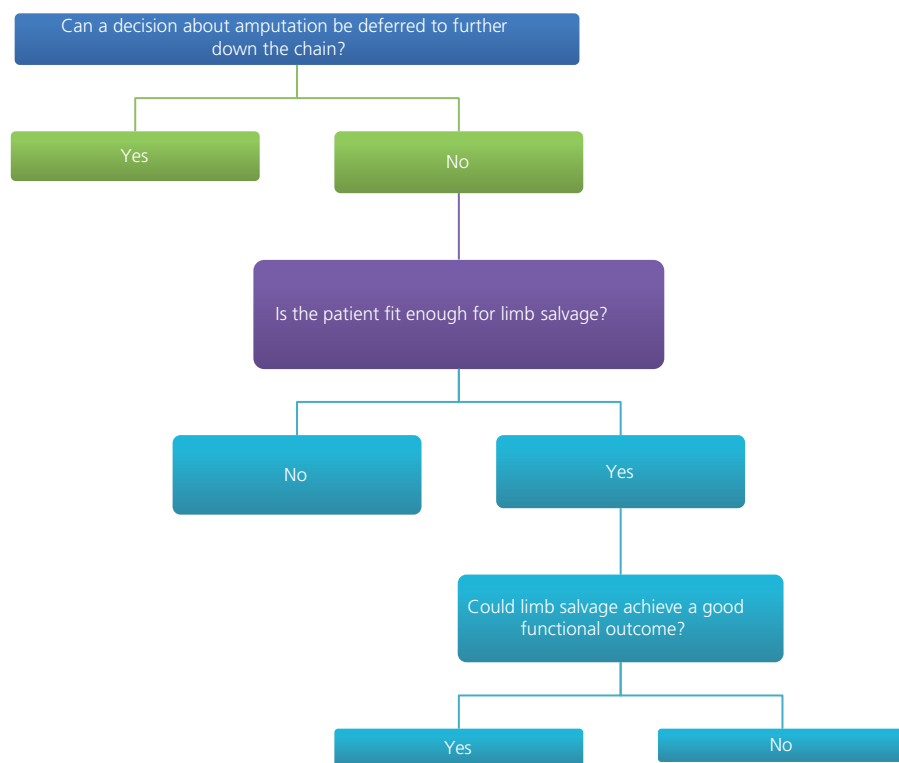


Figure 78.4 Limb salvage algorithm.

within the acute zone of injury may need to be incised and released to pre-empt compartment pressure increase. Prompt lavage of the muscle, its residual sheath and compartment to rid the area of contamination will enable closure with negative pressure therapy to temporize the wound. Furthermore, assessment of limbs to rule out the requirement for fasciotomy is necessary.

Fractures

Early fixation of fractures is paramount in balance with the patient's physiological state and the overall time constraints in theatre. Fixation of long bone fractures to prevent continuing blood loss is important. However, the use of internal fixation is contraindicated due to the susceptibility for infection and osteomyelitis. There have been numerous reports of the use of external fixating devices in the acute setting for salvage of fragmented joints with mixed success. The use of such devices should be following the lead of the orthopaedic teams for optimal success. Any signs or symptoms of contamination of the fixating rods should lead to removal and replacement. The combination of external fixation and topical negative pressure therapy to simultaneously splint the wound and fracture has been utilized with some success.

Dressings

In the acute setting the use of topical negative pressure therapy for its ability to provide a sealed contaminant-free wound has proven to be useful in these patients. The eventual replacement of silver and gauze-based dressings in theatre by topical negative pressure therapy has improved wound care. However, once again such a device cannot be expected in future deployments, therefore the surgeon needs to have a grasp both of the dressings offered in their theatre and the most appropriate dressing for the wound. The use of crepe bandage, gauze and silver dressings was the accepted norm in the early stages of recent conflicts. The use of the marginal necrosis has led to a dressing change and wound inspection at the 24–48 h mark and again this needs to be considered, especially in a prolonged evacuation chain.

Delayed reconstruction

Such severely injured patients enter a prolonged period of catabolism. This can last for up to a year and, combined with a highly sensitive immune system (especially in those after a massive transfusion), the need for surgery should be balanced between 'What *needs* to be done' and 'What *should* be done'.¹

The initial aims for these patients are wound closure, which facilitates the transition from a chaotic systemic inflammatory response syndrome (SIRS) response to one of relatively ordered healing. The goals of preliminary reconstruction in the subacute setting are defined as appropriate coverage of amputation stumps, nerves, tendons and muscle units in a clean wound. Particular attention should focus on fracture fixation as well as covering nerve grafts and musculotendinous units. The priority of reconstruction should be functionally based. This must be

undertaken in a multidisciplinary team approach so as to ascertain the patient's expectations in the context of their injury. As the patient transitions to the rehabilitation phase, the wear and tear of physiotherapy and readjustment to relative self-sufficiency guides the subsequent reconstruction.

Particular reconstructive techniques in the acute setting have proven marginally unsuccessful. Microvascular free tissue transfer has a 15% unpublished failure rate. This again is probably because of the overwhelming SIRS response. The friability and lack of vessels to connect free flaps to is also a major issue. The choice of muscle and flap is important. The use of the latissimus dorsi as a workhorse flap is contraindicated in the bilateral lower limb amputee, who will need this for upper limb function. The gracilis, if not obliterated in the initial injury, has proven an excellent workhorse with minimal donor site morbidity.

In relation to lower limb amputee reconstruction it has been found that the 2-year point is the optimal time for definitive surgery. At this time a staged stump revision that utilizes a rotational flap, excision of any heterotopic ossification and appropriate burying of nerves has good outcomes for prosthesis. In addition, the use of Botox is excellent to arrest sweating into a prosthesis.

Ultimately, the reconstruction of these soldiers needs well-defined aims that consider the patient's understanding of their best effort functional outcome in a multidisciplinary team approach.³⁵ These patients present a reconstructive challenge, but not one that is outside the scope of the basic principles of the reconstructive ladder.

Conclusions

Military plastic surgery requires a breadth of skills from the surgeon to overcome a vast array of unforeseen clinical and environmental challenges. Success in this field is not just about applying high-quality surgical techniques, it requires the ability to make sound judgements on the most appropriate short- and long-term surgery that is best for the patient.

Recent conflicts have given war surgeons a relatively fast evacuation time from point of wounds to specialist centre. This has allowed surgeons to use reconstructive techniques early to achieve some of the best outcomes ever seen from such injuries. However, future conflicts may be conducted under very different circumstances. Military plastic surgeons need to have the surgical skills to deal with a wide range of injuries and to be flexible enough to cope with unpredictable circumstances that war brings.

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Plastic surgery fellowship and board exams

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FRCS(Plast) examination

FRCS(Plast) is the Intercollegiate Exit Examination in Plastic Surgery run jointly by the four Royal Colleges of Surgeons in the United Kingdom. Passing this exam, along with satisfactorily completing 6 years of higher surgical training in plastic surgery, leads to award of a Certificate of Completion of Training (CCT), which allows entry onto the Specialist Register. This enables a surgeon in the UK to work as a consultant in their speciality within the National Health Service or in private practice.

The format and regulations relating to the FRCS(Plast) examination are revised from time to time and for up to date information the website of the Intercollegiate Speciality Boards (ISB) and Joint Committee of Intercollegiate Examinations (JCIE) is best consulted (<http://www.intercollegiate.org.uk>).

In brief, there are two sections to the exam, which cover the whole of the plastic surgery syllabus, including basic sciences, medical ethics and statistics. Section 1 consists of two written papers: multiple choice questions (2 h) and extended matching item questions (2.5 h). The papers are not negatively marked, though the pass mark varies each sitting as it is set for that particular paper by a separate panel of examiners. In the past it has been in the region of 60–70%. Of note, some questions are subsequently withdrawn if they are deemed by the panel to be ambiguous or not of the appropriate standard.

Candidates successfully completing Section 1 can then progress to Section 2, which is usually held a few months later. Both sections have sittings twice a year.

Section 2 is the clinical part of the FRCS(Plast) and is held over two days: one day clinical cases, the other oral exams. The clinical cases are made up of two medium/long cases lasting 15 min each and two rounds of short cases, each round consisting of five short cases and lasting 30 min. Candidates may be asked to interpret X-rays, CT scans and other pieces of diagnostic imaging related to the case in question.

The oral exam is made up of three separate 30 min vivas, each with a different pair of examiners. The broad subject areas for each viva are:

- 1 Trauma (hand/maxillofacial/lower limb) and burns
- 2 Acute head and neck tumours, cleft management and genitourinary reconstruction
- 3 Basic sciences and generic plastic surgery, aesthetics, ethics, medicolegal and consent issues.

Each viva has three distinct parts on different topics. One examiner will ask the questions while the other marks, alternating roles for each topic. Although there are separate topic areas, questions can be on any part of the plastic surgery syllabus and candidates may be shown clinical photographs or asked to draw certain schematic diagrams, such as the cross-sectional anatomy of the lower leg, geometry of local flaps, different cleft lip repairs, etc.

Marking

Each constituent part of Section 2, the short cases, the long cases and the oral exam, contains a certain number of marking events which are each given a score between 4 and 8 (6 is a pass). A breakdown of the weighting of scores for the different parts is given in Table 79.1. Thus a single short case or individual viva topic equates to one marking event, each long case contains three marking events. The scores for each part is doubled (individual pass marks given) and added together to give an overall score. The overall pass mark is 300.

When attributing a score, the examiners take the following areas of a candidate's performance into consideration: overall professional capability, knowledge and judgement, quality of response and 'bedside manner'. A copy of the marking descriptors that will be used by the examiners is sent to candidates in advance with their examination pack and there is also an up to date copy on the JCIE website (<http://www.intercollegiate.org.uk>). It is advisable for candidates to check this for any changes to the marking scheme that may subsequently occur.

Personal experience – learning resources

Preparation is very much an individual thing, as are textbooks, but one extremely useful resource to be recommended is the *frcpsplast.com* website, which contains a panoply of revision notes, landmark papers and tips from previous candidates. Registration is via

Table 79.1 Breakdown of the weighting of scores

	Number of events	Pass mark
Long cases	6	72
Short cases A	5	60
Short cases B	5	60
Viva 1	3	36
Viva 2	3	36
Viva 3	3	36
Total overall		300

email and access free. The Intercollegiate website has a few sample MCQs and EMQs, although these are quite limited in number.

A point for overseas candidates is that management practices do vary slightly from country to country and it is important to be familiar with certain UK guidelines, such as those for skin cancer (produced jointly by the British Association of Dermatologists and the British Association of Plastic Reconstructive and Aesthetic Surgeons (BAPRAS)) and lower limb trauma (produced jointly by the BAPRAS and the British Orthopaedic Association). The latter can be downloaded from the BAPRAS website. The National Institute for Health and Care Excellence (NICE; www.nice.org.uk) also produces a range of clinical guidelines (in addition to evaluating new interventions or technologies and cost/benefit analysis), some of which are particularly relevant to plastic surgery (e.g. pressure sore management, fat grafting, venous thromboembolism prophylaxis), and candidates should be familiar with these.

Good (if a little long) summaries of guidance on consent and ethics can be found on the General Medical Council website (www.gmc-uk.org) under the section entitled Good Medical Practice.

Personal experience – the exam

Taking the FRCS(Plast) examination is a little like taking your driving test. The key to passing the test is not about being a great driver; it is about demonstrating you are a competent driver, safe to be released in your car unsupervised onto the unsuspecting public. Likewise, success in the FRCS(Plast) is not about trying to show the examiners that you are a brilliant surgeon, what is paramount is that you are a safe surgeon, who, when released unsupervised as a new consultant, will come up with sensible management plans to the clinical problems you encounter.

So, just as the learner driver does not try to impress his or her examiner with wheel spins and handbrake turns out of the test centre, so the plastics trainee should be wary of allowing complicated flaps or the latest niche aesthetic techniques to be the first things to come out of his or her mouth when asked how they would tackle common clinical scenarios. The examiners are not thinking ‘how clever is this candidate?’, they are thinking ‘If this candidate was appointed as my consultant colleague next week, is he or she going to get into trouble which I would then have to sort out?’

A bit like driving, everyone’s an expert and sometimes the volume of advice candidates are given can be overwhelming, but below are a few tips I found useful.

Five tips

1. Each long case is worth a whole viva

One of the editors of this book gave me some very sage advice while I was studying for my FRCS(Plast) exam: ‘Everyone obsesses about the viva, but if you look at the people who fail it’s invariably a long case that has sunk them.’

There is a natural tendency, given the broad range of the syllabus, to obsess about the viva. It is worth remembering, however, that although a bad viva topic or short case can be compensated for by a strong performance elsewhere, if a long case goes badly it is much more difficult to recover from. It all starts with the history, and although it is easy to think that taking a history is something you have done countless times and do not need to practice, in the exam situation it is easy to forget things if you do not have a well-drilled routine. Long cases that go awry invariably stem from an inadequate history and examination, and the examiners’ ‘difficult questions’ are simply a consequence of this.

2. Be nice

It may appear to stretch credibility to say that, after 6 years of medical school and countless years of surgical training, your success will be influenced by mere pleasantries. But it is, and may well be the last thing on your mind while trying to retain a myriad of plastic surgery information in your head.

First, you are marked, both directly (and subconsciously), by the examiners on your manner with the patient. Second, the patients can be themselves a little nervous (especially some of the elderly ones) and will provide a far more coherent history if they are made to feel at ease. Third, and perhaps most importantly, in the clinical exam impression is everything. ‘Niceness’ comes across as calmness, and calmness conveys competence. And it is competence that the examiners are looking for.

3. ‘I don’t know’

There is nothing wrong with saying this – within reason (clearly if you say this when asked to draw a Z-plasty the exam is not going to end well). Obviously in an ideal world you would know everything, but in the real world there is a limit to everyone’s knowledge and the examiners know this.

In response to correct answers, the examiners will ask more searching questions in an effort to allow you to score more marks for that section. If you are asked an extremely difficult question then the chances are you are doing extremely well, so if you don’t know, it is reasonable to say so or make it clear you are making an educated guess and give your reasoning. Do not undo previous good work by making up something ludicrous or waffling for ages using up time when you could be scoring marks on something else.

4. If you do know the answer – be succinct

There was a perceived (and dubious) wisdom when I was at medical school regarding vivas. The theory went that if you were asked a question on a subject that you knew something about, you should try to spin the answer out for as long as possible. Because the longer you did, the less time would be left for the examiners to ask you questions on subjects about which you knew nothing. Here, the exact opposite is true. If you know the answer, give it fully but succinctly and move on to the next question – this is the only way to accrue more marks. Likewise, in the clinical cases, the time goes very quickly and a candidate who starts promulgating at length in response to a straightforward question may find the bell ringing before they can be asked anything else (thus limiting the marks they can score).

5. Be yourself

By this stage in their careers most candidates will have successfully completed many exams and this one is no different. For the clinical cases, imagine you are in the outpatient clinic and run through your history and examination routines. If you have always examined a hand in a particular sequence then stick to it, do not change two weeks before the exam because someone says you should do it differently.

One minor difference in exam technique from previous trainee exams is that answers that might have begun with 'You could ...' must now become 'I would ...'. You are answering from the point of view of a first day consultant.

Perhaps the best bit of exam advice ever was given to me by a non-medic. Years ago I was taking my MRCS (a junior surgical exam) viva at the Royal College of Surgeons in Edinburgh. We candidates were standing lined up in the corridor, knees knocking in our rented suits, as we waited to enter the examination hall. As we filed in to confront our examiners, an elderly porter, who was tending to one of the display cabinets near the entrance, growled at us in a thick Glaswegian brogue, 'Look 'em in the f***ing eye boys'.

FRACS(Plast)/EBOPRAS examinations

The fellowship or boards of plastic surgery equivalent exams are the final trial of ordeal for most trainees, who have done exams over a large portion of their adult lives. Their respective examining boards put the general structure of all four exams together, with the aim of presenting a safe surgeon who has adequately covered the entire plastic surgery curriculum. This is perhaps the most stressful academic period in a prospective surgeon's training career. All exams have their own unique quirks of exam technique and examiners habits or favourite topics. In recent years the various boards have attempted to provide a more standardized format to ensure reproducibility and fairness. As they begin to prepare for the final exam, each candidate finds their favoured study modality. Group study with a long-term timetable and regular tutorials from recently passed surgeons

and more experienced senior surgeons form the basis of most trainees' approach to the exams. The structure of the FRACS(Plast), FRCS(Plast), FRCSC and European Board of Plastic Reconstructive and Aesthetic Surgery (EBOPRAS) exams is described below with reflections on personal experience.

The Australasian College of Surgeons invites final year trainees from a 5 year specialist training programme conducted by the Royal Australasian College of Surgeons to sit the fellowship exams. All candidates must have passed a PRSSPE (PRS Science and Principle Examination) multiple choice exam usually as a SET 2 (second year of training) candidate. The final fellowship has the following components:

- I Two written papers, each of 2 h duration, two questions in each paper and multiple parts in each questions
- II Clinical – long cases: two cases/patients, each of 30 min duration
- III Clinical – short cases: six cases, each for 5 min
- IV Surgical Pathology & Operative Surgery (SPOS) 1 viva: two scenarios, each of 12.5 min
- V SPOS 2 viva: six slides, each of 5 min
- VI Anatomy viva: 25 min.

The scoring is a closed marking system – 8, 8.5, 9, 9.5. Support can be given in each of the marks; 9 is a clear pass, 8.5 is borderline fail, 8 is a clear fail. Any candidate can be failed on one critical error irrespective of the marks. Critical errors are considered those decisions that may be life- or limb-threatening. Although all seven segments are considered equal in value, during court of examiner discussions more emphasis is placed on clinical components in particular long cases. These exams are held twice a year.

The EBOPRAS exam is divided into written and viva components. The written exam is a multiple choice exam covering clinical as well as basic sciences. The viva component is divided into two 25 min sections. Clinical scenarios covering congenital, trauma, tumour management, aesthetic and general reconstruction are conducted by two examiners.

As a candidate I had the dubious pleasure of sitting three of the four exams discussed here (FRACS(Plast), FRCS(Plast), EBOPRAS). Each had their own strengths and weaknesses, however the common theme is that the examiners were not intent on failing the candidates, but to discover what their knowledge limits were and, more critically, where their clinical limits laid. The FRACS is the most rigorous of these exams in terms of expected depth of knowledge, but it is not necessarily the broadest exam. The written form is significantly more detailed and in my opinion more in line with the expectations of the specialist surgeon. The FRCS exam is the broadest exam; each candidate is assured that a series of congenital, trauma, aesthetic and general reconstructive viva questions will be asked. The arrays of short cases were undoubtedly the best outpatient's clinic any candidate is ever likely to attend. The EBOPRAS exams similarly seek to cover the same broad base. The candidates are often qualified surgeons and, as such, the exam format may seem more like a semi-formal discussion

among colleagues than the tutor to pupil format of the FRACS and FRCS exams.

The most pertinent advice for a prospective candidate is a broad formal clinical experience. This experience is best gathered in the trenches of interminable dressings and initial consultation clinics. Formal long case, short case practice and presentation is essential not only to success but also to crisp and succinct decision-making process. This will become less formal but nevertheless form the crux of your clinical practice for years to come. The examiners are destined to have more clinical experience than the candidates and this is where the candidate can compensate by depth and breadth of knowledge. The more a candidate reads and knows around a topic the more confident and systematic they will become in replying to questions and putting clinical scenarios into a correct perspective. Review CME articles from *Plastic Reconstructive Surgery Journal*, *Clinics in Plastic Surgery* and *Hand Clinics* formed the basis of my own reading in addition to the standard single-volume texts (the latter I often found inadequate). Revision and complete command of surgical anatomy is mandatory in clinical practice of plastic surgery. Unfortunately, neither the FRCS nor the EBOPRAS exam stress this component. This results in what is a palpable deficit in many of the British and European candidates' knowledge base.

FRCS exams

At the end of plastic surgery training in Canada, candidates are invited to sit the Canadian Plastic Surgery Royal College examination. The title Fellow of the Royal College of Surgeons of Canada (FRCS) – Plastic and Reconstructive Surgery is awarded following successful completion of this examination. The FRCS is a renowned designation recognized worldwide and is usually a requirement for specialty licensure in Canadian jurisdictions. This final certification exam consists of two components: a written section and an oral examination approximately two weeks later. Candidates are required to pass both the written and the oral components in order to successfully complete the exam. The examination addresses all facets of plastic and reconstructive surgery, including basic science, basic principles and techniques, paediatric plastic surgery, craniofacial surgery, burns and wound care, head and neck reconstruction, aesthetic surgery, trunk and lower extremity reconstruction, breast and hand surgery. A more detailed description of the plastic surgery curriculum can be found at www.royalcollege.ca.

Personal experience

Although this examination is stressful and comes at the end of a long and arduous residency training, it is considered to be a fair exam. There is an enormous quantity of information to master (as evidenced by the content of this book), but in the end, review and critical appraisal of the material while preparing for the exam will make any potential candidate a better plastic surgeon.

Failure is usually a function of two primary deficiencies: lack of knowledge and lack of examination-taking skills. The former shortcoming, a lack of knowledge, occurs simply because candidates did not read enough. Some may be more proficient at memorizing the material, but ultimately, with weeks, months and years of commitment everyone should be able to learn the required knowledge. There are no excuses. Preparation for the Plastic Surgery Royal College exam does not begin a few weeks or months before the exam, it starts at the beginning of the candidate's residency. The latter point cannot be emphasized enough. It is of the utmost importance that early on residents read around their cases and on the topics relevant to their current rotation. Consistent and continued acquisition of information throughout residency is key. A strong knowledge base is a function of discipline and dedication to acquisition of the material. It is simple: if one does not have sufficient knowledge, one will not pass this exam. So, the secret to success? Read, read and re-read.

It remains an unfortunate truth that even when armed with the knowledge required to pass the exam, failure may ensue due to a lack of examination-taking skills. However, as the word skill implies, this can be gained through experience and training. With regard to the oral examination, very often anxiety can overshadow one's ability to relay information in a proficient and succinct fashion. In like manner, for the written component, failure to pace oneself can often result in one falling short of completing the entire series of questions. The latter limitations can be improved with continued practice. Indeed, preparation for the exam can be likened to training for a marathon, and as any seasoned runner can attest, in order to achieve the objective of a long run, one must first devise a practice plan of shorter runs. Likewise, at the outset of one's final year of residency, formulation of a good reading and oral exam practice plan is essential. Training is always easier in a group, and organizing a study group of 3–4 residents with whom one can review the material, questions, cases and articles is recommended. Each member is likely to have a particular strength, which would assist the other residents in the group in gaining a better understanding on that topic.

Remember, the objective of the exam is not to impress the examiners nor is it a platform to exhibit innovative solutions to common clinical problems. Rather, it is to demonstrate that once in practice, without the direct guidance and supervision of senior colleagues, one has the necessary competence and will exercise the safety requisite to independent practice.

Written examination

The written examination, which can be taken in French or English, consists of two 3-h sessions of short answer questions (approximately 45 questions per session). The questions cover the entire breadth of plastic and reconstructive surgery, including basic science, embryology, anatomy, classifications, etc. The questions are prepared by plastic surgeons across Canada and submitted to the Examination Board of Plastic

Surgery. Following a rigorous review process, a final set of questions is selected to appropriately assess candidates' competence. These questions vary in complexity, from listing a series of answers to completing a number of questions relating to a specific clinical scenario or particular pathology.

Tips and pearls for the written examination

- 1 Do not be late.
- 2 Pay attention to the instructions provided by the invigilators and outlined in the booklet.
- 3 In order to answer all the questions, manage the allotted time thoughtfully. For instance, if there are 45 questions worth one mark each to be completed in 3 h, approximately 4 min should be allocated to each question.
- 4 Answer the question in a clear and concise manner. If, for example, the question is to 'list three risk factors,' then provide three, not four or five. Extra credit is not given for any additional answers provided.
- 5 Make an effort to write legibly. If the examiner cannot read the answer, then credit will not be granted.
- 6 Answer all the questions to accumulate the maximum number of points.

As an adjunct to point 6, it is highly recommended to not linger on those questions where the answers are not straightforward. Rather, mark these more difficult questions with 'post-it' stickers, so that they can easily be identified and answered once the rest of the exam is completed.

Oral examination

The oral examination can also be taken in French or English. It consists of two 1-h sessions, each session consisting of eight clinical scenarios with associated questions (six of 8 min duration and two of 4 min). The objective of the oral component of the exam is to assess the candidate's application of theoretical knowledge and clinical judgement. More importantly, the examiner seeks to ascertain that the candidate is safe with regard to clinical decision making.

The examinee is permitted 15 min prior to the start of the examination to review the series of clinical scenarios. Typically, two examiners will independently evaluate each candidate. Upon entering the room, the examiners will introduce themselves and give the candidate a short explanation about the oral exam. The clinical vignettes, illustrated with photographs, radiological imaging, etc., cover all areas of plastic and reconstructive surgery and with questions related to each. The aim of a scenario may be to evaluate a clinical problem, design a treatment plan, manage a complication, etc. Ultimately, it is important to understand that the examiners want candidates to pass the exam, and as such, they will guide you through the scenarios when necessary in an effort to elicit all information critical to passing the question. Examiners are instructed to not lead the candidate, so one should not expect any positive or negative reinforcement for responses given. Examiners may, however, redirect the candidate if responses are completely erroneous. At

the end of each session, the candidate is allotted 4 min for so-called sober second thoughts, where modifications or clarifications of any answers can be made.

Tips and pearls for the oral examination

- 1 Practice, practice and practice oral examinations. Whereas the written component of the exam is a reflection of knowledge acquired, the oral exam is, in principle, a skill that requires much practice to master. The latter also pertains to drawings and/or markings.
- 2 Answers are an indication of the candidate's thought process, and so structured and succinct answers demonstrate that the candidate's approach to a clinical problem is organized. It exhibits experience and preparedness for clinical practice.
- 3 As a corollary to point 2, it is important to listen carefully to the question and answer the question specifically. For instance, if asked for a specific investigation, do not elaborate on history and physical exam. Likewise, if asked about 'your preferred flap option for the given defect,' do not list a number of potential reconstructive options. Instead, state your surgical plan.
- 4 Show confidence, but avoid arrogance.
- 5 In the event you do not know the answer, it is acceptable to state it, rather than present a guess. In like manner, pausing (briefly) to reflect and properly formulate a response is appropriate.
- 6 Pertinent and selective consultation of other subspecialties is advisable. However, referral of every problem and/or every patient is inappropriate and demonstrates a lack of knowledge, judgement and/or confidence.
- 7 The Royal College exam is not the time to conceive of a new treatment option or suggest an innovative operation. Be safe and provide a well-established solution for the clinical problem at hand, and preferably, one seen or done during your training. If presented with a controversial topic, begin by explaining that this is an area of controversy and exercise caution when stating your response. Remember, ultimately, the examiners want to ensure you are a safe surgeon.
- 8 Stay positive and focused. If one question/scenario did not go well, do not dwell on it and instead concentrate on the next question. Do not let one question disturb your focus or confidence for the following questions. Keep in mind that at the end of the session, there will be time for any sober second thoughts.
- 9 Remember, the examiners ask the questions, not the examinee. By asking for more than the occasional clarification, the candidate may give the examiner the impression that they lack knowledge or confidence. Moreover, this is a waste of time, time which should be maximized to answer the question.
- 10 Formulating a standard template for each potential topic is useful as the type of cases tend to be repeated.
- 11 Finally, approach each case as though confronted with the clinical scenario in actual practice.

Conclusion

To conclude, it is indeed a difficult process, but with the right approach and adequate preparation, a passing result is an achievable task. Ultimately, review and critical appraisal of the material while preparing for the exam will make any potential candidate a better plastic surgeon. These are all fair exams, and

most graduating residents are well prepared to pass. Candidates would do well to remember that the objective is not to impress the examiners, but to demonstrate that once in practice, without the direct guidance and supervision of senior colleagues, one has the necessary competence and will exercise the safety requisite to independent practice.

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